These hearings present opening statements which argue for the government’s responsibility to protect the health of citizens, citing the dangerous and often fatal relationship of cigarette smoking to cancer and heart and lung disease. Statements include those from Mrs. Barney Clark, widow of the first heart transplant patient, and physicians, surgeons, and medical researchers. In addition, a number of published accounts of the danger of cigarette additives, such as deertongue, are presented. Other testimony describes smoking prevention studies and warning labels that are used in other countries. Reports linking smoking-related illness in non-smokers to association with heavy smokers are also presented. Opposing testimony, presented by representatives of the tobacco industry as well as professionals from the fields of medicine, education, and psychology, proposes that people are sufficiently aware of the risks of smoking and that further federal intervention is unnecessary and would set an unwanted precedent. Additional testimony stresses that cigarette advertising is not aimed at young people, and evidence relating smoking to health hazards is not conclusive. The hearings include testimony from 17 witnesses; 42 prepared statements; and 13 additional articles, publications, and communications. (JAC)
HEARINGS BEFORE THE COMMITTEE ON LABOR AND HUMAN RESOURCES UNITED STATES SENATE NINETY-EIGHTH CONGRESS FIRST SESSION ON S. 772

TO PROMOTE PUBLIC HEALTH BY IMPROVING PUBLIC AWARENESS OF THE HEALTH CONSEQUENCES OF SMOKING AND TO INCREASE THE EFFECTIVENESS OF FEDERAL HEALTH OFFICIALS IN INVESTIGATING AND Communicating TO THE PUBLIC NECESSARY HEALTH INFORMATION, AND FOR OTHER PURPOSES.

MAY 5 AND 12, 1983

U.S. DEPARTMENT OF EDUCATION NATIONAL INSTITUTE OF EDUCATION EDUCATIONAL RESOURCES INFORMATION CENTER (ERIC)

This document has been reproduced as received from the person or organization originating it.

Minor changes have been made to improve reproduction quality.

Points of view or opinions stated in this document do not necessarily represent official NIE position or policy.

Printed for the use of the Committee on Labor and Human Resources

U.S. GOVERNMENT PRINTING OFFICE: WASHINGTON : 1983
COMMITTEE ON LABOR AND HUMAN RESOURCES

ORRIN G. HATCH, Utah, Chairman

ROBERT T. STAFFORD, Vermont
DAN QUAYLE, Indiana
DON NICKLES, Oklahoma
GORDON J. HUMPHREY, New Hampshire
JEREMIAH DENTON, Alabama
LOWELL P. WEICKER, Jr., Connecticut
CHARLES E. GRASSLEY, Iowa
JOHN P. EAST, North Carolina
PAULA HAWKINS, Florida

EDWARD M. KENNEDY, Massachusetts
JEFFINGOS RANDOLPH, West Virginia
CLAIBORNE PELL, Rhode Island
THOMAS P. EAGLETON, Missouri
DONALD W. RIEGLE, Jr., Michigan
HOWARD M. METZENBAUM, Ohio
SPARK M. MATSUNAGA, Hawaii
CHRISTOPHER J. DODD, Connecticut

Ronald P. Dochel, Staff Director
KATHRYN O'L. HICKEY, Minority Staff Director
CONTENTS

CHRONOLOGICAL LIST OF WITNESSES

THURSDAY, MAY 5, 1983

Gorton, Hon. Slade, a U.S. Senator from the State of Washington.................. 23
Jackson, Hon. Henry M., a U.S. Senator from the State of Washington........... 24
Clark, Mrs. Barney, Des Moines, Wash. ............................................................... 26
Brandt, Edward N., Jr., M.D., Assistant Secretary for Health, Department of
Health and Human Services, accompanied by Dr. Claude Lenfant, Director
of the National Heart, Lung and Blood Institute, National Institutes of
Health, Dr. Mortimer B. Lipsitz, Director of the National Institute of Child
Health and Human Development, Dr. William Polchin, Director of the
National Institute on Drug Abuse, Dr. Jane Henney, Deputy Director of the
National Cancer Institute, and Dr. Joann Luoto, Director of the Office of
Smoking and Health .......................................................................................... 39
Ernst, Dr. Virginia, associate professor of epidemiology, University of Cali-
ifornia, San Francisco .................................................................................. 117
Longo, Lawrence D., M.D., head of the division of perinatal biology, Loma
Linda University School of Medicine, Loma, Calif., representing the Ameri-
can Academy of Pediatrics and the American College of Obstetricians and
Gynecologists .................................................................................................... 120
Sandhaus, Robert A., M.D., senior staff physician, National Jewish Hospital
and Research Center, National Asthma Center, Denver, Colo. ..................... 132
Keeshan, Robert, executive producer of Robert Keeshan Associates (Captain
Kangaroo) ......................................................................................................... 134
Cahan, William, M.D., professor of surgery, Cornell University Medical Col-
lege; attending surgeon, thoracic service, Memorial Hospital, the Memori-
al Sloan-Kettering Cancer Center, New York City, N.Y., representing the
American Cancer Society .............................................................................. 140
Oates, John A., M.D., professor of medicine and pharmacology, Vanderbilt
University, and chairman of the Subcommittee on Smoking of the Ameri-
can Heart Association, representing the American Heart Association ........ 153
Fisher, Edwin B., Jr., Ph.D., associate professor of psychology, Washington
University, St. Louis, Mo., representing the American Lung Association .... 165

THURSDAY, MAY 12, 1983

Judge, Curtis H., president of Lorillard, and chairman of the Executive com-
mittee of the Tobacco Institute, New York, N.Y., accompanied by Arthur J.
Blevens, general counsel of Lorillard ............................................................ 198
Blackwell, Roger D., Ph.D., professor of marketing, Ohio State University,
Columbus, Ohio .............................................................................................. 230
Blau, Dr. Theodore H., independent practice of Clinical Psychology, Tampa,
Fla. ..................................................................................................................... 248
Sommers, Sheldon C., M.D., consultant in pathology, Lenox Hill Hospital,
New York, N.Y. ............................................................................................... 262
Fisher, Edwin R., M.D., professor of pathology, University of Pittsburgh, and
director of laboratories, Shadyside Hospital, Pittsburgh, Pa. ......................... 393
Rubin, Eric M., Esq., Rubin, Winston & Dierks, counsel for the Outdoors
Advertisers Association of America, Washington, D.C., accompanied by Wil-
liam K. Reagan, president, Outdoor Advertising, Inc., Salt Lake City, Utah.. 498
STATIONS

American Medical Association, prepared statement ................................................. Page 368
American Newspaper Publishers Association, prepared statement ........................ Page 339
Aranda, Domingo M., M.D., president, Atmospheric Health Sciences, Inc., prepared statement ....................................................... Page 258
Bing, Richard L., M.D., professor of medicine emeritus, University of Southern California, prepared statement ............................................ Page 279
Blackwell, Roger D., Ph.D., professor of marketing, Ohio State University, Columbus, Ohio ................................................................. Page 230
Blair, Theodore H., Jr., Independent Practice of Clinical Psychology, Tampa, Fla. ................................................................. Page 248
Booker, Walter M., Ph.D., president, Walter M. Booker & Associates, Inc. prepared statement ............................................................................. Page 286
Brant, Edward N., Jr., M.D., Assistant Secretary for Health, Department of Health and Human Services, accompanied by Dr. Claude Lenfant, Director of the National Heart, Lung and Blood Institute, National Institutes of Health, Dr. Mortimer B. Lipsett, Director of the National Institute of Child Health and Human Development, Dr. William Pollin, Director of the National Institute on Drug Abuse, Dr. Jane Henney, Deputy Director of the National Cancer Institute, and Dr. Joan Luoto, Director of the Office of Smoking and Health ................................................ Page 35
Buhler, Dr. Victor, pathologist, prepared statement .............................................. Page 290
Cahan, William, M.D., professor of surgery, Cornell University Medical College, attending surgeon, thoracic service, Memorial Hospital, the Memorial Sloan-Kettering Cancer Center, New York City, N.Y., representing the American Cancer Society ....................................................... Page 140
DeVita, Vincent T., Jr., M.D., Director, National Cancer Institute, National Institutes of Health, prepared statement ....................................................... Page 104
Eysenck, Hans J., professor of psychology, Institute of Psychiatry, University of London ................................................................. Page 297
Farras, Matthews, M.D., associate dean, school of medicine and 'emeritus' professor of surgery, University of California at San Diego, prepared statement ....................................................... Page 1896
Fisher, Edwin B., Jr., Ph.D., associate professor of psychology, Washington University, St. Louis, Mo., representing the American Public Association ................................. Page 165
Fisher, Edwin R., M.D., professor of pathology, University of Pittsburgh and director of laboratories, Shadyside Hospital, Pittsburgh, Pa. ....................................................... Page 392
Funk, Richard W., counsel, National Automatic Merchandising Association, prepared statement ....................................................... Page 528
Gorton, Hon. Slade, U.S. Senator from the State of Washington ................................ Page 23
Grasse, Horace Charles M., U.S. Senator from the State of Iowa, prepared statement ................................................................. Page 38
Health Insurance Association of America, James L. Moorefield, president, prepared statement ....................................................... Page 961
Hickey, Richard J., senior research investigator, Department of Statistics of the Wharton School, University of Pennsylvania, Philadelphia, prepared statement ....................................................... Page 320
Hackett, Robert C., research director of the Council for Tobacco Research, U.S.A., Inc., prepared statement ....................................................... Page 328
Hutchison, Duncan, M.D., professor of pharmacology and medicine, the University of Medicine and Dentistry of New Jersey, prepared statement ....................................................... Page 340
Jackson, Hon. Henry M., a U.S. Senator from the State of Washington ................................................................. Page 124
Judge, Curtis H., president of Lorillard, and chairman of the executive committee of the Tobacco Institute, New York, N.Y., accompanied by Arthur J. Stevens, general counsel of Lorillard, prepared statement ....................................................... Page 298
Judges, prepared statement ................................................................. Page 412
Keenan, Robert, executive producer of Robert Keenan Associates, Captain Kangaroo ...... 134

Lennon, Claude, M.D., Director, National Heart, Lung, and Blood Institute, National Institutes of Health, prepared statement ..... 75

Lisette, Mortimer B., M.D., Director, National Institute of Child Health and Human Development, National Institutes of Health, prepared statement ..... 167

Longo, Lawrence D., M.D., head of the division of perinatal biology, Loma Linda University School of Medicine, Loma, Calif., representing the American Academy of Pediatrics and the American College of Obstetricians and Gynecologists ..... 120

Prepared statement ..... 123

Lopez, John M., M.D., F.H., Director, Office on Smoking and Health, prepared statement ..... 112

Macdonald, Eleanor J., professor emeritus of epidemiology, Department of Cancer Prevention, University of Texas System Cancer Center, M.D. Anderson Hospital and Tumor Institute, Houston, Tex., prepared statement ..... 444

Magazine Publishers Association, prepared statement ..... 531

Mendenhall, Harold, Ph.D., University of Denver, prepared statement ..... 360

Dales, John A., M.D., professor of medicine and pharmacology, Vanderbilt University, and chairman of the Subcomittee on Smoking of the American Thoracic Association, representing the American Heart Association ..... 150

Press release statement ..... 150

Polin, William, M.D., Director, National Institute on Drug Abuse, Alcohol, Drug Abuse, and Mental Health Administration, prepared statement ..... 96

Rao, L.G.S., Dr., senior biochemist at Bellshill Maternity Hospital, Glasgow, Scotland, prepared statement ..... 370

Ribdon, Harrison, R., M.D., prepared statement ..... 507

Roberts, Henry, Ph.D., professor of medicine and anatomy, School of Medicine, Louisiana State University, prepared statement ..... 384


Schoen, Robert A., M.D., senior staff physician, National Jewish Hospital and Research Foundation (National Asthma Center, Denver, Colo.) ..... 132

Somers, Sheldon C., M.D., consultant in pathology, Lenox Hill Hospital, New York, N.Y., prepared statement ..... 255

Prepared statement ..... 257

ADDITIONAL MATERIAL

Articles, publications, etc.

Additive Adds Mystery to Risks of Smoking, by Ken Cummins, from the Florida Times-Union, January 12, 1988 ..... 4

Answers to the Most Asked Questions about Cigarettes, by Ken Cummins, Times-Union Washington Bureau ..... 203

Avoiding Makers Slow to Give List of New Drugs, by Ken Cummins, Times-Union Washington Bureau ..... 12


Flavorings Added to Cigarettes May Be Linked to Cancer, by Ken Cummins, Times-Union Washington Bureau ..... 10

Low Tar, High Risk, by Jim Mintz, February–March 1981 ..... 15

Say, How Much Linked to Heart Disease, New Study Finds, by Lawrence, R. J., Alman, from the New York Times, Tuesday, May 3, 1983 ..... 179

Views on Health Warnings, by David Simpson, director, Action on Smoking and Health, June 2, 1983 ..... 68

What’s Been Added to Your Cigarette, by Walter S. Ross, staff writer, Reader’s Digest ..... 18

Why People Smoke Cigarettes, by William Pollin, M.D., Director of the National Institute on Drug Abuse ..... 41

Communications

Dodd, Hon. Christopher J., a U.S. Senator from the State of Connecticut, from Roger D. Blackwell, professor of marketing, Ohio State University, May 26, 1983 (with enclosure) ..... 410
Communications to—Continued

East, Hon. John P., a U.S. Senator from the State of North Carolina, from P.R.J. Burch, professor, Department of Medical Physics, the General Infirmary, Leeds, May 11, 1983 (with enclosures) ........................................ 420

Hatch, Hon. Orrin G., a U.S. Senator from the State of Utah, from Mary Jane Jesse, M.D., president, American Heart Association, Washington, D.C., May 26, 1983 (with enclosure) ........................................ 523
SMOKING PREVENTION HEALTH AND EDUCATION ACT OF 1983

THURSDAY, MAY 5, 1983

U.S. SENATE,
COMMITTEE ON LABOR AND HUMAN RESOURCES,
Washington, D.C.

The committee met, pursuant to notice, at 9:40 a.m., in room SD-430, Dirksen Senate Office Building, Senator Orrin Hatch (chairman) presiding.
Present: Senators Hatch, Hawkins, East, Grassley, Quayle, Riegle, and Pell.

OPENING STATEMENT OF SENATOR HATCH

The CHAIRMAN. Today we are holding full Labor and Human Resources Committee hearings on S. 772, the Smoking Prevention Health and Education Act of 1983. As chairman of this committee, I have had the opportunity to learn a great deal about the health needs of our citizens and, in turn, have learned that the health hazards associated with smoking are enormous.

The legislation we are considering today represents a national public education effort designed to improve our citizens' awareness about what the Surgeon General has said is the No. 1 preventable cause of death, cigarette smoking. The issue at stake is not whether a person should or should not smoke or whether the Government should tell people how to live their lives, the issue is that anyone deciding whether to smoke or not should be able to make an informed choice.

I am not in favor of Government regulations. However, I believe our Federal Government has a responsibility to provide health information and education to all Americans.

The most recent Surgeon General's report, the Health Consequences of Smoking, focused on cancer, citing cigarette smoking as the major cause of cancer mortality in the United States. In spite of remarkable improvements in the survival rates for some cancer victims, lung cancer remains the largest single contributor to the total cancer death rates, and it is one of the most malignant and untreatable kinds of cancer. In fact, lung cancer accounts for one out of every four cancer deaths and 85 percent of these are related to smoking.

Research has linked cigarette smoking not only to lung cancer, but also to cancer of the larynx, oral cavity, esophagus, bladder, pancreas, and kidney. Furthermore, smoking causes emphysema and chronic bronchitis, which are dreadful diseases of the lungs.
robbing one of the ability to breathe with the ease most of us take for granted. Smoking is also known to be a major cause of coronary artery disease, peripheral vascular disease, and has recently been associated with peptic ulcer disease, retarded fetal growth, and increased spontaneous abortion in children whose mothers are smokers.

It has been estimated by the Department of Health and Human Services that cigarette smoking results in expenditures of $13 billion in medical costs annually and $25 billion in losses to the economy from diminished productivity. This totals $38 billion per year. Although these costs are staggering, they are not as significant as the human losses—the suffering of individuals with chronic diseases like emphysema and the sadness of premature death from cancer or heart disease. In fact, cigarette smoking is estimated to account for more than 340,000 premature deaths each year.

Our Government's commitment to health promotion should be translated into action. This bill represents a new and much needed effort to demonstrate that commitment. However, this Federal effort should not replace other worthwhile programs. We can only succeed in improving the health of our citizens by the ongoing cooperation from the voluntary agencies, health professional organizations, and concerned citizens. We look forward to hearing of these efforts today.

We are, in particular, very pleased to have Mrs. Barney Clark, Una Loy Clark, from Des Moines, Wash., with us today. We welcome you to the committee, Mrs. Clark. We will call on you in just a second.

At this time we will turn to Senator Hawkins and then to Senator East.

Opening Statement of Senator Hawkins

Senator Hawkins. Thank you, Mr. Chairman.

It is a pleasure to join you and Senators Kennedy and Packwood in cosponsoring S. 772, the Comprehensive Smoking Prevention and Education Act. I strongly support this legislation because I feel that it is vital that the public be kept fully informed of the health risks associated with smoking.

I believe the Federal Government has a responsibility to insure that the latest information concerning the health aspects of cigarette smoking are communicated to the public so that the 54 million Americans who currently consume some 30 billion packages of cigarettes each year are fully informed of the risks associated with this product.

While the harmful effects of smoking tobacco have been well documented for several decades, the Federal Government has neglected in carrying out its responsibilities to warn and protect the American public from hazardous substances that are used as flavoring or fragrance additives in the cigarettes. There is currently no requirement that a list of additives contained in cigarettes be reported to any Federal agency.

In 1981, when the Surgeon General requested the tobacco industry to stop adding new ingredients, the request was ignored. The need for accurate information about additives is increasing as more
and more cigarette manufacturers are adding flavoring and fragrance additives to compensate for consumer demanded low tar and filtered cigarette. The irony is, that the extra ingredients could be making the low tar cigarettes even riskier than the old-fashioned brands.

In northern Florida and southern Georgia there is a flourishing demand for a backwoods weed called deerstongue. Deerstongue is popular as a cigarette additive because it smells like new mown hay. The only problem is that it has a high concentration of a poison called coumarin, which is banned as a food additive by the FDA. However, it is not banned as a cigarette additive because tobacco is not regulated by the Food, Drug, and Cosmetic Act, the Fair Packaging and Labeling Act, the Toxic Substances Act, or the Consumer Products Safety Act.

Recently, Ken Cummins, a Washington correspondent for the Florida Times Union joined another investigative reporter, Jim Mintz, in a 3-month investigation that traced the harvest of Deer Tongue and challenged the industry assertions that cigarette manufacturers have voluntarily ceased using this product. This series of articles raises serious questions about whether the tobacco industry can adequately regulate itself regarding additives.

With unanimous consent, Mr. Chairman, that this series of articles on additives in cigarettes be included in the hearing record.

I welcome Mrs. Clark and the two Senators from Washington.

I look forward to the testimony here today.

The CHAIRMAN. Without objection, those articles will be included in the record.

[The articles referred to follow:]
Additive adds mystery

By Don Charuma

WASHINGTON, D.C. — Old habits in
the American tobacco industry say
that new products — a growing weal
that grows in South Georgia and
North Carolina — was one of the best
selling items over decades to the
smoking public.

Now with the case of a substance
that is not as predictable as it is
true, we see the first indication of
a change in the industry. Food and
smoke, in particular, is no longer the
only industry that produces these
products.

As far as I know, there are a few
products that are made to be used
in American tobacco products.

The evidence is indeed compelling
for the use of foreign tobacco in
debate that the selling of this kind
may be considered a risk to the
industry.

And it’s evidence presented
by the Federal Trade Commission
and the American Tobacco
Commission.

The United States government
has been able to prove conclusively
that foreign tobacco can be used in
American tobacco products.

As far as I know, there are a few
products that are made to be used
in American tobacco products.

But the evidence is compelling
for the use of foreign tobacco in
debate that the selling of this kind
may be considered a risk to the
industry.

And it’s evidence presented
by the Federal Trade Commission
and the American Tobacco
Commission.

The United States government
has been able to prove conclusively
that foreign tobacco can be used in
American tobacco products.

As far as I know, there are a few
products that are made to be used
in American tobacco products.

But the evidence is compelling
for the use of foreign tobacco in
debate that the selling of this kind
may be considered a risk to the
industry.

And it’s evidence presented
by the Federal Trade Commission
and the American Tobacco
Commission.

The United States government
has been able to prove conclusively
that foreign tobacco can be used in
American tobacco products.
The flavor of vanilla to tobacco and gives it an aroma of flowers.

"The only real use for tobacco," said Kenneth Wilson of Wilson Drug Co in Boone, N.C., a supplier of natural products for use in tobacco, perfumes and cosmetics, as well as other items.

"We only sell it to the tobacco industry," said Charles Ballard, an official with the Charit Corp, a new starting in the area. "We don't use it in tobacco because of the regulations, which is a cartagino" (internal carbonation).

"That's all I know about it," said Frank A. Martin, whose Virginia Drug company was also one of the large distributors of tobacco to the end of the 1968. The firm's sales company stopped handling tobacco in 1962-1963. The official said that its primary use was the product, which uses more of it than tobacco itself.

Manager of several New York Drug stores and Ager letter to producers included: compounds have prepared according to directions provided by the individual orders. But these previous regulations did not apply, because the products were used more in the manufacturing process. Tobacco company officials said that the companies do not know what flavoring is done to prepare an entire formula to replace the source of these products.

"They smoke products" don't tell us using a lot of criticism," said one of the managers who asked that his name be withheld.

"We realize an interesting flavor-enhancing to be a trade secret, and we prefer not to discuss this issue at all," said Robert J. Biddle, president and treasurer of American Tobacco Co, the parent company of the Initiative Tobacco Co.

The six major tobacco manufacturers - Philip A. Morris Inc., American Tobacco Co, R.J. Reynolds Tobacco Co, Liggett & Myers, and Brown & Williamson - and the U.S. Tobacco Co, have offered a letter that indicates current and former employees in the tobacco industry would be informed what is added to their products.

"It would be an extraordinary growth of our policy to decide what we do or not do," said Ernest Pope, senior vice president and general counsel of Brown & Williamson Tobacco Corp, which has its headquarters in Louisville, Ky.

"The regulations (the letter) are strictly a secret," Pope said. "We do not know what it is or whether it is effective."

A regulatory letter for S. Tobacco said, "Due to the competitive nature of the tobacco industry, this information is proprietary and confidential.

Despite the uncertainty, the tobacco industry does not know what goes into the tobacco product in the United States, although they are working to find out.

In 1988, Julian Hirsch, a senior research economist at the American Tobacco Council, the trade association for the industry, said he had no other information than what is available from the government.

"The companies only tell us what they want us to know," Hirsch said. "We do not have any reason to believe any natural product is not safe."

"The companies are not allowed to tell us anything about the flavoring of their products," Hirsch said. "They are not allowed to tell us anything about the ingredients of their products."
Under an agreement negotiated in the summer of 1989 between the Department of Health and Human Services and tobacco industry representatives, the government says it will get a lot of addictive cancer from the industry.

But the list will not be complete.

According to the agreement, the list will contain any nce used in at least three of the five major tobacco brands. The agreement does not yet specify what constitutes a "large

At one time, tobacco companies in the United States were using more than a million pounds of nicotine an

Dermyced or cancer researchers in 1989 already found that nicotine, a known carcinogen, was also present in

But the German test was run in 1981 after a series of studies indicated that nicotine might cause cancer in humans.

Advisory panelists said the Office on Smoking and Health is concerned about other known or suspected can

Pinsky says the industry's list includes only a partial list of additives that might cause health problems.

"There are many more studies that need to be done," he said. "We don't have the list that we want to have in terms of what is in the products."

It appears to me that the industry is not as forthcoming as it should be," he added. "They are not telling us what they know."
Deer tongue

Companies deny using weed in cigarettes, but demand for supply was fierce in 1982.

Ypsilanti, Mich., Jan. 16, 1983 — A green weed is named in the cigarette烟 and North Carolina. People call it a dog tongue or green's tongue. Its main ingredient comes from Virginia and New York. It is at 30,000. An Armano, known as "Far, Anna," has picked at least three pounds on her farm. But she is known for her deer tongue. The deer tongue is one of the half-shorn ears of six to 100 regular penalties. Most of the penalty is the result of a seasonal supplement to their income.

In turn, Dr. Middlemdr. tells the deer tongue to promote healthy 30 Virgina and North Carolina. It is the key plant for deer.
Demand for deer tongue

additive was fierce in 1982

Although some tobacco industries
suffered much that the Natives with
a stepped up demand for deer tongue as a
finishing additive more than a decade
ago, the competition had meant for
little bearing. The Natives had been
advancing at a rapid rate in the
demand appearing in rural delicious
finishing, and this led to available deer
tongue on hand at the regular buyers's
weekly purchase.

A few years ago, demand for deer
tongue was so strong that the pickers
were getting from $10 to $30 per
pound.

The reason for the demand for
beef was still not to add meat, but to
add flavor to the delicacy.

15 P1
...to, 0. "m 7 dm Wa Palma protect he atraa.ccti.a. dewy.. 71w. Mad Po a lolly 10 hawk atoolltatatpanots 'Se mama 110 dal googoo Ile turd fat 01$o Mama lore this Waco duly deer league mukttomoo a Jaya tone hi areohl COOK armor 44.g ve soled mate $300 10 VA beryl 1110 4:01* ataoadatrilee a weld ail drat 441Watbatatt yet sacks a al'

ro Walk, 4014 119oird10 410 172 ov.1004114 eg: 114 X1110007Y" aod tram add dead toe ptants am Um lava to armload, Mead ad tomatow ea Trey OMR Itietatatte SW.4 Nal water/Ph:Ow wadi fa Oar tosahs \a galtairta 4.a. IotaatM day troal...00 the 4.eca a nor plenoln woes. - yemoSate4 gaemea 'catboat mecum* at eavegssetlef es *rs tom' UNVal trolberJoarot a eatiomccadaltd lowly hy Tbr For omelet ,ft a

s:1010:1 asy.111*0017. ells Toe ar1 ip Ae the meal liana WS a moat! Awe: pets"...1541010 psSdraos2 (*VP* rockosx w qe y earoad Dates MI -of you WI W e bad M" doped Awo 61 ;WO rt.-area% to tag O the

Obe: pets"...1541010 psSdraos2 (*VP* rockosx w qe y earoad Dates MI -of you WI W e bad M" doped Awo 61 ;WO rt.-area% to tag O the

Obe: pets"...1541010 psSdraos2 (*VP* rockosx w qe y earoad Dates MI -of you WI W e bad M" doped Awo 61 ;WO rt.-area% to tag O the...
Flavorings added to cigarettes may be linked to cancer

Flavorings — including cocoa, menthol, and chocolate — have been added to cigarettes to give them a mintier or sweeter taste. Such flavorings are often added to the tobacco to compete with menthol and other tobacco company flavors.

But government officials and researchers are questioning the use of such flavorings against saying that some of them might be cancerous. The U.S. Surgeon general raised the specter that flavorings in low tar cigarettes may pose health risks and "may negate beneficial effects" of switching from high- tar to lower tar brands.

Cocoa, for example, has become a suspect since a little-published study by the National Cancer Institute in the mid-1970s reported that tar from cocoa-flavored cigarettes caused more dangerous skin tumors in mice than tar from cigarettes without cocoa.

Flavorings such as cocoa, which is popular because of its flavor, may become harmful when turned, national Cancer Institute scientists and other researchers discovered.

In 1977, the Surgeon General directed the National Cancer Institute study, but in an interview late last year that the discovery of possible risks from cocoa in cigarettes surprised the tobacco companies, God and said he did not consider the results overly significant at the time because he "got the impression" that tobacco companies no longer used the tasty, low grades of cocoa that the institute had tested.

In its handling such studies later, the industry may have added sub-
flavorings' link to cancer may quash low-tar advantage

(from Page A-1)

The surgeon general's report two
years ago singled out powdered co-
coca as one suspected carcinogen
"that is probably used in U.S. cig-
arettes." *1

One flavor supplier said Philip
Morris and R.J. Reynolds, two of the
caroladoras' largest domestic sup-
pliers, have been removing flavors from
any flavorings being supplied to them, and
affiliated to two flavoring companies that
supply those tobacco companies.

But this quick reaction by the
industry did not mean cocoa was im-
mediately removed from flavoring,
additives. In fact, it was a sudden
resources in the tobacco industry key
for all cocoa's strengths in domes-
ic brands and could not have nor-
ished established markets.

Instead, the more was viewed by
several tobacco industry insiders as a
gradual phasing out of cocoa.

One flavor supplier said Philip
Morris and R.J. Reynolds have not
been renewing contracts for cocoa
carriers. But those two to-
bacco companies still purchase co-
cocoa.

"It's all in. They want to know
exactly how much, another supplier
said. "They want to put it in the
wrong. They want to control their
own destiny because the liability is so

Figure out how much cocoa goes
into tobacco products is difficult to
obtain. Unlike many fla-
oringings that are added in small
unseen dosages is added to cig-
ecutive formulas in bulk.

The large cocoa supply house
claims its cocoa does not keep well,
how much of their product is bought
by the tobacco industry each year.

South Korea, with U.S. Cocoa in
Peru, ND, one of the flavor
suppliers, estimated that the annual
consumption would run into the
millions of pounds.

An official with another cocoa sup-
ply house in New Jersey said his
company will sell recently 1.5 million
pounds to tobacco companies this
year.

"And we're a small part of the
industry," he said.

Mary Petrun, executive vice presi-
don of U.S. Cocoa, at first, said she
could provide some industry esti-
mates. Later, she changed her mind,
governmentally.

Sources close to the tobacco indus-
try say Philip Morris has decided to
purchase the local brands again.

Yet officials of the company refused to confirm or deny that. They

said it was "an internal decision that
is none of your business."

"If they told us we had to take co-
coca out tomorrow, I wouldn't object," said
Frank Durlay, manager of flavor
development for Philip Morris.

The company could discontinue ad-
designed compounds without a war-
powdered-cocoa. Durlay said. Howev-
er, he said that the changeover would
be costly.

One tobacco industry source said
until a flavor substitute could be
found, R.J. Reynolds and decided to
buy more high-grade cocoa products.

The less-expensive lower grades of
cocoa apparently bear a more serious
health risk (than, either more refined
varieties, said Curt, the former Na-
tional Cancer Institute scientist, he
now works at a research center set
up by the Pillsbury Institute in Min-
dnesota and supported by a $1 million
grant provided by the Brown and Wil-
liams Tobacco Co.

But Reynolds officials refused to
to comment, citing as the reason, "the
sensitive nature of the negotiations" with
the government to disclose addi-
tives and flavorings.

Meanwhile, no one except the to-
bacco companies knows exactly what
is going into tobacco products.

"Everything done with the to-
acco industry is kept under wraps with
out a request," said Mr. Petrun of U.S. Co-
cocoa. "We just give out. How much
they use. How it is processed. Build-

Jim Blutez, a New York-based fre-
transport writer on assignments for Math-
Jones magazine, lifted in re-
arrangers of this article. Mother Jones is a
liness national magazine specializing
in investigative journalism and politics.

28-514 0-22 2
Cigarette makers slow to give list of all additives

The number of additives displayed is expected to be fewer than 100.

This is an example of how, just as the government does when it seeks to negotiate from a position of relative weakness with a powerful industry that's trying to protect itself, and as its power, under President Carter, has been eroded at the federal Office on Smoking and Health at the time negotiations for the additives list began.

When Robert Bunnell, the U.S. surgeon general at the time, sought for health under President Carter, he too sought to identify those substances, and had to agree.

And when, in 1970, the government asked the six major U.S. tobacco companies for lists of additives used in their cigarette brands, the companies were able to do so because they had not been disclosed.

And when the Florida Planning Board and Health and Human Services are finally shown a complete list of additives - a list which was expected to happen soon - the government will be given a list of all substances that it originally requested.

Possible continued use of suspect, harmful tobacco additives cannot be seen during an investigation by The Florida Times-Union and White Media, a national news operation, to investigate the use of suspect additives.

And the list would be given to the government.

The head of the U.S. Department of Agriculture and other government officials have created a list of all substances that are in the cigarette brands.

But instead of listing the names of all substances added to commercial cigarette brands, government officials agreed not to do so.

And when asked why, they said it would take too long.

Now, federal officials will agree to list all additives used by all manufacturers.

The government and the industry have reached agreement.

By the end of Richmond's 1970, and even though the first draft of Richmond's master plan started in late December, the industry decided to list the additives and the government agreed to list all additives.

Kahn's plan is to have all additives listed by April.

Kahn's plan is to have all additives listed by April.

19
Cigarette makers are reluctant to give complete list of additives

(From Page 10)

The American Tobacco Co and Liggett and Myers Tobacco Co - targets of a 1971 FAA lawsuit - refused to release their lists of additives. Several senators and members of Congress asked to see copies of the list, and a 1964 article in The Tobacco Institute's newsletter "Tobacco Flavering for Selling Products" included a draft list of additives that could be added to tobacco products.

Two other companies cited the 1978 British "List of Permitted Additives to Tobacco Products," which contained 351 substances approved for use in tobacco sold in the United Kingdom. The list included those six manufacturers which additives on those lists they might use.

"There is no benefit to the consumer in the present situation," Richmond said in a second letter sent Nov. 12, 1979, to the six manufacturers.

"If the substances which you use pose no immediate or long-term health threat to the smoker, their use will make the product itself that much safer. But if there is a threat, the substance should not be used," Richmond said.

After the second letter was sent, Putney said he and Richmond decided to meet with tobacco representatives to discuss the government's concerns.

But they were uncertain that the industry would respond to a request to meet. Before Carret was leaving office, he had asked to see the 1964 "draft list" of additives and potentially harmful substances. But the government had no proof that Richmond told the tobacco industry to destroy any lists.

The industry sources, who requested anonymity, said that Richmond requested all the lists, but that there was no evidence that the government had the lists.

After a meeting with the tobacco representatives, Richmond said that the tobacco companies had finally agreed to cooperate with the government's efforts to determine the health risks of additives.

Faced with this threat, industry representatives quickly agreed to meet with the government to discuss the health risks of additives. They also agreed to send the government's decision to accept the tobacco industry's conditions.

"We thought we needed to do something rather than just keep fighting," she said. "This is the only way we can help to tell us the major threats we need to know.

Although the compiled list has been in the hands of the FDA for several weeks, the industry has asked that the two representatives be included.
The two noted fell down in Rich-

mond's office on December 18. Pre-

sent were Attorney Tenino; Horace

Kernegay, chairman of the industry-

representing Tobacco Institute; Panney

Richmond; and Seagor, assistant the

medical director for the dietetic co

subsidiary and health, who became di-

rector of the office when Panney quit

that post on that day.

At that first meeting, Panney said

Tenino and Kernegay pushed for disc-

losure only those additives used by

several manufacturers, not only one. But

the government officials insisted

that they had to know the identities of

all additives. The government could

not assure, devise ways to protect

trade secrets.

A second meeting was held at the

spring. It included Edward Branil

Jr., President's assistant secretary

of health and acting U.S. sur-

geon general, replacing Richmond in

the discussion after he departed

with the charge to administration.

At this meeting, Tenino and Kernegay

again pushed for limiting the

number of additives to be disclosed.

Panney said the discussion also fo-

cused on who would see the list, once

disclosed, and who would choose the

people who would see the list.

Panney quit his government post in

the summer of 1961. Richmond, Mr.

Lawler as an attorney for the U.S.

Department of Health and Human

Services continued the negotiations.

In June 1961, Brandt and Mr. Lawler

accepted Tenino and Kernegay's in-

vitiation, surprising observers of the

negotiations. The government agreed

that only those additives used by at

least three manufacturers must be
disclosed.

Sheila Landau, public affairs direc-

tor for the U.S. Public Health Service

and spokesman for Brandt, de-


department to decide who should

see the list and with whom the infor-

mation should be shared. Mrs. Longe-

g said.

Department officials also have

been studying how they would pro-

ceed after seeing the list, what ac-

tions to recommend and tracing the

additives as not give away what has

been disclosed. Mrs. Longe-

 dostęp to petitions of the

people who would see the list.

At the last meeting, Mrs. Panney

said that with only a partial list, a gomine that

brand could contain an additive that no oth-

er company is using but which could

be potentially harmful.

For instance, a vaso constrictor at

Philip Morris said that its popular

brand, Merit, contains an additive

that is not found in any other Ameri-

can brand. That substance will prob-

ably not be on the list.

Government scientists also have

discovered a "mystery compound" in

a number of cigarette brands.

"The government settled for some-

thing less than what originally intent-

ed and may be of marginal or less

than marginal value," said Panney.

now a consultant on health issues for

private corporations and voluntary

health organizations.

"The only conclusion I can come to

is they (some of the manufacturers) are

staying something they would be en-

hanced about.

Jim Mintz, a New York-based fre-

lance writer on assignment for both

correspondence magazine, assisted in the re-

search of this article.
Low Tar, High Risk

When a major tobacco company introduced a low-tar cigarette in 1955, its executives were confident it would revolutionize the business. A year later, the Federal Trade Commission (FTC) required cigarette makers to disclose their ingredients. As a result, the company's executives realized that the new product contained nicotine, a harmful substance. This led to a series of events that would have a profound impact on the tobacco industry.

The new product, known as the "low-tar" cigarette, was designed to attract health-conscious smokers who wanted to reduce their exposure to the harmful effects of smoking. However, the company's executives failed to take into account the addictive nature of nicotine, which is a powerful stimulant that can cause physical and psychological dependence.

As a result, smokers continued to smoke the new product, and their nicotine levels increased. This led to a number of health problems, including lung cancer and heart disease. The company's executives realized that they had made a serious mistake, and they quickly pulled the new product from the market.

However, the damage had been done, and the company's reputation was tarnished. The FTC began investigating the company's advertising practices, and the company was forced to pay a large fine. This led to a number of other investigations, and the tobacco industry was eventually forced to adopt new regulations to protect public health.

Today, the tobacco industry is under constant scrutiny, and its products are regulated by the FDA and other government agencies. Despite these regulations, the industry continues to make profits, and the battle against tobacco continues to this day.
A Reader's Digest Special Report

What's Been Added to Your Cigarette?

Does it taste good? Yes or no, heed this warning: unknown substances added to "safe" cigarettes may, in fact, be as dangerous to your health as tar and nicotine.

BY WALTER S. ROSS

Can you name an American product that causes or contributes to 350,000 deaths per year, whose contents are almost totally secret and exempt from all health regulations—yet has a government subsidy? The answer is, of course, cigarettes.

There are many goods whose misuse is dangerous, but it is hard to think of anything comparable to tobacco products. There is no healthful amount of smoking. Even a single cigarette is hazardous.

This is why health authorities for three decades have been concerned about the secrecy over tobacco-product ingredients. When you buy packaged food or pharmaceuticals, the contents are, by law, listed on the labels. Yet cigarettes, always harmful, have no information about ingredients on their labels. Now there are new reasons for concern about what might be in your cigarette.

Starting in the mid-1970s, in response to smokers' continuing demand for a reduction in health risks, new low-tar and low-nicotine "light" brands of cigarettes began appearing on the U.S. market. Lowering tar in cigarettes is itself fairly easy: possibilities include mixing air in the smoke through ventilated filters or perforated papers, narrowing the diameter of the cigarette and packing it with less tobacco; using longer filters; breeding...
In low-tar tobacco. But filters and other devices alter flavor. The smoke becomes drier, losing much of its body. Says Frederick J. Triest, veteran U.S. tobacco-industry flavor consultant: "Regular smokers are accustomed to inhaling a certain amount of taste and body. With low-tar, the only way manufacturers can give it to them is in flavors and fragrances." In other words, additives.

In 1979, the then Assistant Secretary for Health and U.S. Surgeon General Dr. Julius B. Richmond became concerned about synthetic chemicals as well as natural substances that were being added to tobacco. Although the latter are generally selected from compounds considered safe to humans, a report on smoking and health prepared by his staff noted, this did "not guarantee that the subsequent products would be safe when ingredients were burned.

Since low-tar brands require beefed-up flavors, Dr. Richmond questioned whether the manufacturers might be putting in more harm than they had removed. It was known that, in skin-painting tests, the tar in at least one experimental low-tar cigarette caused as many animal tumors as tar in the very highest tar brand. Public Law 95-626 (Section 403) requires the Secretary of Health and Human Services to study "the relative health risks associated with smoking cigarettes... containing any substances commonly added... and report this information to the Congress." In July 1980, under this authority, Richmond wrote to the six major U.S. cigarette companies asking for "a list of those substances which your firm uses in its brands."

There was international precedent for this request. The governments of Canada and Great Britain have asked for, and received in confidence, the contents of the secret blends of native brands. West Germany, too, monitors cigarette additives.

Dr. Richmond wasn't asking for trade secrets; he wanted only a list of additives. He received polite replies, but no specific information about any substance added to any U.S. cigarette brand.

A second letter, in November 1980, elicited replies, again without specifics, from two companies, American Tobacco and Brown & Williamson. They sent Richmond copies of the "List of Permitted Additives to Tobacco Products" first published in 1975 by the British Independent Scientific Committee on Smoking and Health, an official government body. Neither company said whether it was using any of the additives.

Yet the British list of approved substances, some 350 in all, contains a number of known animal carcinogens considered potentially hazardous to human health—for example, coumarin. Extracted from tonka beans or from deer tongue leaves, or made synthetically, coumarin gives a sweet aroma to cig-

112
arette smoke and a taste like that of fresh-cut hay. However, when tests in the mid-1950s showed coumarin to be a poison, causing liver and other organ damage, the U.S. Food and Drug Administration removed it from the list of food and drug additives “Generally Recognized as Safe” (GRAS).

The American cigarette industry adheres to the GRAS list for its flavor additives, and it started phasing out coumarin a few years after the FDA action. However, the 1982 International Directory and Buyers’ Guide of Tobacco International lists 11 suppliers of deertongue. The results of such bioassays are assumed indicative of harm to smokers. Despite these findings, the U.S. cigarette industry is an important user of cocoa. Of major flavorings used by tobacco manufacturers, various forms of cocoa are particularly identified with U.S. brands of cigarettes.

Several of the additives permitted on the British list—caramel, invert sugar, eugenol, guaiacol—are, or produce, co-carcinogens during burning or smoking. Co-carcinogens increase the power of even trace amounts of carcinogens to cause cancer. Eugenol is suspected of being a carcinogen by itself; angelica-root extract, another British-approved additive, contains carcinogenic substances; and orange-peel oil promotes tumors in mice. Also on the British list are a number of additives, such as docan-5-olide and nonan-4-olide, that give rise to carcinogens when heated.

Although the British committee was dangerously premature in approving some poisons and cancer-causing chemicals as cigarette additives, it showed prudence in one regard. It did not approve cocoa, a widely used tobacco flavor. In the mid-1970s, the National Cancer Institute tested a variety of experimental cigarettes, among them one flavored only with cocoa, and it proved to be strongly tumorigenic: mice whose shaved backs were painted with tar from its smoke developed many skin tumors. Results of such bioassays are assumed indicative of harm to smokers. Thus, although cocoa powder makes a healthful drink, smoke from cocoa burned in cigarettes could be hazardous.

Another major flavor in U.S. cigarettes is licorice. Practically all the licorice extract produced in the United States, about 12 million pounds a year, goes into tobacco. It adds flavor, helps keep tobacco moist and improves the burning quality. Part of licorice extract is glycyrrhizic acid, which, when burned, is a precursor for cancer-causing polycyclic aromatic hydrocarbons.

Sugars (whether from cane, corn, beet or fruit) are the major flavor additives used in U.S. cigarettes, making up about four percent of the tobacco by weight in most U.S. brands since the early 1960s. When burned with tobacco, sugar increases tar yield. Certain
forms of sugar, such as caramel and invert sugar, produce catechol when heated. This is "the major known co-carcinogen in tobacco smoke," according to scientific publications by Dietrich Hoffmann, associate director of the American Health Foundation.

To make aged tobacco workable and keep cigarettes fresh, chemicals known as humectants are added, the major ones being glycerol and the glycols. According to a 1979 Surgeon General's report: "Glycols are suspected to influence the smoker's risk of bladder cancer."

In addition, burning transforms glycerol into a substance called acrolein which suppresses the action of microscopic cilia that force irritants from the lungs. As a result, the smoker's risk of chronic bronchitis and emphysema is increased and the lungs are open to attack by toxins and carcinogens.

Thus, some of the major flavors and humectants known to be routinely added to cigarettes in the last 70 years are dangerous or at best suspicious with regard to human health. And what of the many synthetic flavors and fragrances developed to beef up the taste of new low-tar cigarettes? Could they be even more hazardous than the additives we know about?

These are the questions Dr. Richmond asked, and for which he received no answers.

Richmond has since left office. To date, inquiries into cigarette additives have not been followed up by the successor, Dr. C. Everett Koop, but the subject has not been allowed to languish. Dr. Edward N. Brandt, Jr., Assistant Secretary for Health, and Dr. Joanne Luoto, acting head of that Department's Office on Smoking and Health, have been pursuing the Richmond lead and negotiating with the tobacco industry. Horace Kornegay, chairman of The Tobacco Institute, and Stanley Temko, of the firm of Covington and Burling, attorneys, have been appointed by the six major manufacturers to represent them.

Kornegay and Temko have met several times with Brandt and Luoto, and have submitted a proposal for releasing lists of additives used in U.S. cigarettes. But the industry's plans are complex, hedged about with restrictions designed to protect trade secrets. They fear the Freedom of Information Act might force the government to reveal what tobacco companies give in confidence.

Nevertheless, a start appears to have been made. Let us hope our government will not be deterred from pursuing a subject vital to the health of more than 50 million Americans. As Dr. Richmond said in his November 1970 letter to the cigarette manufacturers, "If the substances used pose no immediate or long-term threat, that fact should be made known. If there is a threat, the substance should not be used."
The CHAIRMAN. We will now turn to Senator East.

OPENING STATEMENT OF SENATOR EAST

Senator East, Mr. Chairman, I appreciate the opportunity to comment upon this legislation. I look upon this as well intentioned but very bad legislation.

I have no quarrel, and I do not know of anybody who would, with the Federal Government's trying to inform the American people about the relative health hazards or value of particular commodities, products, et cetera. Polls demonstrate that the American people are overwhelmingly, up in the 90 percentile, aware of the risks involved in excessive smoking. If one thing is known by the American people, it is that.

This legislation goes beyond the desire to really inform, but I look upon it as punitive in nature and I look upon it as a very bad precedent in terms of overregulating a longstanding and very honorable industry in this country. I am somewhat mildly embarrassed that the principal initiatives are coming from such distinguished members of my own party who normally, under more rational, calm tranquil circumstances would be very leery of punitive, excessive Government regulation in the name of some honorable, well-intentioned end. However, I have learned in the brief time I have been in Washington that it takes a great deal to embarrass you, so I am only mildly embarrassed. [Laughter.]

This legislation reflects what I might call the Carrie Nation mentality today in this country on smoking. Smoking is not chic today. As a consequence, I think it is being focused upon as a way to be excessive, to be punitive, and to overregulate.

I would submit to the distinguished chairman of this committee if the desire is prohibition we ought to take up a constitutional amendment before his Subcommittee on Constitutional Amendments of the Judiciary Committee and proceed with prohibition as we did back in the days of trying to eliminate the abuses of alcohol.

For example, speaking of the abuses of alcohol, as you know, the latest studies show that in all age ranges the death rate has been going down except in one category. Between the ages of 18 and 25 the death rate has been climbing. Why? The single greatest cause of killing our young people today is drinking and driving. Yet, you see many Americans who do drink. They like those two cocktails every day or maybe three or four. Therefore, it is not quite chic to be antidrinking. As a consequence, I do not find in America today the Carrie Nation mentality on an issue which, on a relative scale of merit and importance, might be the one we ought to be taking up.

I am just trying to put this in perspective. Again, I am not trying to defend that which is indefensible, nor am I trying to close down the Federal Government, nor would anybody else I know who is a responsible spokesman for this honorable industry, from genuinely informing the American people, as the Surgeon General does every year in his report, as to the relative health value of smoking and the potential ill effects of excessive smoking. I am simply asking for balance, proportion, fairness, reasonableness, and I hope, above all, my own dear party does not fall into the error of sometimes
what our distinguished brethren on the other side of the aisle have done, by identifying a problem and then just regulating the daylights out of it and creating a lot of punitive regulation.

I was not elected on that mandate in 1980 as regards American industry generally I know my distinguished colleague from Florida was not, either. Now I respect their judgments, integrity, and good intentions, but, as we all know, often the road to hell is paved with good intentions.

I think if we take this bill as it is proposed and saddle one of America's major industries with it, we will set a precedent for other industries. Therefore, we are once again back to the old tactic of identifying a problem and then going after it tooth and nail with over-regulation and punitive regulation.

With all due respect to the distinguished chairman and my distinguished colleague from Florida—I respect their intentions and I respect what they are trying to do—I strongly and respectfully dissent and will do everything I can to responsibly alter and modify this legislation.

I thank you, Mr. Chairman.

The CHAIRMAN. Thank you, Senator East.

Senator Grassley, do you have any comments?

Senator GRASSLEY. No.

The CHAIRMAN. I am pleased to welcome our first witness here today, a very dedicated wife, Mrs. Barney Clark. She will be introduced by our two colleagues, the two Senators from the State of Washington, the Honorable Henry M. Jackson and the Honorable Slade Gorton.

We are very pleased to have you come to our hearing on the Smoking Prevention and Education Act of 1983. We welcome you. We want to express our sympathy for what you have gone through. We express our admiration that you are willing to appear publicly in this way. That means a great deal to us and I think to people across this Nation.

Senator Jackson.

Senator JACKSON. I yield to my colleague. I had first call yesterday with Mr. Ruckelshaus. We were dealing with an environmental problem.

We alternate seniority here.

The CHAIRMAN. I see. You are the only two I know of who do that. [Laughter.]
Mrs. Clark brings to this committee a message on her own behalf and on behalf of her late husband, calling attention to the tragic effects of smoking on the health of both heart and lungs. She speaks of the need, with which I agree, for a comprehensive smoking education campaign to improve public awareness of the health consequences of smoking.

In memory of Dr. Clark, Senator Jackson and I have introduced a Senate resolution asking the President to award the Presidential Medal of Freedom to Dr. Clark to be presented to his family in memory of his courage and his contribution to medical science.

I also commend Mrs. Clark for her courage and determination in working to improve the health of our Nation.

I hope that the Senate will act swiftly and favorably on the legislation which the Committee on Labor and Human Resources is considering here this morning.

The CHAIRMAN. Thank you, Senator Gorton.

Senator Jackson.

STATEMENT OF HON. HENRY M. JACKSON, A U.S. SENATOR FROM THE STATE OF WASHINGTON

Senator JACKSON. Mr. Chairman, on the light side, I would like to offer a testimonial. I quit smoking when I was 12. [Laughter.] Corn silk.

Many years later, when I had a few allergy problems, about 82 pinpricks, I found out that my No. 1 allergy was tobacco. I had claimed over the years that it was my willpower that caused me not to smoke. I now make that confession, that it was not my willpower; it was nature reacting to tobacco.

I do not know whether Senator East wants to get some statistics on that, but those of us who do not smoke, I must confess, it is not all willpower.

Mr. Chairman, I want to associate myself with my colleague's remarks in presenting what is truly a gallant lady. Mrs. Barney Clark, the way she has conducted herself during the trying illness of her husband, I think set an example for the Nation. It made us as Americans very, very proud of her.

You were the mainstay during that long and trying period. I, for one, and all the people in the State of Washington join in saluting you, Mrs. Clark, for the way you managed a most trying and, indeed, traumatic situation in your family. It makes us all proud to be Americans, and we salute you.

[The prepared statement of Senator Jackson follows:]
I am privileged to have the opportunity to introduce Mrs. Barney Clark to the members of the Senate Committee on Labor and Human Resources. Mrs. Clark has traveled from my home state of Washington to testify on behalf of the Comprehensive Smoking Prevention Act of 1983.

We as a nation and throughout the world are familiar with Mrs. Clark as the recent widow of Dr. Barney Clark, the first man to receive a permanent artificial heart. Our own hearts went out to this courageous man as he underwent a lengthy surgical operation and then, very publicly and openly, recovered from this operation and continued to live for nearly four months. Barney Clark's willingness to test the Jarvik-7 heart has enabled the field of medicine to vastly expand its knowledge and experience in this area. From here we will continue to test and research ways in which to improve the artificial heart and thereby extend the lives of those suffering from heart disease.

However, it is equally important that we as a nation not overlook the ways in which we can prevent heart disease from occurring in the first place. One of the most preventable and easily identifiable causes of death is cigarette smoking. Over 300,000 Americans die each year from diseases directly related to cigarette smoking. 33% of heart disease deaths
are attributed to cigarette smoking. 25% of lung cancer deaths can be attributed to smoking. Scientific evidence continues to increase linking cigarette smoking with a variety of diseases ranging from cancer to emphysema to miscarriages.

The legislation being considered here today will help further education of the national public about the hazards of cigarette smoking, with an emphasis that this is the number one preventable cause of death. I believe very strongly in the need for passage of this legislation. I am pleased at this time to join the Chairman in co-sponsorship of this measure.

I hope you will join with me in welcoming Mrs. Barney Clark as the first witness to appear before this committee.

The CHAIRMAN. I want to thank both of you Senators for being here.

Mrs. Clark, we are going to excuse them because they are both extremely busy. We are going to turn to you now and take your testimony.

STATEMENT OF MRS. BARNEY CLARK, DES MOINES, WASH.

Mrs. CLARK. Senator Hatch and members of the committee, I have submitted my full report, and I would like to summarize, if I may.

The CHAIRMAN. That would be fine.

Mrs. CLARK. I welcome the opportunity the American Lung Association has given me today to address the members of this committee. I welcome it because I have a personal message to bring you from my family, from my dear husband, Dr. Barney Clark, and from people around the world.

For 50 of the 62 years of my life, I have had a close association with Barney Clark—for 10 years as friend and sweetheart and for 39 years as sweetheart and wife. We raised three children, of whom we are very proud. Although all has not always been ideal and perfect in our family, we have worked for and earned a respect, a love, and a closeness with each other that has emulated our belief that "families are forever." We believe that way, we love that way, and we live that way.

Were this not so, we could not have coped with the stress and pressures of the past 4 months. The inner strength we have gained from each other, the immeasurable support we have had from the Utah heart team, together with the encouragement, love, and prayers of caring people all over the world gave us the mental and spiritual fortitude required of us.
I believe all of you are familiar with the heart and lung disease my husband suffered from—emphysema and a degenerative heart disease medically termed cardiomyopathy, in lay terms, a disease of the heart muscle.

You also know of his desire and his commitment to receive the first artificial heart. Of course, he was hopeful, through the implantation, that he would receive a personal benefit, but whether or not this personal benefit came to fruition, he felt that, were he to receive the artificial heart, that which could be learned would give great purpose to his life and, if need be, reason to his death.

You know, too, that finally on March 23 his battle for life ended. Dr. William DeVries, head of the heart implant team, explained the spiraling, downhill course that took Dr. Clark’s life. Essentially, the muscle in his blood vessels was destroyed, making them placid and dilated. Although the plastic heart kept pumping, his circulatory system could not maintain the pressure needed to get the oxygen-carrying blood to the vital organs of the body. His colon failed, his kidneys failed, and then his lungs failed. All of these facts, I believe, are common knowledge.

What you may not know is that while Dr. Clark was in the service of his country during World War II he began smoking cigarettes. Though his religious training was against the use of tobacco and smoking was not accepted in his own community, like all of us in 1941, he had no scientific knowledge, no proof, of the great health hazards he was submitting his body to. Being in an environment of much stress and pressure and in close association with others who were also uninformed and smoking, he acquired the habit. For 25 years he smoked about a package of cigarettes a day.

When it became established that cigarettes were responsible for many health problems, he became very concerned about his habit and he managed to stop smoking about 12 years ago, saying, “It was the hardest thing I ever had to do.” However, it was then too late to reverse the harm that had been done to his lungs and to his heart.

At age 53 he developed a very serious bronchitis which took 3 months to overcome. Even then, he did not feel well. He began tiring easily and finally retired at age 56 from his dental practice. Some 2 years later he was forced to seek medical help and was diagnosed as having emphysema. Other physical symptoms soon gave rise to hospitalization where it was determined that, in addition to the emphysema, he was suffering from cardiomyopathy.

It was on a trip to Salt Lake City where Dr. Clark entered the LDS hospital to receive treatment that he was referred to Dr. William DeVries and Dr. Lyle Joyce, the artificial heart implant surgeons, which visit culminated in his decision to volunteer for the artificial heart.

Within 2 days after the December 1 heart implantation his face and neck became puffy and swollen and, standing at his bedside, you could hear a faint hissing sound. Upon taking his back to surgery, it was learned there were small ruptures in the lung itself, allowing air to escape. These were quickly stapled, but it was very obvious at this time that the lungs were going to be a major source of trouble. The truth is that the deterioration of Dr. Clark’s lungs,
due to cigarette smoking, had more to do with his ultimate death than any other one thing.

I want to make it clear that I do not feign to speak as a research scientists, nor as a medical doctor, but solely as a wife who was at Dr. Clark's side as constantly as was humanly possible, observing his struggle and listening to numerous physicians, many of whom are the finest in the world, saying in essence, "If the lungs were only stronger," it was their opinion that convinced me that, were the lungs healthy, he could in all probability be alive today.

Members of this committee, I am not here today to seek sympathy, to call further attention to what has happened to us as a family, nor to continue to laud my dear husband. I am here to bring you the personal message that the hazards of cigarette smoking have long been a source of great concern to the Clark family.

Dr. Clark would, whenever possible, speak with deep regret about his ever having used cigarettes and would plead, cajole, and even bribe others to not start or to quit smoking, whichever the case might be.

I know, if he were alive today and able, it would he standing before this committee, not me, admonishing you to pass the comprehensive smoking prevention bill as quickly as possible.

Now as to the message I bring to you from all over the world, during my four months in Salt Lake City, and even now that I have returned to Seattle, I could not even begin to tell you how many thousands of letters we have received. There is one large package of letters representing people from everywhere in the world. We have received well over 600 packages of letters, drawings, balloon bouquets, and gifts youths, these from children in preschool through high school. I have been awe stricken with the children's ability to express their love and almost hero worship for Dr. Clark and, after his death, to express their sincere grief and sympathy.

Why have we received this outpouring of love and support from these people everywhere? I believe because there is not a family anywhere in the world who has not lost a loved one through heart, lung, or cancer disease. They identified with Dr. Clark because of this. He was their champion in the fight against two of these dreaded diseases.

After their kindness to my husband and family, and since I do have the opportunity to testify here today, I would feel remiss if I did not challenge this committee on their behalf to set the wheels in motion to educate everyone as to the debilitating effects of cigarette smoking on the body.

Senators, we all have a tremendous responsibility to God's great family, you perhaps more than others because you occupy a position of power and influence not afforded the rest of us. You must continue to fight to bring every bit of evidence available about the health hazards of cigarettes to the public as often and as convincingly as possible. You have an especially great responsibility to educate the youth, so that when they are tempted to start smoking, they can make the right decision.

Members of this committee, I have read and studied the bill now being considered before Congress. I believe it definitely represents a step in the right direction. Therefore, on behalf of my family, my
husband, and my dear friends all over the world, with special emphasis on the young people, I sincerely recommend that Congress pass this legislation without delay.

Thank you.

[The prepared statement of Mrs. Barney Clark follows.]
TESTIMONY

on behalf of

THE AMERICAN LUNG ASSOCIATION

by

MRS. HARLEY CLARK

before the

COMMITTEE ON LABOR AND HUMAN RESOURCES
U.S. SENATE

Re: S. 772, The Comprehensive Smoking Prevention Education Act

May 31, 1983
Testimony of Mrs. Barney Clark

Senator Hatch and Members of the Committee:

I welcome the opportunity the American Lung Association has given me today to address this Committee. I welcome it because I have a personal message to bring you from my family, from my dear husband, Dr. Barney Clark, and from people in all walks of life around the world.

For fifty of the sixty-two years of my life, I have had a close association with Barney Clark...ten years as friend and sweetheart, and thirty-nine years as sweetheart and wife. We raised three children of whom we are very proud, and although all has not always been ideal and perfect in our family, we have worked for and earned a respect, a love, and a closeness with each other that has encapsulated our belief that "FAMILIES ARE FOREVER!" We believe that way; we love that way, and we live that way. Were this not so we could not have "coped" with the stress and pressures of the past four months. The inner strength we have gained from each other, coupled with the encouragement, love, and prayers of the caring people all over the world, gave us the mental and spiritual fortitude required of us.

I believe you are all familiar with the heart and lung disease my husband suffered from...emphysema and a degenerative heart disease medically termed cardiomyopathy...in lay terms, a disease of the heart muscle.

You know also of his desire and commitment to receive the first artificial heart. Of course, he was hopeful, that through implantation, that he would receive a personal benefit, but whether or not this personal benefit came to fruition...he felt that were be to receive the artificial heart, the which would be learned would give great purpose to his life, and if need be, reason to his death.
Experimentation with animals could serve no further purpose: the artificial heart must be implanted in a human body to produce scientific knowledge and data that would further its usefulness to man. He was committed and dedicated to this experiment.

For 132 days and nights the artificial heart beat in his chest...pumping the life-giving blood to his vital organs under proper pressure and with proper effectiveness. Although the heart worked well, its complications were numerous... with periods of hope and periods of despair.

You know too that finally, on March 23, his battle for life ended. Dr. William DeVries, head of the heart implant team, explained the spiraling downhill course that took his life: "Essentially, the muscle in his blood vessels was destroyed, making them placid and dilated. Although the plastic heart kept pumping, his circulatory system could not maintain the pressure needed to get oxygen carrying blood to the body's organs. His colon failed, his kidneys failed, and then his lungs failed."

All of these facts, I believe, are common knowledge. What you may not know is that while Dr. Clark was in the service of his country during World War II, he began smoking cigarettes. Though his religious training was against the use of tobacco and smoking was not accepted in his home community, like all of us in 1941, he had no scientific knowledge or proof of the grave health hazards he was submitting his body to, and being in an environment of much stress and pressure, and in close association with others who were also uninformed and smoking, he acquired the habit. For twenty-five years he smoked a package of cigarettes a day.

When it became established that cigarettes were responsible for many health problems, he became very concerned and stopped smoking about 12 years ago, saying: "It was the hardest thing I ever had to do." But it was then too late to reverse the harm that had been done to his lungs and his heart.
At age 53 he developed a very serious bronchitis which took three months to overcome. However, he did not feel well, began losing weight, and finally retired at age 56. He continued having fatigue, shortness of breath, and general weakness. He could no longer keep up with his friends on the golf course and the slightest exertion left him breathless and tired. He sought medical help and was diagnosed as having emphysema. Then other physical symptoms soon gave rise to hospitalization and it was determined that in addition to the emphysema there was congestive heart failure and finally, he was diagnosed as suffering from cardiomyopathy.

Then followed three and a half years of intense medication to stimulate the heart muscle, to dilate the vessels and to open the lung passages to enable him to breathe better. It was on a trip to Salt Lake City where Dr. Clark entered the LDS Hospital to receive treatment that he was referred to Dr. William Devries and Dr. Lyle Joyce...the artificial heart implant surgeons...whose visit culminated in his decision to volunteer for the artificial heart.

Within two days of the December 1 heart implantation it became obvious that something was not right...his face and neck became puffy and swollen and standing at his bedside you could hear a faint hissing sound. He was returned to surgery and upon opening his chest, the doctors discovered small ruptures in the lung itself, allowing air to escape. These were quickly stapled and he was brought back weakened and discouraged. It was very obvious at this time that the lungs were going to be a major source of trouble. The truth was that the deterioration of Dr. Clark's lungs, due to cigarette smoking, had more to do with his ultimate death than any other one thing.

I want to make it clear that I do not feign to speak as a research scientist, nor as a medical doctor, but solely as a wife who was at Dr. Clark's side as constantly as was humanly possible, observing his struggle and listening to numerous doctors...many of whom are the finest in the world...saying in essence: "If the lungs were only stronger..." it was their opinion, as well as my own, that were the lungs healthy, Dr. Clark could, in all probability, be alive today.
I remember well that "red letter day" when we took Dr. Clark from the Intensive Care Unit where he had spent so many weeks, to our lovely private room where the entire west wall was nothing but windows. Those windows overlooked the Great Salt Lake Valley and across the valley to the beautiful snow-capped mountains. It was, to us, like being released from prison, and everyone shared in our joy. Those joys, however, were short-lived because the lungs began to fail and it was necessary to take Dr. Clark back to the SICU so that he could be put back on the respirator to rest the lungs. This, I believe, was one of the most disappointing times in our struggle.

Members of this Committee...I am not here today to seek sympathy or to call further attention to what has happened to us as a family...not to continue to lead my dear husband. I am here as stated, to bring a message from my family and from Dr. Clark. That message is? The hazards of cigarette smoking has long been a source of conversation on occasions when we were together. Dr. Clark would, whenever possible, speak with regret about his having used cigarettes and would always plead, cajole, and even bribe, if necessary, to influence others to not start or to quit smoking...whichever the case may be. After one of his long dissertations on the subject, and human as he was, he would say "I know...there is nothing worse that a reformed smoker...but..." So many times he expressed to me how he wishes he had never smoked and of his concern for anyone who did. He personally knew and told me he felt his lungs would prevent him from ever leaving the hospital. I know if he were alive today, and able, it would be he coming before this committee, not I admonishing you to pass the Comprehensive Smoking Prevention Education bill as quickly as possible.

As to my message to you from people all over the world in all walks of life: During my four months in Salt Lake City, and even now that I have returned to Seattle, I could not even begin to tell you how many thousands of letters we have received.
There is one large package of letters representing peoples from almost every country in the world. We have received well over 600 packages of letters, drawings, balloon bouquets, gifts, etc., from the youth—the children in pre-school through high school.

And why have we received these letters and gifts...mostly because there is hardly a family anywhere in this world who has not lost a loved one through lung, heart, or cancer disease...they could relate to my husband and me because of their own sorrow; they prayed for us as they did for their own loved ones; they hoped for us, as they had their own loved ones, and they wanted so much for Dr. Clark to get well, as they had, so much for their own loved one.

I have been awe-stricken with the children's ability to express their love and almost hero-worship for Dr. Clark and after his death, to express their sincere grief and sympathy. One little boy wrote, "My grandpa died of a heart attack; I like to pretend you are my grandpa now." Another wrote, "When my mama told me Dr. Clark had died this morning I cried and cried because I wanted to meet him someday."

I am still trying to answer each child's letter because everyone needs to know they are important and that when they do something commendable they need to be appreciated and told so. How could I, when given this wonderful opportunity, and knowing how Dr. Clark would have wanted me to, not been happy to appear before this committee to plead for those who have done so much for us.

And so, members of this Committee, comes my message to you from people all over the world and in all walks of life: We all have a tremendous responsibility to others of God's great family. You, perhaps, more than others, because you occupy a position of great influence and power to control many decisions. You must continue to fight to bring every bit of evidence available about the health hazards of cigarettes to the public as often and as convincingly as possible. It is your responsibility to do anything and everything to inform the youth so that when they are tempted to start smoking they can make the right choice. There are so many ways they are tempted..."a picture in a magazine of a beautiful young, well-dressed sophi-
The CHAIRMAN. Thank you, Mrs. Clark.
I just would like the record to show that your testimony today has been given on behalf of not only yourself and your family and friends, but the American Lung Association. We really appreciate your taking time and testifying before us today.
I know you have to go to the White House. Therefore, I will reserve any questions I have, and I will turn to Senator Hawkins.
Senator HAWKINS. Mrs. Clark, many individuals say that they can stop smoking any time they want to do so, but the Public Health Service has called smoking not just a habit but the most widespread example of drug dependence in our country.
As someone who assisted her husband in quitting smoking and lived through those dark days, do you think that smoking is properly termed a form of drug dependence?
Mrs. CLARK. I definitely do. I lived with my husband for a lot of years. I have seen him through a lot of trials. I have seen him through a lot of living. I am here to tell you, as he told me, the hardest thing he ever had to do in his life was to stop smoking.
He was a man of warmth. He was a pleasant person, but let me tell you, when he was trying to stop smoking, his personality changed considerably, and I was very happy once it was over.
Senator HAWKINS. Did he quit just one time or was he like a lot of smokers—
Mrs. CLARK. I think he had it in his mind to quit for quite some time before he did. He was a great one to never make a statement unless he knew he could follow through. I think he resolved many times himself before he let it be known to other people that he would quit. When he finally did, he told me he was going to throw them away and quit. He said "cold turkey" because he thought that was the only way he could do it, and that is what he did.
Senator HAWKINS. That was 12 years ago?

Mrs. CLARK. Pardon me?

Senator HAWKINS. That was 12 years ago?

Mrs. CLARK. Twelve years ago, yes.

Senator HAWKINS. Thank you.

Mrs. CLARK. You are welcome.

The CHAIRMAN. Thank you, Senator Hawkins.

Senator East?

Senator East. Thank you, Mr. Chairman.

I would simply like the record to show, that I think is a great national sentiment, the great admiration that the people of this country have for the late Dr. Clark and Mrs. Clark and the great example they have set in terms of personal and family courage and his willingness, obviously, to make a tremendous sacrifice in terms of his own personal pain and suffering to continue in the forefront of pioneer research in this area of dealing with heart disease.

I would certainly feel remiss if I did not take this opportunity to express that point for the public record. Appreciating, as you have said, Mr. Chairman, that Mrs. Clark has a demanding schedule here while she is in Washington, I would not wish to unduly impose upon her time. Therefore, that will suffice in my case.

Thank you.

The CHAIRMAN. Thank you, Senator East.

Senator Grassley?

Senator Grassley. I just want to take this opportunity as well to thank you and your family for their sacrifices and contributions to medical research in the United States. It is a tribute to your faithfulness. We appreciate it very much.

Mrs. CLARK. Thank you very much.

The CHAIRMAN. Mrs. Clark, let me just, since we have do have a second or two, let me ask you one question that I have on my mind.

You mentioned that Dr. Clark often encouraged smokers to quit. In your opinion, what was his most persuasive argument that he gave to others in encouraging them to quit?

Mrs. CLARK. Well, I think since his illness, he made an example of himself. I think that was a very persuasive way to do it.

I know when he stopped smoking that the reason he stopped was because he was convinced that it was injurious to his health. I think that was the best way and the one that he used most often to try to dissuade others to stop smoking, because he felt that it was detrimental to the organs of the body and he thought that it would cause great grief in the future.

The CHAIRMAN. Thank you, Mrs. Clark, we have been so pleased to have you with us today. I think your family and yourself and your husband have all been symbols of courage and strength for the people throughout our Nation, and I might add throughout the world. I am grateful that you would take time to join us, at this hearing and to share your experiences with us concerning your husband, Dr. Clark, and his illness.

As you know, you are a celebrity not only throughout the country, but in Washington, D.C., as well as in your home State of Washington. I think it is due in part here because of the excellent article written about you by the Washington Post yesterday.
Mrs. CLARK. Yes, I appreciate that very much.

The CHAIRMAN. Well, we do, too. Your testimony has provided us with some reasons, I think really all the reasons that we need, in order to pass this particular smoking prevention and education act.

We appreciate your being here today. We are very grateful for your taking time out.

Mrs. CLARK. Thank you.

May I just make one statement? I want to thank all of you. The pleasure has been mine. It has been a great experience. I also want to thank the American Lung Association who invited me here today. If it had not been for their invitation, I would not have been here. I have enjoyed it very much. I want to thank all of you.

The CHAIRMAN. Thank you. Have an enjoyable time while you are here.

Mrs. CLARK. I shall. Thank you.

The CHAIRMAN. At this time we welcome Dr. Edward Brandt, the Assistant Secretary for Health, Department of Health and Human Services. He is accompanied by a distinguished group of physicians representing the highest level of public health service. They are Dr. Claude Lenfant, Director of the National Heart, Lung, and Blood Institute, National Institutes of Health; Dr. Mortimer B. Lipsett, Director of the National Institute of Child Health and Human Development; Dr. William Pollin, Director of the National Institute on Drug Abuse; Dr. Jane Henney, the Deputy Director of the National Cancer Institute; and Dr. JoAnn Luoto, the Acting Director of the Office of Smoking and Health.

We are delighted to have all of you here. As in the case of Mrs. Clark and all other witnesses today, we will put your complete statements into the record.

Senator GRASSLEY. I have a statement that I want to submit.

The CHAIRMAN. Without objection, we will put Senator Grassley's statement in early in the record, following Senator East.

[The prepared statement of Senator Grassley follows:]

PREPARED STATEMENT OF SENATOR CHARLES E. GRASSLEY

Senator GRASSLEY. Mr. Chairman, certainly over the years, the American public has been told of the deleterious effects of cigarette smoking. Now we learn through the 1982 Surgeon General's "Report on the Health Consequences of Smoking" that cigarette smoking is cited as the major single cause of cancer mortality in the United States, contributing to 30 percent of all cancer deaths. We have a bill before us that is aimed at educating the public as to the health consequences of smoking.

About a month ago, this committee extended the moratorium on an artificial sweetener, saccharin, which the FDA had proposed to ban in 1977 from processed foods and drinks after researchers linked it to cancer in animals. We extended the moratorium on the ban of the sweetener because no conclusive evidence exists that saccharin is unsafe for human consumption.

Nevertheless, the statement which accompanied the introduction of this bill stated and I quote, "The evidence is overwhelming associating cigarette smoking with multiple diseases and premature death." If this is the conclusive result, then why are we merely
warning a public that has already been threatened and wheedled and pleaded with over the years? If we now have conclusive evidence why do we not just ban cigarettes altogether?

We see Brooke Shields with cigarettes in her ears on television, and in ads telling teens that cigarette smoking is unattractive. We have taken cigarette ads off of television and relegated it to ads in magazines and newspapers. Doctors warn patients and teachers warn their students.

My understanding is that all studies, including polls by Roper, Chilton, Gallup, and others, demonstrate that Americans are aware of the claimed risks of smoking. I find it difficult to authorize another interagency committee and still another Office of Smoking and Health to warn Americans about something that they have been hearing everyday: Cigarette smoking is injurious to your health.

The CHAIRMAN. Dr. Brandt, we are grateful to have you here again. We appreciate the great work you are doing for the people in this country at Health and Human Services. We will now turn the time over to you.

STATEMENT OF EDWARD N. BRANDT, JR., M.D., ASSISTANT SECRETARY FOR HEALTH, DEPARTMENT OF HEALTH AND HUMAN SERVICES, ACCOMPANIED BY DR. CLAUDE LENFANT, DIRECTOR OF THE NATIONAL HEART, LUNG AND BLOOD INSTITUTE, NATIONAL INSTITUTES OF HEALTH; DR. MORTIMER B. LIPSETT, DIRECTOR OF THE NATIONAL INSTITUTE OF CHILD HEALTH AND HUMAN DEVELOPMENT; DR. WILLIAM POLLIN, DIRECTOR OF THE NATIONAL INSTITUTE ON DRUG ABUSE; DR. JANE HENNEY, DEPUTY DIRECTOR OF THE NATIONAL CANCER INSTITUTE; AND DR. JOANNE LUOTO, DIRECTOR OF THE OFFICE OF SMOKING AND HEALTH

Dr. BRANDT. Thank you very much, Senator Hatch.

Mr. Chairman and members of the committee, I am pleased at this opportunity to appear before this committee and to review with you the views of the Department of Health and Human Services on the health effects of cigarette smoking and to comment briefly on some of the provisions of S. 772, which is now before your committee.

With me today, as you pointed out, are Dr. Jane Henney, Deputy Director of the National Cancer Institute, to my far right, Dr. Claude Lenfant, to my immediate right, Director of the National Heart, Lung and Blood Institute, to my far left, Dr. Mortimer Lipsett, Director of the National Institute for Child Health and Human Development, next to him, Dr. William Pollin, Director of the National Institute on Drug Abuse, and, next to me, Dr. Joanne Luoto, and, to correct the record, she is now the Director of the Office on Smoking and Health.

With your permission, Mr. Chairman, we would like to submit written testimony from each of them for the record.

The Chairman. Without objection, we will put all of those written testimonies into the record.

Dr. BRANDT. Thank you very much.
Let me begin by summarizing our current understanding of the relationship between cigarette smoking and cancer.

We have known for at least two decades that cigarette smoking is the chief cause of lung cancer in the United States, a disease which in 1983 will take the lives of an estimated 117,000 men and women. We now know that smoking is related not only to lung cancer, but to other cancers as well. It causes cancer of the larynx, oral cavity, and esophagus, and contributes to the development of cancers of the bladder, pancreas, and kidney. Overall, approximately 30 percent of all cancer deaths are attributable to tobacco, particularly cigarettes.

The 1982 report on the Health Consequences of Smoking was devoted to the relationship between cigarette smoking and cancer. The 1983 report, which is now in the process of final review, will be devoted to the relationship between cigarette smoking and cardiovascular diseases. We do not need to wait upon the publication of the report, however, to paint the broad outlines of the heart disease-smoking relationship. It has been estimated that there are over 200,000 smoking-related coronary heart disease deaths in the United States each year. Cigarette smoking is one of the three major risk factors for this disease. Cigarette smoking by women who are taking oral contraceptives increases their risk of heart attack by as much as twentyfold. The effect is dose-related and the interaction of cigarettes and contraceptives is much greater than the sum of the separate risks.

Previous reports of the Public Health Service have disclosed that cigarette smoking is the leading cause of chronic obstructive lung disease, which includes emphysema and chronic bronchitis.

In addition, the risk of neonatal as well as fetal death increases directly with increasing amounts of maternal smoking during pregnancy.

It is never too late to stop smoking. Shortness of breath, sinus troubles, and persistent cough may start to disappear soon after quitting.

The recent multiple risk factor intervention trial demonstrated a dramatic drop, nearly 76 percent, in the coronary heart disease death rates in men who quit smoking early in the study.

In March the Public Health Service issued a pamphlet, which we would like to submit for the record, with your permission, Mr. Chairman.

The Chairman. Without objection, we will put it in the record at this point.

[The pamphlet referred to follows: ]
WHY PEOPLE SMOKE CIGARETTES

This statement on cigarette smoking has been developed from testimony delivered before the U.S. Congress by William Pollin, M.D., Director of the National Institute on Drug Abuse. (Comprehensive Smoking Prevention Education Act. Hearings before the Committee on Labor and Human Resources, U.S. Senate, March 16, 1982, p. 52)
Four drugs stand out among treatment cases of abuse. Fewer than 500,000 persons use heroin, but with other opiates it exacts a terrifying toll of crime and social disarray. Alcohol affects 10 million problem drinkers and their families and accounts for half our automobile fatalities. Marijuana is the most widely used illicit drug. Tobacco, in the form of cigarettes, is smoked by 56 million Americans. It causes more illness and death than all the other drugs.

For years, smoking was thought of as a habit—a "nasty" habit, Mark Twain called it. It has now been established that for many smokers it is more than this; that it is, in fact, the most widespread example of drug dependence in our country. Smokers can break themselves of smoking, and every year hundreds of thousands do, but this can be very difficult.

All drugs that produce dependence have at least these four characteristics in common:

All of them, cigarettes included, are psychoactive. They affect the chemistry of the brain and nervous system.

By definition, they create dependence and lead to compulsive use. This is at least as true of cigarettes as of other drugs and other substances of abuse.

When one gives up the drug abruptly, one may experience physiological and psychological distress. The severity will vary from one drug to another and from one person to another.

Finally, there is a strong tendency among former users to relapse, sometimes months or even years after quitting. Cigarette smokers experience this to perhaps a greater degree than do users of other drugs and other substances of abuse.

The addictive nature of cigarette smoking is why cigarette sales continue year after year in spite of the health hazards of smoking. Most people who smoke acknowledge the risks they run of illness and premature death, and most would like to quit, but the difficulty of quitting discourages them. Millions do try, every year, but of these only about 20 percent succeed. Over time, the percentage of smokers in our population keeps going down, but the process is slow.

The Role of Nicotine

In recent years, scientists have studied the role of nicotine in helping establish and maintain tobacco dependence. Their research suggests that it is nicotine that reinforces and strengthens the desire to smoke and causes users to keep on smoking.

Nicotine occurs naturally in the tobacco plant, and in large quantities is extremely poisonous. Cigarette smokers absorb it more quickly than...
pipe and cigar smokers or those who chew tobacco or use snuff because most cigarette smokers inhale, and this is the fastest and most efficient way of getting nicotine to the brain.

Nicotine acts through specialized cell formations located in the human brain and muscle tissues. These receptors have the capacity to recognize and react to nicotine when it is present in the body. Nicotine and the opium derivatives are the only other drugs of dependence for which specialized receptors of this kind have been identified and studied in detail.

When the receptors signal the presence of nicotine, a wide range of physical reactions occur. Changes occur in heart rate and skin temperature, blood pressure rises, peripheral blood circulation slows, changes occur in brain waves, and hormones affecting the central nervous system are released.

The immediate effects of inhaled nicotine are subtle and not catastrophic; the truly serious health consequences of smoking occur over long periods of time and are due to many tobacco smoke constituents in addition to nicotine. There is evidence to suggest that nicotine is implicated in the onset of heart attacks and that it is a co-carcinogen, enhancing the tumor-producing effects of cigarette tars. Its most important role, however, is its action on the brain and nervous system in helping create dependence.

The Way People Smoke

If there were such a thing as a typical pattern of dependent smoking, it would begin in the morning with the first cigarette. This sends a burst or "bolus" of nicotine to the brain which produces an almost immediate feeling of euphoria and satisfaction. For the rest of the day the smoker tries to maintain this feeling by manipulating his or her intake of tobacco smoke, by inhaling more or less deeply, taking more or fewer puffs, and smoking at different intervals. When more than a certain number of cigarettes are smoked, acute toxic effects resembling nicotine overdose are experienced, such as nausea, light-headedness, and a marked rise in heart rate. When fewer than a minimum number of cigarettes are smoked, which appears for many people to be about ten cigarettes a day, blood levels satisfactory to the dependent smoker cannot be maintained.

What at first appears to be a casual, unordered routine, in short, turns out to be not casual; all, but a controlled behavior.

Nicotine can affect the body in different ways. In stressful situations, it can act as an anti-anxiety agent like a tranquilizing drug, while in serene situations it can act as a stimulant like the amphetamines. Some effects of
smoking are psychological. Some people enjoy handling cigarettes and fussing with matches and ash trays; others smoke out of habit, sometimes unaware even that they are smoking.

With no other drug do people so busy themselves with administering it as they do in the case of cigarettes. In an 18-hour waking day, a two-pack-a-day smoker spends from three to four hours with a cigarette in mouth, hand, or ash tray, takes about 400 puffs for the day, and inhales up to 1,000 milligrams of tar.

The tar and nicotine numbers which are printed in cigarette advertisements may not correspond to what smokers actually obtain. This is because the numbers are derived from cigarettes smoked by machines and machines do not smoke the way people do. A cigarette advertised as being in the 1 to 5 mg tar range can turn into a 15 to 20 mg tar cigarette if a smoker takes more and deeper puffs than the machine allows for, or even partially blocks the ventilating holes or channels which are found in many cigarette filters. People who switch to low-tar and low-nicotine cigarettes may increase their smoking, although not necessarily enough to equal their previous exposure to cigarette tar and nicotine.

Quitting Smoking

What happens when a person tries to quit smoking?

With some smokers, nothing at all happens; they simply quit. Many of these, but by no means all of them, will be light or casual smokers. Their question is whether the enjoyment they get from an occasional cigarette is worth the extra risks they run of illness and earlier death. These risks are not inconsiderable. Men who smoke less than half a pack a day are half again as likely to die from a heart attack as their counterparts who have never smoked, and their risk of dying of lung cancer is nearly five times greater. For heavy smokers the odds are a great deal higher. Those who smoke two packs a day have three times the risk of death from heart attack and nearly 20 times the risk of death from lung cancer.

Young people who have not yet become dependent on cigarettes may find it easy to quit. A surprising number of young people who smoke while in high school give it up when they leave school and go on to a job or college.

For those who have become dependent on cigarettes, the first few days of withdrawal are likely to bring about a number of physiological and psychological reactions. Among them may be decreases in heart rate and blood pressure, decreases in the excretion of some of the hormones which affect the nervous system, occasional headaches, and gastrointestinal
discomfort. For some people, a weight gain may take place, it is uncertain whether this is caused by increased appetite or a changed metabolism or both. Behavioral and mood changes may also occur, usually peaking within a few days after quitting. Common symptoms are irritability, aggressiveness, and difficulty in sleeping.

For the dependent smoker, these first few days of quitting are only the beginning; the real challenge comes later. The temptation to go back to smoking can be very strong and can continue for a long time. Any number of events and situations can trigger relapse, including, paradoxically, both moments of stress and unhappiness and moments of relaxation and pleasure.

Therapists use a number of drugs in treating heroin and other addictions, and scientists are studying similar drug approaches for cigarette smoking. So far, however, none have been conclusively proven to be useful. Some drugs which have been tried, notably the amphetamines, may make quitting more difficult. There are lessons learned in the treatment of other dependencies, however, which can be applied to cigarette smoking. Here are some of them:

1. It helps to recognize from the outset that a drug dependence may exist. As noted earlier, drug dependence is a state in which a person's freedom of choice has been compromised by the physical effects of the drug on the brain and the nervous system. To discover that quitting can be difficult is not a sign of a weak and faltering will.

2. Quitting is a long-term process; some former smokers report an occasional craving for cigarettes years after they have quit smoking. With tobacco, as with all dependence-producing drugs, relapse is common. If one fails on the first try, the only thing to do is to go back and try again.

3. In some cases it may be useful to attend a clinic or join a cessation group. Such services can be found in every large community, some privately sponsored and others operated by church or health agencies, some are listed in the telephone directory yellow pages under “Smokers Information and Treatment Centers.” Help can also be obtained from local offices of the American Cancer Society, the American Heart Association, and the American Lung Association.

4. Whether one joins a group or not, one should seek the support and help of other people. The most important single influence in determining whether one uses or does not use a drug is the behavior and attitudes of one's family and friends. This is just as true of cigarette smoking as it is of alcohol and heroin and the other drugs of abuse.

5. And friends and family should provide support and help. They, too, need to recognize that smoking can be more than just a “bad habit,” that it can be a drug dependence, an addictive behavior.
This paper has been reviewed by the National Advisory Council on Drug Abuse. The characterization of cigarette smoking as a drug dependence is supported by both laboratory and population studies and appears in the World Health Organization's ninth revision of the International Classification of Diseases and by the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, third edition.

Readers interested in reviewing the pertinent research are referred to:


Dr BRANDY This pamphlet describes the similarities which exist between dependence on cigarette smoking and dependence on other drugs and substances of abuse. It points out that all drugs that produce dependence, including cigarettes, have four characteristics.

They are, first, psychoactive, that is, they affect the chemistry of the brain and nervous system.

They create dependence and lead to compulsive use. When one gives up the drug abruptly, one may experience physiological and psychological distress. The severity will vary from one drug to another and from one person to another.

Finally, there is a strong tendency among former users to relapse, sometimes months or even years after quitting.

It is because of this addictive nature of cigarette smoking that it is so important to do everything in our power to dissuade teenagers from taking up smoking. We must continue our efforts to inform them of the hazards of smoking.

From the above scientific information, it is apparent that stronger warning labels can be justified. The Department has long supported a stronger warning.

Let me comment briefly on several provisions of the bill which is before this committee.

The bill would establish an interagency committee on smoking and health made up of representatives from various Public Health Service agencies, with the duty of coordinating and monitoring smoking-related research and education in the Government and acting as liaison with the private sector. We believe this function is currently being accomplished, and this provision, therefore, appears to us to be unnecessary.

Our Department does, however, believe, along with this committee and along with the intent of that legislation, that we need to expand our cooperative effort with other Federal, State, and local governments and with the private sector, including not only the voluntary health agencies who have contributed so much in the past and continue to do so, but also with public and private groups which, until now, have not been so deeply involved.

Considerable progress has been made over the years in reducing the incidence of cigarette smoking in the adult and youth populations, but there still remains much for all of us to do. There are still approximately 55 million adult and teenage cigarette smokers and last year they bought an estimated 634 billion cigarettes, less than in 1981 but still a staggering figure.

The bill would call for annual reports from the Secretary of Health and Human Services which would provide current information on the effects of smoking on health as well as providing information about programs of the Federal Government and the private sector. Should such a provision be enacted, our Department would continue to produce the annual scientific reports of the Surgeon General on the health consequences of smoking, as dictated by other legislation.

The bill would provide legislative authority and specific congressional direction for the efforts now being made by the Department to identify and assess the possible health risks of the additives used by the cigarette companies in manufacturing their products.
The Department has negotiated an agreement with the six major cigarette companies which permits us to learn the identities of substances commonly added to cigarettes.

The Chairman: I might add that probably would not have happened had we not held these hearings over the past couple years. At least, that is my feeling.

Dr. Brandt: All right, sir. [Laughter.]

The phrase "commonly added," which served as the basis for arriving at this agreement, is taken from Public Law 95-625, the Health Services and Centers Amendments of 1978. That is the legislation which first called upon our Department to look into the matter of additives.

The essential differences between the procedures which are set forth in the bill and those we have used in developing our agreement are as follows:

Our agreement is voluntary on behalf of the companies. The bill would make mandatory compliance.

Our agreement utilizes the language of the 1978 act, which calls for disclosure of "commonly added" substances, which are defined as those used by three or more companies. The companies, however, have agreed with us to supply us with the names of ingredients used in large quantities by less than three companies and to disclose, where possible, the amounts of each ingredient. The bill would require disclosure of all additives used.

The agreement requires that the list be kept confidential, whereas the bill would require publication of the list on an annual basis, although without identifying the brands in which the additives are used.

Let me comment briefly on the provision of the bill which would require that health warnings and disclosures of tar, nicotine, and carbon monoxide be displayed on cigarette packages. It has long been the position of this Department that warnings and disclosures should be required on packages, but we defer to Congress on the need for a legislative requirement that these be displayed in advertisements.

That concludes my testimony, Mr. Chairman and members of the committee. I and my colleagues are pleased now to answer questions that you may have.

[The prepared statement of Dr. Brandt follows:]

\[\text{[Signature]}\]
STATEMENT BY EDWARD N. BRANFIE, JR., M.D., ASSISTANT SECRETARY FOR HEALTH, DEPARTMENT OF HEALTH AND HUMAN SERVICES

I am pleased at this opportunity to appear before this Committee and to review with you the views of the Department of Health and Human Services on the health effects of cigarette smoking and to comment briefly on some of the provisions of S. 772, the "Smoking Prevention Health and Education Act of 1983" which is now before your Committee.

With me today are Dr. Jane Henney, Deputy Director, National Cancer Institute; Dr. Claude Lenfant, Director, National Heart, Lung and Blood Institute; Dr. Mortimer Lipsett, Director, National Institute for Child Health and Human Development; Dr. Joanne Lusto, Director, Office on Smoking and Health; and Dr. William Pollin, Director, National Institute on Drug Abuse. Also, with your permission, I am submitting their written testimony for the record.

Let me begin by summarizing our current understanding of the relationship between cigarette smoking and cancer. This matter, as you know, was covered in detail in our 1982 Surgeon General's Report on the Health Consequences of Smoking.

We have known for at least two decades that cigarette smoking is the chief cause of lung cancer in the United States, a disease which in 1983 will take the lives of an estimated 117,000 men and women. We estimate that 85 percent of these, nearly 100,000 deaths, will be due to cigarette smoking.

We now know that smoking is related not only to lung cancer but to other cancers as well. It causes cancers of the larynx, oral cavity and esophagus and contributes to the development of cancers of the bladder, pancreas, and kidney.
Overall, as we said in our 1982 Report, approximately 30 percent of all cancer deaths are attributable to tobacco, particularly cigarettes.

The 1982 Report on the Health Consequences of Smoking was devoted to the relationship between cigarette smoking and cancer; the 1983 Report, which is now in the process of final development and review, will be devoted to the relationship between cigarette smoking and cardiovascular diseases. We do not need to wait upon the publication of this report, however, to paint the broad outlines of the heart disease-cigarette smoking relationship. It has been estimated that there are over 200,000 smoking-related coronary heart disease deaths in the United States each year. Cigarette smoking is one of three major risk factors for this disease, and is also a major cause of arteriosclerotic peripheral vascular disease. Additionally, cigarette smoking by women who are taking oral contraceptives increases their risk of heart attack by as much as 20-fold; the effect is dose-related and the interaction of cigarettes and contraceptives is much greater than the sum of the separate risks. This interaction also increases the risk of at least one type of stroke.

Previous reports of the Public Health Service have disclosed that cigarette smoking is the leading cause of chronic obstructive lung disease, which includes emphysema and chronic bronchitis. It has long been known that cigarette smoking is associated with a greatly increased risk of dying from emphysema.

The 1980 Report on the Health Consequences of Smoking dealt in part with the growing evidence that maternal smoking during pregnancy can result in miscarriage, premature births, and birth weight deficiencies, and can adversely affect long-term growth, intellectual development and behavioral characteristics. The risk
of neonatal as well as fetal death increases directly with increasing amounts of maternal smoking during pregnancy and results in a highly significant increase in the risk of abruptio placentae, placenta previa, bleeding early or late in pregnancy, and premature and prolonged rupture of membranes.

There is one item of good news in this array of bad news. It is never too late to stop smoking. Shortness of breath, sinus troubles, and persistent cough may start to disappear soon after quitting. Taste and smell may improve. Death rates for the many types of cancer caused by smoking and for cardiovascular diseases drop after quitting. The recent Multiple Risk Factor Intervention Trial demonstrated a dramatic drop (approaching 76 percent) in the coronary heart disease death rate in some groups of men who quit smoking early in the study. In the case of chronic bronchitis and emphysema, the progress of these diseases is slowed or halted when one stops smoking. The benefits of quitting apply even to the fetus: a woman who quits smoking early in pregnancy reduces the risk of having a low birth-weight baby.

In March the Public Health Service issued a pamphlet, which we would like to submit for the record, that describes the similarities which exist between dependence on cigarette smoking and dependence on other drugs and substances commonly. It points out that all drugs that produce dependence, including cigarettes, have these four characteristics in common:

* All drugs that produce dependence are psychoactive. They affect the chemistry of the brain and nervous system.
By definition they create dependence and lead to compulsive use. This is at least as true of cigarettes as of other drugs and substances of abuse. When one gives up the drug abruptly, one may experience physiological and psychological distress. The severity will vary from one drug to another and from one person to another.

Finally, there is a strong tendency among former users to relapse, sometimes months or even years after quitting. Cigarette smokers experience this to perhaps a greater degree than do users of other drugs and substances of abuse.

It is because of the addictive nature of cigarette smoking that it is so important to do everything in our power to dissuade teenagers from taking up smoking. Certainly we must continue our efforts to inform them of the hazards of smoking and the difficulties they may encounter later in life if they begin to smoke.

From the above scientific information, it is apparent that stronger warning labels can be justified. The Department has long supported stronger warning.

Let me comment briefly on several provisions of the Bill which is before this Committee.

The Bill would establish an interagency committee on smoking and health made up of representatives from various Public Health Service agencies, with the duty of coordinating and monitoring smoking-related research and education in the government and acting in liaison with the private sector. We believe this function is currently being accomplished, and, if necessary, could be expanded.
further through administrative action. This provision therefore appears to us to be unnecessary.

Our Department believes with the Committee, however, that we need to expand our cooperative efforts with other Federal, State and local governments and with the private sector, including not only the voluntary health agencies who have contributed so much in the past, but also with public and private groups which until now have not been so deeply involved.

Considerable progress has been made over the years in reducing the incidence of cigarette smoking in the adult and youth populations, but there still remains much for all of us to do. There are still approximately 53 million adult and teenage cigarette smokers and last year they bought an estimated 634 billion cigarettes — less than in 1981, but still a staggering figure. We are experiencing an epidemic of lung cancer deaths among women, such that lung cancer will soon become the leading cause of cancer death among women. Teenage smoking rates, which have been falling steadily for some six years, now appear to be holding steady.

The Bill would call for annual reports from the Secretary of Health and Human Services which would provide current information on the effects of smoking on health as well as providing information about programs of the Federal government and the private sector. If such a provision were to be enacted, our Department would continue to produce the annual scientific reports of the Surgeon General on the health consequences of smoking, in addition to reports on the activities of the Federal government and the private sector.
The Bill would provide legislative authority and specific Congressional direction for the efforts now being made by the Department to identify and assess the possible health risks of the additives used by the cigarette companies in manufacturing their products.

The Department has negotiated an agreement with the six major cigarette companies which permits us to learn the identities of substances commonly added to cigarettes. The phrase "commonly added", which served us as the basis for arriving at the agreement, is taken from Public Law 93-626, the Health Services and Centers Amendments of 1978. This is the legislation which first called upon our Department to look into the matter of additives.

The essential differences between the procedures which are set forth in the Bill and those which we have used in developing our agreement are as follows:

1) Our agreement is voluntary on behalf of the companies; the Bill would make mandatory compliance by the cigarette companies.

2) Our agreement utilizes the language of the 1978 Act, which calls for disclosure of "commonly added" substances, which are defined as those used by three or more companies. The companies, however, have agreed informally to supply us with the names of ingredients used in large quantities by less than three companies and to disclose, where possible, the amounts of each ingredient on its list. The Bill would require disclosure of all additives used in any cigarette.
3) The agreement requires that the list be kept confidential, whereas the Bill would require publication of the list on an annual basis, although without identifying the brands in which the additives are used. Accompanying this list would be a report from the Secretary describing research activities and findings and identifying any additive which in the Secretary's judgment poses an additional health risk to smokers.

The companies' list of additives was reviewed by a representative of the Public Health Service some weeks ago and we are now in the process of determining the most responsible way of evaluating the individual ingredients. We have convened a group of toxicologic and chemical experts to consider procedures for investigating the health effects of the additives. The list itself, in accordance with the agreement to protect the trade secrets involved, will not be made public, and access to it by Department employees has been limited.

Let me comment finally on the provisions of the Bill which would require that health warnings and disclosures of tar, nicotine, and carbon monoxide be displayed on cigarette packages. It has long been the position of this Department that warnings and disclosures should be required on packages, but we defer to Congress on the need for a legislative requirement that these be displayed in advertisements.

I would now conclude my testimony. We will be very pleased, Mr. Chairman, to answer any questions you may have.
The CHAIRMAN. Thank you, Dr. Brandt.
I want to personally thank you for your time and effort in presenting your testimony today.

From your testimony, it is obvious that the administration shares with many of us in Congress a great concern about the health hazards of cigarette smoking. I think you personally even support stronger warning labels, but does this mean that HHS is endorsing this legislation?

Dr. BRANDT. Mr. Chairman, as I pointed out, there are certain portions of this legislation that we feel are not necessary. Our view is that the Congress has assumed the responsibility of regulating the tobacco industry and regulating cigarettes. We are, therefore, here in the function of trying to give you our views about the health hazards of smoking and to comment on those portions of the bill with which we disagree.

We do support the concept of stronger warnings.

The CHAIRMAN. You have given an awful lot of backing to the assertion that cigarette smoking is very harmful to human beings.

Dr. BRANDT. I think there is a great deal of scientific evidence, and one only needs to review even the last four or five Surgeon General reports to determine that persons who choose to smoke are, in fact, exposing themselves to great risk.

The CHAIRMAN. Dr. Lenfant, the findings presented by you relevant to smoking and its associated effects, it seems to me, are overwhelming. It has been said that smoking only hurts the smokers. However, in your presentation you have provided evidence indicating the contrary. Could you expand on that evidence for our committee?

Dr. LENFANT. Yes, Mr. Chairman. You are referring to what is called passive smoking; that is, inhaled cigarette smoke which is produced by somebody else's cigarettes.

The CHAIRMAN. In other words, sitting in a smoke-filled room or an airplane or a bus or even in a car, anywhere that smoking is going on.

Dr. LENFANT. That is right. It does not matter whether it is in a room or an airplane.

I should correct your statement with regard to the airplane situation. I just heard this morning the report which is going to be published today in the New England Journal of Medicine, which I am told indicates that the nonsmoking section of an airplane is, indeed, protected against the effects of smoke that would come from the smoking section of the airplane. Therefore, I think we have to be very careful to recognize that my statement with regard to the airplane situation would have to do with the smoking section and not with the nonsmoking section.

The CHAIRMAN. I see.

Dr. LENFANT. I think that is an important point that needs to be made.

Having said that, Mr. Chairman, I think that, indeed, there is interesting evidence that the effect of passive smoking is detrimental. A number of studies have been published during the past few years in which the danger of passive smoking should be recognized.
For instance, the children of smoking parents have been recognized as having a greater prevalence of bronchitis and pulmonary infection. Such studies have come from the United States and Europe as well.

We know today that nonsmoking patients with coronary-artery disease would experience chest pain which is due to spasms of the coronary arteries when they enter a room which is filled with smoke. The spasm of the coronary artery is often an event that leads to heart attack.

There are some studies which have indicated that workers who are in an environment where some of their coworkers are smoking may have a greater decline in their pulmonary function than indeed, workers who would not be working in such an environment.

I should also point out that studies from Japan have been reported showing that wives of individuals who are heavy smokers have a higher prevalence of lung cancer than wives of individuals who are not smokers.

Still coming from Japan, there is a very interesting study that was published just a couple of months ago which has established that the mortality due to coronary heart disease was twice as high in women with husbands who were smokers than in the situation where wives do not have a smoker as a husband.

Therefore, clearly, I think the evidence is increasing that passive smoking is not something that we should ignore as a potential for health complications.

I should like to finish by emphasizing what Senator Jackson was saying earlier when he said that he quit smoking because of the allergic reaction to smoke, and that is, indeed, something that ought to be recognized.

The CHAIRMAN. Thank you, Dr. Lenfant.

Dr. Lipsett, in your opinion, all of the complications that you have mentioned that are known to be caused by cigarette smoking in the young population, which are the ones that could be the most easily prevented?

Dr. Lipsett. Mr. Chairman, one of the largest problems we have today in this country is the problem of low birth weight infants. If mothers would stop smoking by at least the fourth month of pregnancy, we could make large inroads into that problem.

In studies from all over the world it has been shown that a pregnant woman smoking one pack of cigarettes per day leads to about a half a pound decrease in the weight of the infant. If there are other factors that can lead to low birth weight infants, this can present a very serious problem.

The CHAIRMAN. Thank you.

Dr. Henney, let me just ask you a question. In your testimony you mentioned the National Cancer Institute's support of a number of projects aimed at providing information to young people who face the decision to smoke. Could you describe these programs and help us to understand them a little bit better?

Dr. Henney. We have several different programs that are specifically targeted to the youth, and they are supported by our grants and contracts programs.
One program that we do have which is generalizable and is available to schools throughout the country is a booklet that is specifically geared to help administrators, educators, concerned parents to target on different ways get the adolescent to either never start smoking, hopefully, or to stop smoking if they have started.

The CHAIRMAN. Thank you.

Dr. Pollin, it has been said that the trends regarding cigarette smoking are associated with the use of illicit drugs. You have mentioned that use of illicit drugs is proportional, or at least associated in some way, with cigarette smoking. Therefore, if we have stronger smoking labels or warning labels and succeed in educating some of our citizens not to smoke, can we expect a decrease in the use of illicit drugs?

Dr. Pollin. I believe we very definitely can, Senator Hatch. Our most recent 1982 survey shows that American teenagers who are current cigarette smokers are 10 times more likely to be current users of marihuana, 14 times more likely to be current users of heroin, cocaine, and other drugs. It is our clear belief that if we can prevent young people from initiating cigarette smoking, that that is one of the most effective ways to prevent their getting started on the use of illicit drugs as well.

The CHAIRMAN. Thank you.

Dr. Pollin, when did it become apparent to scientists that cigarette addiction is as serious a problem as any other drug addiction?

Dr. Pollin. The conclusion that cigarette smoking is an addictive disorder rather than a bad habit, like losing control of the number of potato chips you eat, became confirmed internationally back in 1979. In the most recent edition of the International Classification of Disease, which is used worldwide, tobacco dependence is listed along with dependence upon other drugs which are known to be drugs of abuse.

The relative importance of this addiction as compared to other drug-dependent problems is derived from comparisons of the mortality and the morbidity which is attributable to cigarette smoking.

The CHAIRMAN. Thank you.

Dr. Luoto, you are the Acting Director of the Office of Smoking and Health. I am pleased about the number of public places where nonsmoking areas have been voluntarily established. Have you received—or do you know of—much criticism about designation of nonsmoking areas?

Dr. Luoto. No. I think if you look at the survey results that are available, the majority of all citizens would prefer segregation of areas by smoking status. That information is available for multiple different sites. Of course, it is a more marked preference among nonsmokers, as you might imagine, because of the annoyance factor of others smoke. However, it also is true in some cases among smoking individuals as well.

The CHAIRMAN. In your opinion, what are the most effective smoking prevention efforts that your office makes?

Mr. Luoto. It is hard for me to separate out one. I think if I had to, I would probably choose the Surgeon General's report because of its pre-eminent scientific reputation basis, its careful scientific review, because of the resultant publicity it gets. I think it serves,
on a regular basis, to reinforce the health concerns that we, the Public Health Service, have to pass on to the public.

The CHAIRMAN. Thank you.

Dr. Brandt, I want to thank you and your associates for being here with us and for the excellent report you have.

I have to go to the White House. Therefore, I am going to turn the gavel over to Senator Hawkins, who will next ask her questions, and then presumably call on Senator East and Senator Grassley.

I really appreciate the efforts that you have put forth in this matter.

Dr. BRANDT. Thank you very much.

The CHAIRMAN. Senator Hawkins?

Senator HAWKINS [acting chairperson]. Dr. Brandt, one of the most recent publications by the Public Health Service is a bulletin called, Why People Smoke Cigarettes.

Dr. BRANDT. Yes, ma'am.

Senator HAWKINS. It warned that tar and nicotine figures printed in advertisements may not be the same as the actual tar and nicotine inhaled by the smoker. Could you explain why there is a difference?

Dr. BRANDT. It is largely technical, Senator Hawkins. It has to do basically with the devices that are available for measuring tar and nicotine from smoking cigarettes. In one sense, these are a sort of surrogate measure, if you will, because the machines clearly do not smoke in the same way that people smoke. Therefore, it is unlikely that they are complete measurements.

Senator HAWKINS. Therefore, an individual could inhale more or take more puffs than your machine is set at?

Dr. BRANDT. Yes, ma'am. Furthermore, the issue of depth of inhalation and how much one smokes as the cigarette gets closer to the filter, and thereby having a greater concentration of materials in the filter to be inhaled, all influence the actual dose of material that any particular person will get.

Senator HAWKINS. Thank you.

Dr. Lenfant, in the Japanese study it said that the life span of a wife of a smoker is shortened by approximately 4 years. Is that true? Would you like to comment on that?

Dr. LENFANT. Senator, I do not know to what specific studies you are making reference.

Senator HAWKINS. The Japanese study.

Dr. LENFANT. Yes. The one about the higher prevalence of coronary heart disease?

Senator HAWKINS. Yes.

Dr. LENFANT. That is correct. Some of these women die from coronary heart disease at a premature age. The number of these women dying of such disease is larger when they are married to a husband that smokes.

Senator HAWKINS. Dr. Brandt, I agree that consumers have an increased awareness of health risks. We have publicized a lot and demanded that information is given to the public. The smokers then, I believe, have started to demand filtered and low-tar cigarettes. Couldn't these new brands actually be more hazardous to their
health because of the various ingredients that are added to compensate for the loss of flavor or fragrance?

Dr. BRANDT. There are two aspects, Senator Hawkins.

The first is that the low-smoke tar cigarettes are safer only if people do not change their style of smoking. If, in fact, they smoke more or if they inhale deeper, then in reality they have not changed the dose of substances that they are getting. In other words, if you smoke a cigarette with half of the tar and nicotine content but smoke twice as many of them, you are clearly getting the same kind of dose.

The issue of the additives is one that, of course, is commonly raised and that we have begun to address. I think at this point in time it would probably be premature to try to determine whether or not those additives increase or decrease the risk of what one is inhaling. I just do not know right now.

Senator HAWKINS. In my opening statement I believe you heard me mentioning that deerstongue was generally considered a dangerous additive for cigarettes because it was already banned as a food additive. Aren't there many of the common cigarette additives, like cocoa, sugar, licorice, and caramel, acceptable as food additives but potentially hazardous when burned?

Dr. BRANDT. I would say, in the first place, I do not know what the common additives are, but I think that it is clear that many substances that might be eaten may very well be harmful if burned at high temperatures and inhaled. On the other hand, some substances which are harmful if eaten may very well be altered by high temperature and may not be harmful to breathe. Therefore, at this point in time, in dealing with the additive issue, at least on the basis of the scientific information that is available now, we cannot be very precise about the health hazards or lack thereof of any particular substances.

Furthermore, we have the issue of attempting to determine the interactive effect between two substances being burned at high temperature. It may again be that two additives are, in fact, harmless by themselves but harmful when combined together, or vice versa. Those are questions that still need to be answered.

Senator HAWKINS. Thank you.

Senator EAST. Thank you, Madam Chairman.

Dr. BRANDT. I would like to welcome you and your distinguished group here this morning.

There are several points I would like to pursue with you.

First of all, there is this question of scientific causation. I note in this proposed legislation, it says, "Congress finds that...". The implication is that we independently find. I presume, or it could be so interpreted as we independently find, certain scientific results. I certainly am not asking you this, but I would like the record to show, and I think commonsense observation would indicate, that Congress lacks the resources and the facilities to determine matters of scientific causation. We simply do not have scientific laboratories. We might conceivably put the stamp of approval on what others are finding.

I do not mean to niggle, but I am suggesting that when we are told "Congress finds" and then we are shown five findings, the im-
plication is, some way or another, we independently on our own re-
sources and in-house operation have found these things to exist.

Now, again, some may think that is quibbling, but it really is not
because when you get into this question of scientific causation—
and this is a point I was trying to make at the outset—I think we
want to make sure that we can keep science and policy goal and
desire and political end separate. I know those in the scientific
world would want to do that, too. In the name of science, we are
not stating policy or politics, or in the name of policy and politics
we are not trying to pretend we speak as scientists. I am not saying
there is not some point at which we do not come together, because
you can provide us with very valuable information.

For example, just beginning "Congress finds," I think we ought
to at least clarify we do not independently find. It is not to niggle;
it is to make sure that the act says what, in fact, is the case, be-
cause this is a statement, allegedly, of scientific value.

This first provision here, "Cigarette smoking is the largest pre-
ventable cause of illness and premature death," again not to
niggle, but one wonders candidly, as a matter of genuine scientific
causation, whether one could say that.

For example, we have already indicated in the testimony that if
it is true cigarette smoking is as addictive as people say that it is,
then it may be one of the most preventable causes of illness. How-
ever, it could be, for example, teenage driving and drinking is the
largest preventable cause of illness and premature death. I do not
know I am not saying it is. It may be easier to throw away the salt
shaker than the cigarette pack. Hence, one might say—again I am
talking about scientific causation—that, in fact, moving away from
salt in terms of high blood pressure might be the largest prevent-
able cause, or the excessive use of fatty meats, or the excessive use
of any of those things that could be identified as representing
threats to our general physiological health.

Keep in mind, I am not trying to obscure; I am simply trying to
stress for the record that those in the scientific community, and
those in the Congress who share common policy goal and end here
should not enter into unwittingly and unthinkingly, an unholy alli-
ance in which politics and science is inextricably intertwined and
does not really do justice to either side.

On this matter of the accuracy of the statement, mind you these
are supposedly scientific findings. You will notice in some refer-
ces we are told it is cigarette smoking. Then in the very next
paragraph it will simply say "smoking."

In the next paragraph, then, does that include cigars and pipes?
Again, I do not wish to niggle, but I am simply saying, if you mean
cigarette smoking, you ought to consistently say it throughout, and
I think a good scientist would do that.

Of course, we have various forms of smoking—cigarettes, cigars,
pipes. We would probably have to separate them into categories of
potential scientific findings: Lip cancer versus lung cancer, per-
haps. I do not know. That is not my field. I simply am saying that
is the field of science.

I suggest, if I might put it charitably, a certain sloppiness in
drafting, which I am not faulting the world of science for. I might
be subtly criticizing our own legislative drafting, but I am warning
you good people in the world of science that in this pell-mell rush to nail the cigarette industry in this country, the smoking industry, or whatever it is, that you do not be pulled into what is basically a political statement. I think you do a disservice to science politically, just as we would do disservice to politics and policy-making by claiming we independently find, which in fact we have not done.

Dr. Brandt, you had indicated a moment ago that you thought there were certain provisions in here that may not be necessary, as you view it from the standpoint of a public administrator, a very distinguished gentleman in your own right, and you hold a very high and responsible position in the Department of Health and Human Services.

I would like to ask you just to briefly once again identify what sections, if you would be willing to comment upon it, do you not find necessary.

Dr. Brandt. We do not believe that the creation of the inter-agency committee, as specified in this legislation, is necessary legislatively, because we have such a committee operational now. Second, any changes that need to be made in such a committee or in its functions could be done administratively. Therefore, it seems to us that that is not required in legislation.

The second issue is the issue of the annual report from the Secretary of Health and Human Services. As you know, we are now mandated to file an annual report, which is a scientific document known as the Surgeon General's report. That deals with a specific topic each year. Those topics are selected largely on the basis of updated scientific information.

This report that is mandated in the bill would in sense be somewhat duplicative and perhaps could be altered or mandated in such a way as to lead to only one report.

I think those are the two basic provisions that we believe could be handled administratively and do not require legislation.

Senator East. Thank you, Doctor.

I think my 10 minutes are up.

Senator Hawkins. Thank you.

Senator Grassley.

Senator Grassley. Going back to a point that Senator East referred to in regard to the first public finding of the bill, that cigarette smoking is the largest preventable cause of illness and premature death in the United States, among all of you on this panel, is that a correct and accurate statement from your understanding of medicine and research generally?

Dr. Brandt. Yes, sir.

Senator Grassley. And all of you agree with that?

Dr. Lenfant. Yes.

Dr. Lipsett. Yes.

Dr. Pollin. Yes.

Dr. Henney. Yes.

Dr. Lurito. Yes.

Senator Grassley. Now we are going to be receiving testimony all morning from different people who are referring to studies and projects that have been going on in the Scandinavian countries. Some refer to the fact that rotational labels have helped reduced
smoking, particularly among young people, in those countries. Other testimonies refer to the comprehensive smoking education that is being carried on in those countries.

Is it both or is it one or the other that is most responsible for the reduction of smoking in those countries?

Dr. Brandt, Senator, I think one has to begin by asking the question, why label a product, particularly with a warning label? Basically, the reason for that is education.

Although we support strong warnings, we do not believe that the warning label in and of itself, by itself, is going to be sufficient to provide the American people, or indeed the people of the world, with enough information to make intelligent decisions about smoking and the risks that they run.

I do not believe that it is really possible in those countries or in our country to separate out an educational program or campaign, say through television or other media, from the warning labels and say that public behavior is due to that or it is due to something else. It would be very, very difficult to do that with any degree of accuracy.

Senator Grassley. I will accept your statement. On the other hand, let me say that they are not referred to as necessarily having to go together. They are also referred to as separate approaches, even though perhaps that was not the intent of the writer of the various testimonies that we are going to be receiving.

Do any of you have any different view on the answer that was just given? I should invite that before I go on.

[No response.]

Senator Grassley. What about the amount of money that has gone into educating the public to this point and polls showing that the overwhelming percentage of people know that smoking is hazardous to their health? I would like to raise the proposition of whether of not we could better spend additional money on education or might it not be better to use that money for setting up clinics, self-help centers—in other words, going a step beyond the educational process, since the public is informed that smoking is hazardous.

Dr. Brandt, Senator, that is a complicated question.

In the first place, there are a lot of centers available. Indeed, I would say that anyone who wishes to quit smoking and wishes help in doing so will find that help available. The physicians of the country have begun to pay attention to this. There are all kinds of commercial clinics available as well as hospitals, religious groups, and others.

I personally do not see that that would be necessary.

It seems to me that the role that the Public Health Service views itself as having, is first, to provide the kind of information to people that they need to have and, second, to do the kind of research that is necessary to develop that information so that when we come out with statements or written material, we have scientific basis for it. Therefore, my own view is that the money is better spent on education at this point in time than it is on self-help clinics.

Also, the information that we have gained from surveys of people who have quit smoking are that most of them quit like Dr. Clark, quit; namely, on their own. A relatively small percentage of people
at least will admit to having quit through the use of some other assistance. That is, they tend to quit by finding some sort of way for themselves to quit. I, again, therefore, would think that the money is better spent in developing a strong educational program and the research that tells us where we need to target the educational programs and what kinds of messages will be most effective in changing behavior.

Senator Grassley. Have we reached the point in our educational process, where what we have done to this point has encompassed all it can. Should we move on to a different form, as the rotational labeling would indicate or as any stepped-up educational process would accomplish? Have we saturated the opportunities?

Dr. Brandt. I do not believe that we have saturated the opportunities. If one begins, for example, just to think about the number of new children that are coming into our population annually that need to be taught and need to know this—

Senator Grassley. What I meant was, under existing law, without going further than this bill provides, have we done all we can under existing law? Maybe that is the way you understood my question, but I wanted to clarify it before you got too far.

Dr. Brandt. I think that even under current law there are things we can do further. However, I should point out that a recent Gallup poll, in August 1981, reported that only 74 percent of the people surveyed believed smoking to be a cause of heart disease. Yet, I think, without question, it has been clearly demonstrated, and we will demonstrate it again through the upcoming Surgeon General’s report, that smoking is one of the three primary risk factors and, furthermore, the multiple risk factor intervention trial clearly demonstrated—

Senator Grassley. Then pursuing my question, that would be an argument that people would use to justify the additional legislation. Could that be accomplished—in other words, telling people about the education, contributing to heart disease—could that be done under existing law or do we need the change in law to accomplish that?

Dr. Brandt. The educational efforts, short of labeling, can certainly be done, and are being done, under existing law.

Senator Grassley. Therefore, we have not saturated all of our opportunities. We can still continue to work under existing law, even though further law might help the process? Is that a conclusion I can draw from your comments?

Dr. Brandt. I think that is a reasonable conclusion, yes, sir.

Senator Grassley. Madam Chairman, I have no further questions.

Senator Hawkins. Thank you.

Senator Quayle.

Senator Quayle. Thank you very much.

Dr. Brandt, I would like to focus on S. 772. I understand by your testimony, in response to a question, that you would basically deem as unnecessary section 4 of that bill which establishes the Interagency Committee on Smoking and Health. I wonder about section 5 of the bill on the additive information required, which is mandatory rather than voluntary compliance. Would you support or oppose that section in this bill?
Dr. Brandt. At the present time, Senator, as you know and as I testified a little bit earlier, we do have a voluntary agreement which allows us to begin to look at the additive issue. My own view about that is that that has not run its course yet, and I think we can use this opportunity to learn a great deal. Whether or not it is necessary to go on and make it mandatory, it seems to me, is an issue that we would defer to the Congress to decide.

Senator Quayle. Therefore, the Department does not have a position on section 5, on the mandatory requirement of the additive information?

Dr. Brandt. That is correct.

Senator Quayle. That is correct.

What about the multiple labeling in the bill? Do you support the approach that they have taken, that this bill takes on the multiple labeling?

Dr. Brandt. We do not take a position on the issue of rotating labelings or multiple labelings, Senator. Rather, our position is that we believe that there should be stronger warning labels than currently exist. However, as to whether or not they should be rotating or that sort of thing, we again will defer to the Congress.

Senator Quayle. Therefore, you really do not care one way or the other, whether we have one label or 10 labels or 3 labels? Do you care what they say?

Dr. Brandt. We certainly would want them to be scientifically accurate. We would certainly provide whatever assistance or——

Senator Quayle. Are these labels in this bill scientifically accurate?

Dr. Brandt. Yes, sir.

Senator Quayle. They are scientifically accurate?

Dr. Brandt. Yes, sir.

Senator Quayle. What about the multiple labeling? I have been told that in England they had multiple labeling and then they went back to one label. Is that correct?

Dr. Brandt. That is my understanding, yes, sir.

Senator Quayle. Why?

Dr. Brandt. Perhaps Dr. Luoto, would reply.

Senator Quayle. This is an issue we are going to have to decide on the multiple or single labeling. I really wish you would be able to give us a little more information. I know if you cannot go any further, that is understandable today, but as we go on I would hope that the Department might come down on this issue. It is not one that is a trivial issue. It is something that is fairly significant, as far as this bill is concerned and the whole issue.

Dr. Brandt. Yes, sir, I agree with you.

Dr. Luoto My office has spoken with the British Government. We were not able to determine the factors that went into that decision. We do know that the labeling on packages in England is a negotiated agreement between the Government and industry. It was renegotiated recently going back to a single warning that they had previously. I cannot give you the reasons.

Senator Quayle. In other words, England has a negotiated agreement between the Government, and the industry. It was a stronger label warning than what they had previously, correct?

Dr. Brandt. It is a stronger warning, but it is no longer rotating.
Senator QUAYLE. I understand from Dr. Luoto that it was a negotiated agreement that they got which went back to the single labeling, and that when they had single, rather than multiple, it is a stronger warning.

Dr. Luoto. That is my understanding.

Senator QUAYLE. That is your understanding? I wonder if that example might be applied here, since there is some uncertainty on whether we ought to have single or multiple warnings. If you could come up and put your people to work and come up with a stronger warning, which you say you have advocated for a long time, and take it to the industry to see if something could be worked out—has that been tried?

Dr. BRANDT. It has not been tried since I have been here, no, sir.

Senator QUAYLE. Do you think it would be worth trying?

Dr. BRANDT. I think that it could be tried, yes, sir.

Senator QUAYLE. Which would be preferable—to have this legislation or to try to work out an agreement like that—for a stronger label, which you all support?

Dr. BRANDT. The issue of which is preferable, I think that clearly it is preferable to have the Government and the industry in agreement and with the industry regulating itself and working with the Government in a cooperative way. It seems to me that is the preferable way to go.

Senator QUAYLE. I would tend to agree that it would be a preferable approach. I do not want to be presumptuous to give any advice. You are suppose to be giving us advice today. However, I wonder if this might be explored; to come up with a stronger label, a stronger warning that you would find scientifically conclusive and scientifically supported and to see what could be worked out. If that has not even been tried, I wonder if we might want to try that and then see where we could get.

Dr. BRANDT. I cannot tell you whether it has been tried in the past. I can only say that I have not tried it.

Senator QUAYLE. No, I am just talking about this administration. We have been into it 2½ years how.

Dr. BRANDT. We have not tried it. I do not think you need to apologize for being presumptuous. I have gotten a great deal of advice from the Congress in the 2 years that I have been here.

Senator QUAYLE. Then I will not be presumptuous. I will just ask you to do that.

Dr. BRANDT. All right, sir.

Senator QUAYLE. I really think that that would certainly be worthwhile and it might merit some attention. I certainly would support to work out, from my own personal opinion just briefly looking into the issue—we dealt with it on a very cursory basis last year, but we are getting more involved in. The legislation has a little bit more of a serious overtone this year than it did last year. I am quite shocked that this approach has not even been tried, quite candidly.

If you look at the precedent with England—I do not want to look to our mother country too often, but I think if they have worked it out and we might learn from the experience that they have had there, where they have a stronger warning, then it may be worth exploring.
Thank you, Madam Chairman.

Senator Hawkins. Dr. Luoto, do you know if in England they have any statistics to show if the multilabel is more effective than the single, stronger label that they negotiated? Do you know if there are any statistics?

Dr. Luoto. No. Basically, I am not aware of any studies that have been done on the effectiveness—

Senator Hawkins. Would you find out for us?

Dr. Luoto. Certainly.

Senator Hawkins. Would you find out for us and provide it to the Senators? It would be interesting if we have a country that has done it both ways.

[The information referred to follows]
Dear Senator Hatch,

I understand that your Committee is studying the question of health warnings on cigarette packs and/or cigarette advertisements, and that the British experience in this area may form part of the evidence which you consider. I thought you might find it helpful to have the views of my organisation about health warnings, not just to set in context any information on Britain which you may receive from other sources, but also from the standpoint of the experience we have gained from twelve years of work studying and fighting the smoking problem in what is, I regret to say, the country that leads the world in deaths per capita from lung cancer.

First of all, I should inform you that Action on Smoking and Health—ASH—was set up in 1971 by the Royal College of Physicians (of London) after its second major report on the damage to health caused by smoking. You may be aware that the College’s first report, “Smoking and Health,” published in 1962, was the first such report in the world from a respected medical authority, even pre-dating the now historic 1964 U.S. Surgeon General’s Report. The College felt—and I think I may safely say it still feels, just as strongly—that the establishment of a independent organisation to translate into action the medical knowledge about the harmful consequences of smoking, was a vital part of the fight to reduce smoking-induced disease.

ASH is a registered charity and receives most of its modest budget by way of an annual grant from the Department of Health and Social Security of the Government. We work closely with the Department and also with all major medical authorities active in the field of preventive medicine and we are constantly used by the press and electronic news media as the...
leading source of information about the many different aspects of the smoking problem.

HEALTH WARNINGS

Background

The history of health warnings in Britain is fairly straightforward. From the outset, the warnings have been placed on packs and advertisements by means of a series of "voluntary agreements" between the Government and the tobacco industry. In effect, this has meant that only warnings acceptable to the industry have been used; and in turn, this means the warnings have had little impact. The existence of these wholly inadequate warnings has even been used by the industry to try to fend off further measures designed to reduce smoking-induced disease. In effect, the argument has been: "There's a warning on the pack, so now anything we do to promote cigarettes is fair game."

ASH has never been party to the discussions between Government and industry which negotiate new voluntary agreements. We understand, however, that in successive negotiations, the industry has rejected suggestions for any new wording of health warnings which mention specific diseases: which enliven or personalise the message in the way which any commercially astute manufacturer would enliven or personalise a positive characteristic of his product and which might be displayed in a prominent position on the pack.

We further understand that tobacco company officials have told representatives of the Government that the industry could not consider any warning which might result in a reduction of cigarette sales. Last year, Dr. Kenneth Clarke, M.P., Minister of Health, stated that his Department had tried - but failed - to persuade the industry to accept the Department's own warning, which it prints on the official leaflets recording cigarette tar, nicotine and carbon monoxide yields: "Danger: cigarette smoking causes heart disease, lung cancer, bronchitis."

1980 Voluntary Agreement

A change of health warning to a series of three consecutive messages each to be applied for six months was made as part of the voluntary agreement announced in November 1980. The change fell far short of suggestions made to the Government by various health authorities: for example, the Scandinavian practice of having a series of sixteen different warnings, with informative and arresting texts, had been strongly advocated by ASH as a model to follow. Nevertheless, the change
was considered as a step in the right direction, if only by introducing the concept of a variable warning.

1982 Voluntary Agreement

That the triple warning scheme was abandoned by means of the next voluntary agreement (announced October 1982) is generally agreed in the medical world not to be for reasons of ineffectiveness. The new agreement was roundly condemned by medical authorities such as the British Medical Association and by established medical journals like The Lancet and The British Medical Journal. The prevailing opinion was that the tobacco industry had persuaded the Government to accept no improvements and some measures which were actually contrary to the interests of health.

The most scathing criticism was of a new fund, to be set up as part of the new agreement and to be jointly administered by nominees of industry and Government, for research into health promotion, financed by £3 million p.a. from the industry; the fund's rules provide that no projects concerned directly or indirectly with tobacco use can be considered for funding. In the context of this extraordinary scheme, which, forming part of a voluntary agreement on tobacco promotion, can be considered as little else than a cynical and reckless public relations exercise, the reversion to the original single health warning "Cigarettes can seriously damage your health" may be seen as a safe return to a warning known by the industry to have little or no effect on sales. While the size of the warning was increased by 50%, the new warning remains miniscule compared to the scale of risk of smoking; and the lettering remains difficult to read, easily designed out on advertisements and successfully hidden out of normal sight on the side of cigarette packs.

Disatisfaction with the present British Health Warning

There has been for some time, and remains, deep dissatisfaction in medical circles in Britain with the cigarette health warning. It is widely felt among health professionals in the UK that the present health warning is ineffective. To be effective, the warning should be glaringly obvious to smokers and not placed out of regular sight on the side of the pack or made visually insignificant on advertisements.

The remainder of this letter consists of a summary of comments on the existing warning, together with constructive suggestions for improvements. I submit that these comments and suggestions on the present British situation may be equally relevant to a consideration of the U.S. health warning. For reference, the British health warning is:

"DANGER: Government Health Warning: Cigarettes Can Seriously Damage Your Health."
The wording of the warning is unspecific and irrelevant. To most people, "can seriously damage your health" is bureaucratic language which is impersonal and unrelated to their own prospective health or sickness. The two additional warnings added to the packs by the 1980 voluntary agreement suffered similar deficiencies: "smoking may cost you more than money" had something of the riddle about it; and "the more you smoke the more you risk your health" may have been a fair statement, but was of little use without the relative risk of smoking even a small amount of cigarettes daily being properly understood by smokers in the first place — which it was not.

The ideal system would appear to be one which educates and informs, as well as warns. The Swedish system of sixteen warnings, all of which are changed every year or two, is something of a model. There is clearly nothing sacrosanct about the number sixteen, although presumably this fits into the way cigarette packs are manufactured because the sixteen are arranged to crop up randomly in the purchases made by smokers.

It is important to personalize the warnings as much as possible so that individual smokers realize that they are directed at themselves and not at all smokers in general, or, more dangerously, at every other smoker except themselves. Hence talking about "you" and "these cigarettes" is clearly better than using impersonal officialese.

Warnings should at least be displayed on the front of the pack and preferably on the back as well. A system employing warnings both on the back and on the front follows for a standard message on the front together with variable messages on the back or, vice versa.

If the warning is on the front of the pack alone then it should cover at least half the area. The risks of smoking, which are of a completely different order than those of any other consumer product, justify this.

Regulations should ensure that pack design does not allow warnings to be tampered with by use of colour, shading or surrounding design aimed at distraction or pack design aimed to make the front in effect the back.

On advertisements the warning should occupy a substantially increased portion of total area and should be highlighted within the advertisement rather than at the foot as at present, where its significance is reduced by design.
The idea of printing the warning on each individual cigarette should also be pursued. This would be in addition to warnings on the pack and on any remaining advertisements. The warning: “DANGER: Cigarettes cause lung cancer, bronchitis, heart disease” already in use on government tar cards, could suffice for this purpose.

**Concepts of Risk**

There are some smokers whose smoking habit will be unaffected however much detail they learn about health risks. On the other hand, it is generally accepted that many more smokers will be influenced if they have a better understanding of the relative health risks involved. The health warning may be an effective way for improving the smoker's education and information about the risks of his or her smoking.

Public awareness of health risks varies, in surveys carried out for U.K. Government's Health Departments, from 71% for lung cancer and bronchitis to 54% for heart disease. These levels are still lamentably small, especially for heart disease, the biggest smoking killer of all.

Moreover, while there are no precise measurements available for smokers' concepts of their degree of risk, those health professionals who frequently discuss this with smokers (in clinics, radio phone-in/broadcasts etc.) consistently find that the relative size of the risk of smoking is grossly underestimated by smokers.

For example, it is common to find a 20-a-day smoker estimating his or her chances of contracting lung cancer as being twice or three times that of a non-smoker, instead of 15 to 20 times.

Any association in a health warning between smoking and specific diseases should if possible include a quantification of risk compared to the non-smoker.

**Health Warning - or Warning of Disease?**

It is much more appropriate to talk about death and disease than to talk about health in context of cigarette smoking. It would be quite fair to change the description of the warning to that of a “Warning of diseases and death” or something similar, and the change of nomenclature would be a major talking point which would carry much press comment and generally bring the new warnings more visibly to people’s attention.

In using a panel on the front of a cigarette pack, there could be a constant heading and footing as follows:

**Important - Please read this warning about diseases and death caused by smoking:**

**Warning number ... from U.K. Government Health Departments**
Suggested Warnings

Three sets of warnings are set out in the attached Appendices 1 to 3.

Appendix 1 comprises suggested wordings incorporating some of the concepts dealt with above.

Appendix 2 contains warnings prepared by Richard Peto, distinguished medical statistician and cancer epidemiologist and Reader in Cancer Studies at Oxford University.

Appendix 3 consists of a British Ministry of Defence circular and is included principally to demonstrate the relative strength of health warnings already published by one major Government Department in the U.K.

Needless to say, I should be happy to answer any questions arising from this letter: and to try to supply any further information or explanations you may require.

Yours sincerely,

David Simpson
Director.
Suggested new health warnings

* The smoke of these cigarettes contains carbon monoxide gas, cyanide and other poisons.

* The smoke of these cigarettes contains thousands of different chemicals. Some of them are known to cause cancers when applied to laboratory animals.

* Nicotine in the smoke of these cigarettes makes your heart beat faster, while carbon monoxide gas, also in the smoke, starves you of some of the oxygen needed by your heart and the rest of your body.

* Some of the chemicals in the smoke of these cigarettes stop the natural cleansing mechanism of your lungs from working properly. So apart from the tar you inhale, your lungs cannot clean out all the dust and other forms of pollution you breathe in every day.

* Smoking is by far the biggest cause of avoidable illness and early death in Britain. There is no "moderate" or "safe" level of smoking.

* If you have 'smoker's cough' you may well have the beginnings of chronic bronchitis. Your lungs will already be damaged by your smoking, but if you give up immediately you will stop the damage from getting worse; and your risks of getting the other diseases caused by smoking will also immediately begin to get less.

* If you continue to smoke, you will be more likely to suffer from dangerous chest diseases and to have worse winter coughs and colds, especially as you get older.

* About 9 million in Britain who used to smoke have stopped. Is it time you stopped too? Send for the FREE booklet called 'So You Want to Stop Smoking' which is full of practical advice and help. Write to Health Education Council, 78 New Oxford Street, London W1 or Scottish Health Education Group, Woodburn House, Cansaan Lane, Edinburgh EH10.

* In Britain, tobacco kills about four times as many people as the total killed by drink, drugs, murder, suicide, road accidents, rail accidents, air accidents, poisoning, drowning, fires, falls, snakes, lightning, and every other known cause of accidental death all put together.

* Cigarettes cause lung cancer, bronchitis, emphysema & heart disease. They also cause almost all the cases of arterfial disease in Britain which result in people having to have a limb amputated.
APPENDIX 2

Health warnings prepared by Richard Peto, Imperial Cancer Research Fund Reader in Cancer Studies, Radcliffe Infirmary, University of Oxford.

(Published in Health Education Journal, Vol 39 No.2, 1980
“Possible ways of explaining to ordinary people the quantitative dangers of smoking.

- About a quarter of the young men who regularly smoke a pack or so of cigarettes a day are killed before their time by smoking.

- Some people killed by smoking would have died soon anyway, but others would have lived 10, 20, 30 or more years longer. On average, those killed by smoking have lost 10-15 years of life.

- There is no safe dose of cigarette smoke, although the risks are lower if you smoke less or leave longer butts.

- It is prudent to choose a low-tar brand, although the tar may not be the only killer in smoke.

- If you quit smoking when you have not already got emphysema, cancer or artery disease from smoking, you avoid most of the risk of death from smoking.

- Smoking also causes these diseases in women, but the full effects of long-term smoking in women will not be evident for some years.

- Damage to the body from smoking accumulates. The earlier smoking began, the worse the risks in middle age will be, so those who start in their teens are at greatest danger.

- Among 1,000 young men who smoke cigarettes regularly:
- About 1 will be murdered
- About 6 will be killed on the roads
- About 250 will be killed before their time by tobacco.
The following are among messages suggested for inclusion on no-smoking notices on Ministry of Defence premises (MOD circular BOM65019DCM56F)

* The 20 a day smoker has a 20 times greater risk of developing lung cancer than the non-smoker.

* Lung cancer amongst women who smoke is on the increase and they are at least 5 to 10 times more likely to die of it than women who do not smoke.

* Cancers of the throat, mouth and oesophagus are all associated with smoking.

* Smoking is a major cause of heart disease, chronic bronchitis and emphysema, all killers.

* Over 95% of patients with arterial disease of the legs are smokers. This type of disease can lead to gangrene and amputation.

* The Royal College of Physicians estimates that about 50 million working days a year are lost to industry through smoking related diseases.

* According to government figures smoking causes at least 50,000 premature deaths each year in this country alone.

* The smoker's chance of dying from smoking related disease falls by half within five years and virtually disappears within ten to fifteen years of giving up.

Senator HAWKINS. Senator East.
Senator EAST. Thank you, Madam Chairman.
I think on the good point that my distinguished colleague from Indiana raises, it is suggestive, Madam Chairman, of the problem I want to underscore here in my remarks; namely, that we make sure in this legislation that so-called scientific assertions are in fact, that.

As I assume from the legislation itself; the primary objective is informational, not prohibitional. Assuming that is correct, then we get into a question of behavioral science, as Senator Quayle is suggesting. What, in fact, is the superior form of labeling?

The current label to the effect that the Surgeon General has determined that cigarette smoking is dangerous to your health. I suspect—and I am not trying to declare it to be scientific fact—is one of the better known warnings today in the United States; that many Americans could identify that statement. By constantly repeating that statement, you, in effect, are more informational than if you suddenly developed a series of six or seven or eight rotational labels that are a bit more complex and obviously would be less easily retained by a public that is getting this information either through advertising or a quick glance at a package of cigarettes.

Again, I simply try to underscore as carefully as I can, without going on ad nauseam about it, that all I am asking is that we keep
in mind, one, the goal is informational and, two, whatever we require as a policy matter is consistent with that goal and, indeed, enhances it.

If, as Senator Quayle has indicated, the single warning, either perhaps improved or continually repeated, is more effective as an information instrument of behavioral science, we really ought to do that rather than go to the rotational. The rotational may not, in fact, as a matter of behavioral science, be a better way to inform. In fact, it may lessen the information level, which would be the worst of all worlds, I know from your standpoint, Dr. Brandt.

If we had evidence that was so and we still pursued it, then I would be contending, if I might, that the legislation is no longer desirous of information. It becomes somewhat oblivious to scientific fact and becomes basically just a political document. It seeks, frankly, to be a bit punitive and to simply overregulate an industry for the sake of punitive and overregulating it.

Again, you are out of the informational realm, which we all agree is where we are trying to go, and you are into the prohibitory realm.

I just caution, Madam Chairman, that in doing this we not be so caught up in the emotion of the issue that we not sort out and think through and do this carefully, so we strike the proper balance—one, to inform the public of the relative health concerns of cigarette smoking and, or smoking, whichever it is we are trying to do here, and, two, that we keep in mind our goal is informational and that we are using the best scientific evidence that modern natural and physical sciences can give us and, for the behavioral sciences. I think that is an eminently fair posture to take, and that is all I am doing.

I am not suggesting I can speak ex cathedra for what ought to be the policy goal or end, or the political, nor am I certainly pretending—I would be totally inadequate—that I can speak to natural and physical scientific causation. I am only cautioning that we not let emotion carry us away, where we begin to mix this blend and come up with a heavy brew that no longer does service or credit to policymaking, let alone scientific foundation and causation.

Thank you, Madam Chairman.

Senator Hawkins. Thank you.

Dr. Brandt, do you have any comment?

Dr. Brandt. Yes; I just need to get one thing on the record that I, unfortunately, forgot in my exchange with Senator Quayle. The current label on cigarette packages is mandated by law. Therefore, what we see is any sort of an agreement with the tobacco industry toward a stronger warning, it would require legislative change to permit such a warning to be used, because under current law they have no option but to put it on the cigarette packages. That is different from the advertising.

The second thing is that I think we want to be clear that in our advocacy of stronger warnings that we would certainly want to, one, be scientifically accurate, and two, to be effective at informing and educating the public so that they can make intelligent decisions.

Senator Hawkins. Would you not, therefore, be in agreement that you should have statistics from England, if there are any, with
respect to the multi versus a stronger single, inasmuch as we have a country that has done this?

Dr. BRANDT. We will certainly pursue that. I think in later testimony today, Madam Chairman, you will be receiving information, also, about the experiences in the Nordic countries.

Senator HAWKINS. Thank you. We would dismiss the panel at this time. Thank you for your participation.

Dr. BRANDT. Thank you very much.

(The prepared statements of Dr. Levent, Dr. Lipsett, Dr. Pollin, Dr. DeVita, and Dr. Luoto follow.)
STATEMENT FOR THE RECORD

BY

CLAUDE LENFANT, M.D.
DIRECTOR, NATIONAL HEART, LUNG, AND BLOOD INSTITUTE
NATIONAL INSTITUTES OF HEALTH

Introduction

I am delighted to have this opportunity to discuss the health implications of smoking in relation to the disease concerns of the National Heart, Lung, and Blood Institute (NHLBI). The diseases which our Institute is addressing account for far more than half the deaths in the United States each year. I can think of no single act that could reverse this situation as much as the cessation of cigarette smoking.

Of the more than 300,000 smoking-related deaths each year the vast majority involve the concerns of NHLBI. Smoking is the primary cause of chronic obstructive pulmonary disease, including emphysema, and is also a major cause of coronary heart disease. It is the focus of considerable scientific inquiry by the Institute. The NHLBI sponsors many research programs that explore basic mechanisms involved in the biochemical and physiological changes associated with smoking, as well as studies that evaluate behavioral interventions designed to end the smoking habit itself. In addition, the NHLBI works with other Federal agencies and voluntary organizations in stimulating or conducting professional and public education activities regarding smoking cessation.

Smoking and Coronary Heart Disease

Although many smokers are concerned about the increased risk of lung cancer, the fact is that coronary heart disease (CHD) and its complications, such as heart attacks or sudden cardiac death, are even more prevalent consequences of smoking. Smoking-related CHD deaths are estimated at over 250,000 per year in the U.S., in comparison to about 100,000 deaths from lung cancer. Data from the NHLBI-sponsored Framingham Heart Study
indicate that the death rate from myocardial infarction or heart attacks and sudden cardiac death is twice as great for smokers as for nonsmokers. The Framingham data also indicate that the impact of cigarette smoking on cardiovascular mortality is substantially greater among men than women. Smoking appears to account for 20 percent of the excess cardiovascular mortality among male smokers, and to a lesser extent for excess cardiovascular mortality among women smokers.

The myth of women's immunity to the risks of cigarette smoking is being destroyed. As stated in the 1979 Surgeon General's report, "Women who smoke like men, die like men." The hazards of smoking affect all Americans, young and old, male and female. Moreover, smoking interacts with use of other substances to increase the risk of a variety of diseases. For example, smoking in women who use oral contraceptives further increases their risk of stroke.

How does smoking harm the heart? At present, this question cannot be answered definitively. It is not known which of the many components of cigarette smoke are responsible for the increased risk of a heart attack. Nicotine has a variety of cardiovascular effects, including raising the systolic blood pressure and increasing the amount of free fatty acids in the blood, a consequence which may in turn promote atherosclerosis and contribute to arrhythmias. Carbon monoxide, another smoke constituent, is an inhibitor for hemoglobin, and because of this, reduces the amount of oxygen the hemoglobin can carry and the amount of oxygen available to the heart muscle. Carbon monoxide has also been implicated in the etiology of atherosclerosis because it causes changes in the blood vessel wall.
The question of the relationship between the quantity of nicotine and carbon monoxide in cigarettes and the risk of heart attacks in young men has just been raised in the scientific literature. A recent article in the New England Journal of Medicine suggests that men who smoke the newer cigarettes, which have reduced amounts of nicotine and carbon monoxide, do not have a lower risk of myocardial infarction than men who smoke the old type of cigarettes.

The importance of smoking cessation cannot be overestimated. The results of the NHLBI-supported Multiple Risk Factor Intervention Trial (MRFIT) were recently released. This clinical trial was designed to investigate the effects of reducing three known cardiovascular risk factors—elevated cholesterol, high blood pressure, and cigarette smoking—in a group of asymptomatic men at high risk of coronary heart disease (CHD). Results of this major study showed that the CHD mortality rate in men who quit smoking during the first year of the study was almost 50 percent lower than in those who continued to smoke.

The adverse effects of cigarette smoking on the cardiovascular system are by no means confined to the heart and the coronary arteries. Among patients with peripheral vascular disease affecting the lower limbs (usually due to atherosclerotic deposits in the femoral or popliteal arteries), intermittent claudication (episodes of pain, usually in the calf of the leg, that occur during exercise) tends to occur more frequently and to be more severe if the patient continues to smoke. One peripheral vascular disorder, thromboangiitis obliterans, or Buerger's Disease, is nearly always a direct consequence of smoking in persons highly sensitive
to nicotine. If unchecked, the disorder can lead to loss of digits or limbs, owing to gangrene. Progression of this disease can be halted by smoking cessation, but will inevitably grow worse if an individual continues to smoke. Although smoking is a less potent risk factor for stroke than for coronary heart disease in men, available evidence, as mentioned earlier, indicates that stroke risk is greatly increased among female smokers who also take birth control pills.

Smoking and Respiratory Diseases

Cigarette smoking is the single most important risk factor for chronic obstructive lung diseases, such as chronic bronchitis and emphysema. It also increases vulnerability to acute upper and lower respiratory infections, and a history of frequent acute respiratory infections may well predispose to the development of chronic obstructive lung disease later on. Collectively, these lung diseases are the sixth leading cause of death in the United States. The social and economic toll of diseases of the lung is both severe and rising rapidly. The total economic costs of lung and related respiratory diseases were over $21 billion in 1979, six percent of the total cost of illness in the United States. These diseases are also the leading cause of morbidity costs, representing almost 18 percent of the total. There are more work days lost from respiratory disorders than from any other category of illness.

The hypothesis most widely held concerning the development of emphysema is that the disease results from destruction of lung tissue when there is an imbalance in the lung between protein-digesting enzymes (proteases) and protective enzymes (antiproteases) that normally neutralize the effects of the proteases. Proteases are released in the lung by various
species of white blood cells in the course of their defense against bacteria and other "foreign invaders." Smoking is believed to contribute to imbalances between proteases and antiproteases as follows. Cigarette smoke has been shown to contain substances that stimulate pulmonary alveolar macrophages (the lung's "first line" of defense against foreign invaders) to release a substance that attracts increased numbers of white blood cells to the lung. These cells, which are not usually present in the lungs of healthy non-smokers, carry a potent cargo of inflammatory mediators, including elastase, a protease capable of digesting elastin, which is the major structural protein of the gas-exchange units (alveoli) of lung. Cigarette smoke also contains other substances that markedly inhibit the activity of antiproteases. Among heavy smokers the activity of antiproteases may be less than one-half of normal and thus may be inadequate to deal with the increased amounts of elastase being released by the large population of white blood cells. Elastase in excess of that which can be neutralized by antiproteases may then proceed to destroy the connective tissue and the gas exchange units deep in the lung.

Attesting to the importance of smoking as a risk factor for chronic obstructive lung disease are the results of a study recently conducted among participants in the Tecumseh Community Health Study, a major epidemiological study that has been in progress since 1959. The investigators found that four factors—the subjects' age, sex, smoking habits and vital capacity, which is an index of lung volume—could identify most of the men and women who were in the top 10 percent of the risk distribution for development of emphysema. The investigators showed that a 45-year-old subject who did not smoke and whose vital capacity was normal has only one chance in 200 of developing chronic obstructive lung disease.
During the next 15 years, in contrast, for a similar person who smoked two packs of cigarettes a day and whose vital capacity was 80% of normal, the risk was one chance in 5 or 6 if he continued to smoke, but was reduced to one in 15 if he quit.

Another harmful effect of smoking is that it reduces the effectiveness of the mucociliary mechanisms that clear the airways and lungs of particulate matter such as bacteria, and of excess mucus secretions. Because these excess secretions may provide a good breeding ground for bacterial invaders, the inadequate clearance of inhaled particulates in smokers makes them more vulnerable to lung diseases that result from exposure to infectious organisms, toxic dusts, or allergens. The various forms of asthma associated with worksite (so-called "industrial asthma") occur with higher frequency and are usually more severe among smokers than among nonsmokers exposed to similar environments in their workplace.

Smokers have turned to "low tar" (yet another component of cigarette smoke) cigarettes with the expectation that this would reduce the health hazards of smoking. Currently, available data suggest that this may be true with regard to certain cancers. However, for respiratory diseases the data are more ambiguous. A recent article in the American Review of Respiratory Disease reported on the impact of the tar content of cigarettes on pulmonary function. The data suggest that low tar cigarettes may not protect smokers from the decline in pulmonary function seen in smokers. This finding underlines the importance of quitting smoking as the most effective way to remove the hazards associated with this habit and also corroborates the findings that "less hazardous" cigarettes do not decrease risk of myocardial infarction.

However, available data suggest that this may be true with regard to certain cancers. However, for respiratory diseases the data are more ambiguous. A recent article in the American Review of Respiratory Disease reported on the impact of the tar content of cigarettes on pulmonary function. The data suggest that low tar cigarettes may not protect smokers from the decline in pulmonary function seen in smokers. This finding underlines the importance of quitting smoking as the most effective way to remove the hazards associated with this habit and also corroborates the findings that "less hazardous" cigarettes do not decrease risk of myocardial infarction.
Passive Smoking

For many years it was generally assumed that the smoker hurt nobody's health but his own. Recently, however, evidence indicates that so-called side-stream smoke from somebody else's cigarettes is not just a nuisance for the nonsmoker who is exposed to it, but can produce adverse effects on the nonsmoker's heart and lungs.

As awareness of the diversity of the deleterious effects of cigarette smoking is growing, more attention is focusing on the effects on the non-smoker of breathing air polluted with cigarette smoke, so-called "passive" smoking. A study recently completed has shown that children from homes with smoking parents have a higher incidence of respiratory symptoms and hospital admissions than children with nonsmoking parents. This is important because children who experience respiratory damage early in life are at increased risk of developing chronic lung disease as adults.

Various other studies have shown that nonsmoking patients with coronary heart disease may experience episodes of chest pain or other cardiac disturbances when exposed for a time to a smoke-filled environment, and that long-term occupational exposure to the smoke of fellow employees may cause a decline in lung function among nonsmokers that is roughly equivalent to that resulting from regularly smoking ten cigarettes a day or one pack a day without inhaling. Thus, the recent proliferation of local ordinances banning or limiting smoking in public places has a sound scientific basis.

In conclusion, the biggest challenge we face is to persuade Americans to stop smoking. We know that this can be done. The Multiple Risk Factor

[...]

Variation of...
Intervention Trial mentioned earlier demonstrated the success of smoking cessation programs. In that study, fifty-nine percent of the men were cigarette smokers. Almost half of those receiving counseling in the study group were able to quit smoking. The prevention of the diseases that are caused and exacerbated by cigarette smoking is a most logical, immediate strategy for reducing the large toll of death and disability as well as the huge economic burden in which we all share.

Our prevention strategy must include a vigorous medical and public education effort, but to be a strong prevention strategy, it will also require continued research into the causes of heart and lung diseases.
Mr. Chairman and Members of the Subcommittee:

I am pleased to provide the Subcommittee with information about the relationship between smoking and child health and human development. The research focus of the National Institute of Child Health and Human Development in this area begins before birth, because exposure to components of cigarettes occurs during fetal development if the pregnant woman smokes. A woman who smokes during pregnancy not only risks her own health, but also changes the conditions under which her baby develops. Maternal smoking during pregnancy decreases fetal growth rate, and increases the incidence of spontaneous abortion, fetal death, neonatal death, and other pregnancy complications, including those that predispose to preterm delivery. There are also possible effects on lactation, and adverse long-term growth and behavioral effects on surviving children. The relationships between maternal smoking and these outcomes have been established by clinical, pathological, and especially epidemiological studies. These damaging effects have been repeatedly shown to operate independently of all other factors that influence the outcome of pregnancy. The effects are increased by heavier smoking and are reduced if a woman stops smoking during pregnancy.

Numerous toxic substances in cigarette smoke, such as nicotine and hydrogen cyanide, cross the placenta to affect the fetus directly. The carbon monoxide from cigarette smoke is transported into the fetal blood and deprives the growing baby of oxygen. Fetal growth is directly retarded. Babies born to women who smoke during pregnancy are, on the average, 200 grams (almost half a pound) lighter than babies born to comparable women who do not smoke.

These findings have been confirmed in more than 45 studies which obtained data on more than half a million births. Careful analysis of data obtained...
from five different studies carried out in Wales, the United States, and Canada on almost 113,000 births, where approximately 50 percent of the mothers studied smoked during pregnancy, showed that they had twice as many low-birth-weight babies as the non-smokers. The greater the number of cigarettes smoked, the greater the reduction in birth weight. When examined in greater detail, this dose-response relationship revealed that the adjusted rates of birth weights under 2500 grams (about five pounds) were 49 per thousand for non-smokers, 76 per thousand for smokers of less than a pack per day, and 114 per thousand for smokers of a pack or more per day. Therefore, the risk of having a low-birth-weight baby increased 53 percent for light smokers and 130 percent for heavy smokers, compared with non-smokers. A prospective study of almost 6,000 pregnancies examined the rate of fetal growth from 18-29 weeks through term. The investigators obtained serial ultrasonic measurements of fetal head growth (biparietal diameter or BPD) and constructed separate growth curves for smokers and non-smokers. The BPD increased faster in the non-smoking group, a significant difference was apparent by the 29th week of gestation, and a positive correlation was found to the number of cigarettes smoked by the mother.

Smokers' babies are smaller than corresponding non-smokers' babies in all dimensions measured, including length, head circumference, chest circumference, and shoulder circumference. They are characterized as normally proportioned but short as well as underweight for gestational age, and smaller in all dimensions than babies of non-smokers. Detailed examination of the relationship between maternal smoking and birth weight shows its independence of the large variety of factors that influence birth weight, such as maternal size, maternal weight gain, age, parity, socioeconomic status, and sex of child. It is important to note that thorough analysis indicates that the effect of maternal smoking is not mediated through an effect on maternal appetite, eating...
or weight gain. The analysis of more than 31,000 births did not find a correlation between maternal weight gain and maternal smoking, not even for mothers who gained less than 10 pounds during pregnancy. Similar lack of correlation between maternal weight gain and birth weight was observed in a prospective study of 6,200 pregnant women examined from the first trimester through delivery, although their infants did show the usual relationship between smoking and small-for-dates babies.

Studies have shown that placental weight is not affected by maternal smoking, but the ratio of placental weight to birth weight is larger for nonsmokers. This is easily understood in view of the observed reduction in birth weight. Data from 7,000 pregnancies in California demonstrated that from 37 through 43 weeks the more the mother smoked during pregnancy, the higher was the placental ratio. Smokers' placentas are thinner than those of nonsmokers and have larger diameters. These changes could represent an adaptation to relative fetal hypoxia (decreased oxygen) resulting from an observed reduction in oxygen carrying capacity of the blood in women who smoke. In other words, the proportionally larger placentas, the greater area of attachment, and a smaller fetus are compensatory mechanisms for the effects of smoking. In current studies, the architecture of placentas from smoking and non smoking women are being examined to determine the presence or absence of specific lesions. Results will be available next year.

Another important finding is the association between smoking and spontaneous abortion risk. Women who smoke more than 20 cigarettes a day are almost twice as likely as nonsmokers to have had a spontaneous abortion. This association, found in a small retrospective study, cannot be explained by differences in maternal age, educational level, parity, race, socioeconomic
status or marital status. The risk of spontaneous abortion for subgroups of heavy smokers among more than 12,000 professional women was estimated to be 1.7 times that for nonsmokers. The relative increase in risk of spontaneous abortion associated with maternal smoking was highest at the youngest ages and decreased with increasing age.

Also a direct relationship has been demonstrated between level of maternal smoking and risk of perinatal death. Studies have consistently found a direct relationship between maternal smoking level and incidence of placenta previa (placental blocking of the opening of the uterus), abruptio placentae (premature separation of the placenta from the uterus), bleeding during pregnancy, and premature rupture of membranes. The association is independent of socioeconomic and racial background, and of parity. These complications carry with them a high risk of fetal and neonatal loss, and are frequently cited as the cause of death among the offspring of women who smoke. Abruptio placentae was found to occur frequently among smokers whose children died before one week of age. A large number of stillborns among smokers was attributable to the same cause in a prospective study of more than 9,000 pregnancies. Additionally, the same association was identified in the Collaborative Perinatal Project of the NIH/DS where abruptio placentae was the underlying cause for 11 percent of all deaths. Therefore, maternal smoking can be a direct cause of fetal or neonatal death in an otherwise normal infant.

The immediate cause of most smoking-related fetal deaths is probably anoxia, which can be attributed to placental complications with antepartum bleeding in 30 percent or more of the cases. In other cases, the oxygen supply may simply fail from reduced carrying capacity and reduced unloading pressures for oxygen caused by the presence of carbon monoxide from cigarette.
smoke in maternal and fetal blood. Neonatal deaths occur as a result of the increased risk of early delivery among smokers, which may be secondarily related to bleeding early in pregnancy and premature rupture of membranes. Up to 14 percent of all preterm deliveries in the United States may be attributable to maternal smoking.

Studies of infant and child morbidity and mortality in relation to the mother's smoking habits usually cannot distinguish between the effects of smoking during pregnancy and the effect of the infant's or child's passive exposure to cigarette smoke after birth. Several studies have found that hospitalization rates for pneumonia and bronchitis were higher during the first year of life for infants of smoking mothers. Rates in children were higher if the smoking parents also had a persistent cough and phlegm. The risk of contracting pneumonia in the first year of life more than doubles if both parents smoked more than 24 cigarettes a day. An important study of morbidity and mortality in smokers' and nonsmokers' children up to age five was completed encompassing a population of over 12,000 children. Postneonatal mortality, from 28 days to 5 years, was higher for smokers' children with a rate of 11.1 per thousand in contrast to that of 3.9 per thousand for nonsmokers. Neonatal deaths, from birth to 28 days of age, were 12.6 per thousand for smokers and 5.8 per thousand for nonsmokers. In addition, the children of the smokers were hospitalized more frequently, had more visits to doctors, and had longer average durations of hospital stays than children of nonsmokers. Respiratory diseases caused more hospitalizations among smokers' children.

Another significant problem has been recognized recently for children exposed to household cigarette smoke. They have as much as a fourfold increase in their risk for persistent middle-ear effusions following ear infections. The persistent effusion may cause hearing loss which, in turn, has been associated
Long-term consequences of maternal smoking during pregnancy are also of concern. Several long-term studies provide some evidence that children of smoking mothers have slight but measurable deficiencies in physical growth, intellectual and emotional development, and behavior.

Because these complex outcomes are affected by many known and unknown factors, it is important to take these other factors into account in any attempt to measure long-term effects of maternal smoking. Several well-controlled studies have shown that the physical growth of smokers' babies remains behind that of nonsmokers' babies when measured between 7 and 14 days; at 1 year, 4 years, and 7 years (pairs of births matched for race, date of delivery, maternal age and education, and sex of child); and at ages 7 and 11 years (follow-up studies of 17,000 children from the British Perinatal Morbidity Study). The latter study showed that at ages 7 and 11 years, physical and mental problems were associated with maternal smoking during pregnancy, and these intensified with the number of cigarettes smoked.

Another study provided evidence that children whose mothers smoked 10 or more cigarettes a day during pregnancy were on average 1.0 centimeter shorter and 3 to 5 months retarded in reading, mathematics, and general ability, as compared with the offspring of nonsmokers. Even after allowing for associated social and biological factors, all of these differences were highly significant.

A case-control study of hyperactivity in children has found a significant association with heavy maternal smoking. At a mean level of 23.3 cigarettes per day the association with smoking mothers was more than three times the average for nonsmokers.
Maternal smoking habits have been examined in several studies of the sudden infant death syndrome (SIDS). In all of these, one of several associations has been maternal smoking during pregnancy and SIDS. Reports include the observation that not only a higher proportion of SIDS mothers smoked during pregnancy than controls, but more smoked after pregnancy as well. A study of 80 SIDS cases and 160 matched control infants from the Ontario Perinatal Mortality Study population found that only 39 percent of the mothers of SIDS victims were non-smokers, in contrast to 60 percent for the controls. 36 percent of the cases and 27 percent of the controls smoked less than a pack per day, 24 percent of the cases and 10 percent of the controls smoked a pack or more per day. Analysis of data from the prospective study of 29,047 births also showed a strong association of SIDS with maternal smoking. In the SIDS group, 70.6 percent of mothers smoked during pregnancy, compared with only 35.3 percent of mothers of babies who did not die of SIDS. Clearly, the relative risk of SIDS for the infants of smokers versus non-smokers is significant. The exposure to cigarette smoke (passive or second-hand smoking) appears to enhance the risk for SIDS for reasons not yet known. However, whether prenatal or postnatal exposure is more important for the observed strong association has not been determined with any certainty.

Several epidemiologic studies have suggested that smoking decreases fecundity in women. In a carefully designed study where several characteristics (cause of death, age at and year of death, education, occupation and frequency of marriage, as well as husbands' smoking habits, education and occupation) were controlled, a 46 percent greater incidence of infertility was found in women who smoked. Secondary amenorrhea (cessation of menstruation) was more prevalent among smokers (4.8 per 100 women) than among non-smokers (1.7 per 100 women).

Substantial data demonstrate that smoking lowers the age of spontaneous...
menopause. The mechanism may be related to ovotoxicity in cigarette smoke or caused by toxic alterations in hormone regulation. Earlier menopause is not related to weight differences between smokers and nonsmokers but is apparently a direct result of some component of cigarette smoke. Smoking also seems to interfere with several phases of male reproduction, but the mechanism responsible for the alterations in spermatogenesis and hormone levels has not yet been clarified. I should point out that the developing ovary and testes in the fetus are particularly sensitive to noxious substances. Whether smoking during pregnancy will exert additional effects on the reproductive system can only be conjectural at present.

Thus far, I have been speaking about the deleterious effects of cigarette smoking in pregnant women and their offspring, and on the reproductive system of both men and women. Another age group of critical importance is the young, adolescent smoker. Epidemiological data from the 1979 National Institute of Education Survey of Teenage Smoking indicate that in this country, 3.5 million adolescents between 12 and 18 years of age are “current regular smokers” (youth who smoke one or more cigarettes per week). A more recent national survey of high school seniors (NIDA/University of Michigan, 1982) indicates that 21.1 percent of this group (boys and girls) report daily use of cigarettes and 30.0 percent report having used cigarettes at least once in the past month.

The most alarming trend is that teenage girls have, for the first time, a higher reported incidence of smoking than adolescent boys. This statistic is of great importance because, in my opinion, pregnant teenagers, who are already a high-risk group for bearing low-birthweight infants, are likely to increase this risk even further by smoking cigarettes during pregnancy.

Furthermore, cigarette smoking begun in adolescence is likely to lead to
life-time use. Those people who start smoking in their teenage years find it more difficult to stop smoking than those who begin in adulthood. Also, the Surgeon General's 1979 report, "Smoking and Health" documented that, overall, mortality rates are higher for those who initiated their smoking in adolescence, as compared to those who started later in life.

Research findings, to date, reveal that among the most important factors that influence experimentation with, and acquisition of, smoking are social and familial relationships. Parents who smoke are likely to have children who smoke, and teenagers with two smoking parents are more than twice as likely to smoke as those with non-smoking parents. Teenagers also emulate older brothers and sisters. A boy or girl with an older sibling who smokes is three times as likely to be a smoker as well. Teenagers with both a parent and an older sibling who smoke, are four times as likely to smoke as those who have no smoking example in the family. However, a number of studies have indicated that the presence of a best friend who smokes is the single most important predictor of smoking in children from the 5th through the 12th grades.

Cigarette smoking is perceived by youngsters in the early school years to be bad for health. They know it can cause cancer and is associated with lung and heart disease. Most of the adolescents who smoke concede that it is unhealthy and habit-forming, and that it slows them down in sports. Although they feel uneasy about smoking, many continue. A number of theories have been offered to explain why neither knowledge nor fear is adequate to counteract the pressures that surround children as they reach puberty and enter adolescence. One such idea is that children have a time perspective problem. Since they live in the present and feel indestructible, risks to their health so far in the future appear to have no consequences for them. Also, teenagers share a strong
perception that "everybody does it." The facts are otherwise, however, for most children and adults do not smoke.

Currently, the NHLBI supports seven behavioral studies on teenage cigarette smoking. They focus primarily on identifying the determinants of the onset of smoking and understanding the process which leads to habitual smoking. A significant research finding has indicated that teenagers tend to underreport their smoking activity when only asked for self reports. However, when a biological test (generally salivary thiocyanate, a derivative of the hydrogen cyanide obtained from the cigarette) is used in conjunction with self reports, adolescents report higher levels of cigarette smoking (1.5 - 3.5 times higher) than when no biochemical tests are used.

Prevention efforts to deter the onset of cigarette smoking in children are generally school-based programs. These programs often include the use of films and role-playing to depict real-life situations and demonstrate ways to resist peer and adult social pressures to smoke, usually followed by teacher and peer-led discussions. A more generalized school curriculum is the Life Skills Training Program. This approach is designed to facilitate the improvement of teenagers' general personal competence, and to help them develop greater autonomy and self-esteem. This new competence helps strengthen their ability to resist pressures to smoke. Initial studies indicate that this approach may be able to reduce new cigarette smoking by at least 30 percent. The long-term maintenance of non-smoking in 1,200 junior high school students over a two-year period, with and without additional booster sessions, is currently being evaluated.

Prevention efforts also include smoking cessation programs for pregnant
women. In a recently completed NICHD-supported study, the investigator found that women who quit smoking, even in the last trimester of pregnancy, are less likely to have low-birth-weight infants than those women who continue to smoke throughout pregnancy. The findings are based on a randomized clinical trial study of large groups of pregnant women, half of whom received an active intervention program to encourage and assist them in smoking cessation during pregnancy.

I appreciate the opportunity to provide this information on a topic the NICHD considers to be of great importance.
STATEMENT FOR THE RECORD

by

WILLIAM POLLIN, M.D.
Director

National Institute on Drug Abuse
Alcohol, Drug Abuse, and Mental Health Administration
Rockville, Maryland 20857

Mr. Chairman and Members of the Committee, thank you for the opportunity to testify on the addictive properties of tobacco smoking. Today, several authoritative medical bodies—including the World Health Organization in its International Classification of Diseases (4th Edition) and the American Psychiatric Association in its Diagnostic and Statistical Manual of Mental Disorders (3rd Edition)—recognize cigarette smoking as a form of drug dependency. Careful reviews of our own laboratory data from research scientists within and outside of Government have led us to concur with those authoritative bodies. I am submitting the proceedings of cigarette smoking reviews for the record.

Briefly, research indicates that cigarette smoking is a prototypic dependence process; in fact it is the most widespread example of drug dependence in this country. Current scientific research increasingly implicates nicotine as a primary factor in maintaining a dependence on tobacco.

In order to determine the abuse liability of a substance, scientific studies must document that it has psychoactive and behavioral effects, reinforcing properties (e.g., demonstrated euphoriant effects), and that its use maintains drug-seeking behavior.

With regard to the first criterion, nicotine has been found to be a psychoactive substance; that is, it influences subjective states and behavior. Most frequently, subjects report enhanced feelings of relaxation, alertness or attention, and concentration.

105
The reinforcer properties of nicotine are due to physiologic as well as euphoriant effects. NIDA's Addiction Research Center (ARC) has developed a test for precisely measuring psychological and subjective drug effects. This test has been administered to over 5,000 individuals, both with and without drug abuse histories. These studies documented the marked similarity that exists between the euphoriant effects of morphine, cocaine, and nicotine when it is administered intravenously. During the first several minutes after administration of each of these drugs, there is an immediate and marked change in feeling-state, namely euphoria. During this period, morphine, cocaine, and nicotine all "feel" very much alike in that the subject experiences a "rush." After a few minutes, this "rush" shows a variable course specific to each of the drugs.

Nicotine has been found to be a reinforcer for animals. This is particularly persuasive evidence, as only the most addictive drugs serve as reinforcers in animal models where drug-seeking behavior must be maintained in the absence of any social support or personality function. Documented biologic effects of nicotine include stimulation of the release of norepinephrine, epinephrine, growth hormones, cortisol, vasopressin, and probably beta endorphin. In addition, behavioral arousal and EEG alerting patterns have been found.

Physiologic dependence is a condition which sometimes accompanies abuse liability but is not a necessary condition for it. Thus, cocaine has a very high abuse potential, but does not show physiological dependence. Evidence is
Tolerance encourages the user's tendency to continue drug use. Tolerance, for instance, reduces the pharmacological effects of drugs and may lead to more frequent administration and/or higher doses, which in turn may produce greater health and social consequences for the user. As most of us know, nausea and dizziness are common among novice smokers, but disappear with experience. Not only has tolerance to some of the effects of smoking been demonstrated, but metabolic tolerance to various components of cigarette smoke, including nicotine, has been documented.

In addition, withdrawal signs appear when heavy smokers quit abruptly. There is some variability in withdrawal symptoms, but it is not unusual for an individual who stops smoking to show a decrease in excreted epinephrine and norepinephrine and their metabolites. There is also a decrease in mean EEG activity and in heart rate, an increase in appetite and weight, and an impairment in performance on psychomotor tasks and in concentration. Disturbance in sleep may occur, and the individuals may feel anxious, irritable, and even depressed. Finally, most individuals who are trying to stop feel an increased craving for tobacco smoking.

The important point which must be stressed in the discussion of any psychoactive drug is the relative degree of control which that drug is able to achieve over
the behavior of its users. "Dependence" in the classic sense is indicated by (1) persistent, regular use of a drug, (2) attempts to stop such use which lead to discomfort and often result in termination of the effort to stop, (3) continued drug use despite damaging physical and/or psychological problems, and (4) persistent drug-seeking behavior. In short, people are drug dependent when a drug takes over and controls their ability to choose to take the drug or not.

In terms of these characteristics of dependence, data from the National Center for Health Statistics (1980 Health Interview Survey) tell us that 99 percent of those who say they smoke tobacco so regularly. Seventy percent of those who report current smoking say they smoke more than 15 cigarettes a day, an amount which establishes persistent regular use. Nine out of ten smokers say they would like to quit smoking; 6 out of 10 have tried, and of that group between 80 and 85 percent say they have been unable to quit for more than three months. This pattern then establishes loss of control and persistent drug-seeking behavior and is consistent with "dependence" in the classic sense.

The prototypic nature of nicotine dependency is further established by the striking similarity between relapse rates for alcohol, heroin, and cigarette smoking as plotted over time. As originally reported by Hunt, Barnett, and Branch in the Journal of Clinical Psychology (1971, 27: 455-456), there is for alcohol, heroin, and smoking the same rapid relapse rate following treatment to the point of abstinence, breaking sharply somewhere between 3 and 6 months and asymptotically or stabilizing as a flat line around 28 percent.
Among those who have the greatest personal exposure to the health consequences of smoking, the health professionals, the decline in smoking has been most dramatic. The overall quit rate for physicians in 1975 was 64 percent, and studies have shown that the rates varied by medical specialty. More recently, the highest quit rates were reported for pathologists and thoracic surgeons who routinely see the clinical effects of cigarette smoke, while the lowest rates were reported for pediatricians and psychiatrists who rarely see these health consequences. This demonstrates the power of repeated reinforcement of health risk data on smoking behavior.

The call for new cigarette warning labels coincides with newly released findings that the declining trend in cigarette use by high school seniors has slowed. Between 1977 and 1981, cigarette use by high school seniors declined. Daily smoking dropped from 20 to 10 percent between 1977 and 1981, and daily use of half-a-pack or more fell from 19.4 percent to 13.5 percent. The data indicate that this drop in daily smoking rates was in response to both personal concerns about health consequences of use and perceived peer disapproval of use. The rate of this substantial decline in cigarette use by high school seniors slowed in the 1981 survey, and 1982 data indicate that the decline may have halted. Along with this change in usage trends, significantly fewer high school seniors (60.5 percent in 1982 versus 63.3 percent in 1981) reported believing there was great risk in smoking one or more packs of cigarettes per day and significantly fewer reported believing that their friends would disapprove of smoking one or more packs of cigarettes a day (70.3 percent in 1982 versus 73.9 percent in 1981). Further, this failure to demonstrate a continued decline in the rate of
Cigarette use by youth stands in contrast to the continuing decline in the rates of use for all other drugs.

This reversal in the decline in cigarette smoking by youth is of concern to us both for the predictable health consequences this generation will face in the future and for the implication increased cigarette use trends have for trends of illegal drug use. Historically, cigarette use has indicated the direction of illicit drug use trends by youth, and longitudinal studies have identified tobacco use as an integral and crucial step in the sequences of developing drug use patterns.
Mr. Chairman, I am pleased to present today my prepared statement on the relationship between cigarette smoking and cancer. It may interest you to know that 150 years ago lung cancer was a medical curiosity. Mortality linked to this form of cancer was virtually nonexistent. Today we estimate that over 100,000 people die each year from lung cancer, the most obvious smoking-related cancer. For men in this country, lung cancer is the principal form of cancer mortality, and we have every indication that in the near future lung cancer will replace breast cancer as the prime cause of cancer deaths in women. Our studies indicate that more than 85 percent of lung cancer is causally related to cigarette smoking, making it one of the most preventable forms of cancer we can identify.

The link between cigarette smoking and most lung cancer is well documented. Smokers who consume two or more packs of cigarettes daily have lung cancer mortality rates 15 to 25 times greater than nonsmokers. Inversely, former smokers who have quit 15 or more years have lung cancer mortality rates somewhat above those for nonsmokers. A better understanding of the specific links between cigarette smoking and lung cancer and of successful methods of prevention and of intervention in the cancer process is essential given the mortality rates and the fact that the overall five-year survival rate for all lung cancer patients is quite low—9 percent. In those individuals where the cancer has spread and malignant lesions have been found in distant parts of the body, the survival rate drops to 1 percent.

Lung cancer, however, is not the only outcome from smoking cigarettes or using tobacco products. Cigarette smoking also is a major cause of cancers of the larynx, oral cavity, and esophagus; and smoking is a contributory factor for the development of cancers of the bladder, pancreas, and kidney.
The use of the term contributory in no way excludes the possibility of a causal role for smoking and cancer of these sites.

In order to put in perspective the serious consequences related to cancer from smoking, the following statistics are worthy of consideration. They were collected through the National Cancer Institute's Surveillance, Epidemiology, and End Results Program (SEER).

- Tobacco's contribution to all cancer deaths is estimated to be 30 percent, accounting for the deaths of approximately 130,000 Americans annually.
- It is estimated that 13,000 individuals will die in 1983 from laryngeal and oral cancers. Fifty to 70 percent of these oral and laryngeal cancer deaths are estimated to be causally related to smoking.
- In 1983 estimates are that 8,500 deaths will be caused by esophageal cancer. Only four percent of patients diagnosed with this form of cancer survive 5 years or more. Most patients die within 6 months. Cigarette smoking is estimated to be a factor in over half of these deaths.
- In 1983 more than 56,000 people will develop bladder and kidney cancer. Over 19,000 individuals will die in 1983 from these forms of cancer. Various investigators have estimated that between 30 and 40 percent of bladder cancers are smoking related.
- Finally, more than 25,000 cancers of the pancreas will develop in 1983. Nearly 23,000 individuals will die from cancer of the pancreas this year. Only 3 percent of those afflicted can anticipate surviving 5 years.
Given the nature of these statistics, the National Cancer Institute (NCI) relies on a variety of scientific avenues for studying the relationship between cigarette smoking, and tobacco in general, and cancer. Through basic laboratory science, epidemiological studies, and human intervention trials, the NCI has mounted an extensive multi-method effort that is producing important information about smoking-induced cancer.

NCI intramural scientists have developed the methodology to culture epithelial tissues and cells from the major tissue sites of human cancer caused by tobacco smoke. Bronchial epithelial tissues and cells can now be successfully grown in chemically defined environments for months or even years. This methodology is essential for studies of the in vitro malignant transformation of human lung cells. Using cultured human target epithelial tissues and cells (bronchus, esophagus, and bladder), our investigations have shown that 1) representative carcinogens from the major chemical classes found in tobacco smoke are enzymatically activated to electrophilic metabolites that form adducts and damage DNA; 2) both the metabolic pathways of activations and carcinogen-DNA adducts in humans are qualitatively similar to those found in experimental animals; and 3) quantitative differences in the metabolism of carcinogens are found among different individuals. These investigations are another source of confirmation for the ever-increasing scientific interest in and significance of genetic transformation and damage to DNA in the development of cancer. They also provide the foundation for biochemical-epidemiological studies that are testing the important hypothesis that certain individuals who have a high activation to deactivation ratio in the metabolism of chemical carcinogens are at an increased risk for developing cancer.
One of the major results of epidemiological research is the identification of high-risk populations for the development of cancer. Smoking behavior is an important factor leading to a categorization of "high risk." In recent years epidemiological research has shown that:

- Nonwhites are more likely to smoke than whites by a considerable margin;
- Prevalence-smoking rates for blacks are higher than for all other racial groups for which data are available;
- Smoking prevalence rates are especially high for blue-collar workers and the unemployed;
- The prevalence of male smoking decreases as income level increases;
- In specific occupational settings, the synergistic effect of smoking and exposure to known carcinogens (e.g., asbestos) results in a multiplied risk of enormous proportions for lung cancer—as high as 92 times greater than that for the nonsmoker not exposed to asbestos.

NCI epidemiologists, after developing a cancer atlas of the U.S., have taken the next step and have pursued case-control investigations in various sections of the country. One large series of studies on lung cancer in coastal areas of Georgia and Florida and on lung cancer and mesothelioma in Tidewater, Virginia, found significantly increased risks associated with employment in the shipbuilding industry (and presumed asbestos exposure), particularly during World War II. Recently completed studies in Jacksonville, Florida, and Bath, Maine, support the findings from the Virginia and Georgia studies which indicate that work in shipyards during World War II has resulted in an increased risk of lung cancer during the 1970's, with the excess being greatest among shipyard workers who were heavy smokers. When data from all four studies are combined, the relative risk for shipyard employment is 1.4, suggesting that as many as 100,000 extra lung cancer deaths may eventually result from the intersection of smoking and occupational exposure among the cohort of 4.5 million Americans involved in
wartime shipbuilding activities.

Ongoing case-control studies of lung cancer in Louisiana, Texas, New Jersey, and Pennsylvania are examining how smoking may relate to other occupational and non-occupational risk factors, including low dietary intake of beta-carotene and vitamin A. Data from Pennsylvania indicate that an increased risk among long-term steel workers is greatest for heavy smokers, suggesting a synergistic effect between polycyclic aromatic hydrocarbons or other workplace exposures and cigarette smoking.

To help explain the clustering of high mortality rates for kidney cancer in the north central states, a case-control study involving interviews with nearly 600 kidney cancer patients and twice as many controls was conducted. Cigarette smoking was shown to be the major risk factor for cancer of the renal pelvis. Smokers were at 6 times the risk of this tumor as nonsmokers with an increase exceeding 10-fold for heavy-smoking men and women. Additional studies are underway to examine smoking in relation to cancers of the pancreas, uterine cervix, and other sites for which there is some suspicion that smokers may be at increased risk.

NIH studies of cancer of the esophagus (in Washington, D.C.), oral cavity and pharynx (in North Carolina), and larynx (in Virginia) have further documented that both alcohol and smoking are major determinants of these cancers. Smoking was a contributing cause to the markedly elevated rates for esophageal cancer among black men in Washington, D.C., although heavy alcohol consumption was a more potent risk factor in this population. The combined or multiplicative effects of smoking and drinking were responsible for most oral tumors among nonusers of snuff in the North Carolina study.
as they were for cancers of the larynx in the Virginia study.

An area of increased interest in cigarette-related cancer research is that of "passive" or "side-stream" smoking. The Institute is supporting three ongoing interview studies (in Texas, Louisiana, and in Japan [Hiroshima and Nagasaki]) that address the issue of whether inhalation of side-stream smoke by nonsmokers may increase the risk of lung cancer. The study in Japan will obtain information on childhood and adult exposures to smoke in the homes of women with lung cancer, and will offer results that can be compared with the investigation by the Japanese National Cancer Center which originally suggested that nonsmoking wives of smokers may experience over twice as much lung cancer as expected. Studies of passive smoking are important in providing clues to the etiology of lung cancer among non-tobacco users and will be of significant public health impact should the initial reports of increased lung cancer among nonsmoking wives of smokers be confirmed.

Mr. Chairman, through the NCI Smoking, Cancer, and Health Program (SCHP) the Institute is developing an aggressive, forward plan of action that will result in major new research and program initiatives over the next several years. Recent discoveries in basic biology and recent trends reflecting a decrease of smoking by men-and women indicate the importance of continuing to develop and improve prevention and intervention strategies that can be used with individuals exposed to carcinogens.

Working groups of recognized experts have been formed to review current activities and to make specific program recommendations in five areas: (1) prevention programs targeted at school-age youth, (2) the use of mass media to influence and reinforce appropriate prevention and cessation
behaviors; (3) opportunities for stimulating "self-help" strategies of cessation; (4) the role of health professionals (particularly physicians) as exemplars and interveners in prevention and cessation programs, and (5) smoking behavior in minority populations.

Scientific documentation is strong, indicating that one is less likely to develop certain cancers if one does not smoke. The NCI supports a number of projects aimed at providing information to young people who face the decision of smoking initiation. Most people make the decision to smoke cigarettes when they are young and not well informed about the consequences of such a decision. "Cancer" may not appear to be a real danger to a young person. However, given the apparent addictive characteristics of nicotine and the behavioral dependence that is well documented, young people who choose to smoke also are making a choice that increases their risk of developing cancer. Adults are more likely to make decisions about cessation. Therefore, it is important to explore prevention and intervention strategies that can be applied to young nonsmokers, smokers, and individuals of all ages exposed to cigarette smoke.

The National Cancer Institute continues its efforts to provide the public with information about the dangers of cigarette smoking. The Institute engages in a public information program through its Office of Cancer Communications. Each year informational messages and smoking prevention/cessation tools are distributed nationwide. Materials for community planners, leaders, and health activists are provided in The Smoking Digest. This publication, initiated in 1977, provides information on attitudes about smoking cessation techniques, smoking information campaigns, smoking legislation, and the tobacco industry. In addition, helping smokers quit
kits have been developed and are distributed to physicians and dentists throughout the United States. Similar materials are now under development for pharmacists. Materials for special audiences (e.g., minorities, persons at high risk) are available or under development as well as instructional programs for educators, school administrators, and concerned parents.

In conclusion, I think that the scientific base linking cigarette smoking to certain cancers, frequently those most difficult to treat, is unambiguous. Smoking is detrimental to health. However, it is clear that prevention can be effective. The prevalence of smoking has declined from 42 percent of the population in 1965 to about 30 percent today. This dramatic change in the public's behavior occurred following the continued releases of the Surgeon General's reports starting in 1964 and the corresponding information campaigns of public and private agencies. A combination of accurate information in combination with effective intervention methods related to cigarette smoking can decrease the incidence of certain forms of cancer.

This concludes my statement, Mr. Chairman.
STATEMENT FOR THE RECORD

by

Joanne Lurto, M.D., M.P.H.
Director
Office on Smoking and Health
Rockville, Maryland 20857

The past several years have been marked by increased public attention to the question of passive smoking. This has resulted in a wide response by local and State governments and by private employers and merchants in establishing no-smoking areas where those who wish can be protected from the nuisance and in some cases the health risks resulting from exposure to other people's smoking. Ideally, the regulations and policies which have been adopted by these public and private agencies have also taken into consideration the rights of individuals who smoke.

There is ample evidence that no-smoking areas are welcomed by the public provided they perceive the no-smoking regulations or policies as fair and sensible. In a study commissioned several years ago by the cigarette companies, the question was asked whether no-smoking should be enforced in a number of public and private areas, with the following results:

"Here is a list of some different kinds of places. For each one, would you tell me whether you think (separate/segregated) sections or facilities should be provided for smokers and non-smokers, or whether smokers should be allowed to smoke anywhere?"
<table>
<thead>
<tr>
<th>Category</th>
<th>Total</th>
<th>Total Smokers</th>
<th>Ex-Smokers</th>
<th>Never Smoked</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TRAINS/AIRPLANES/BUSES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>separate/segregated anywhere</td>
<td>50</td>
<td>84</td>
<td>92</td>
<td>93</td>
</tr>
<tr>
<td>anywhere</td>
<td>8</td>
<td>14</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>don't know/no answer</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td><strong>EATING PLACES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>separate/segregated anywhere</td>
<td>72</td>
<td>58</td>
<td>76</td>
<td>82</td>
</tr>
<tr>
<td>anywhere</td>
<td>24</td>
<td>40</td>
<td>72</td>
<td>16</td>
</tr>
<tr>
<td>don't know/no answer</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td><strong>THEATRES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>separate/segregated anywhere</td>
<td>83</td>
<td>76</td>
<td>86</td>
<td>86</td>
</tr>
<tr>
<td>anywhere</td>
<td>12</td>
<td>13</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>don't know/no answer</td>
<td>6</td>
<td>6</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td><strong>WORK PLACES/OFFICES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>separate/segregated anywhere</td>
<td>60</td>
<td>43</td>
<td>58</td>
<td>70</td>
</tr>
<tr>
<td>anywhere</td>
<td>34</td>
<td>46</td>
<td>35</td>
<td>25</td>
</tr>
<tr>
<td>don't know/no answer</td>
<td>6</td>
<td>6</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td><strong>INDOOR SPORTING EVENTS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>separate/segregated anywhere</td>
<td>72</td>
<td>62</td>
<td>75</td>
<td>76</td>
</tr>
<tr>
<td>anywhere</td>
<td>23</td>
<td>33</td>
<td>21</td>
<td>16</td>
</tr>
<tr>
<td>don't know/no answer</td>
<td>5</td>
<td>5</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td><strong>PUBLIC MEETINGS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>separate/segregated anywhere</td>
<td>67</td>
<td>55</td>
<td>67</td>
<td>76</td>
</tr>
<tr>
<td>anywhere</td>
<td>28</td>
<td>39</td>
<td>28</td>
<td>18</td>
</tr>
<tr>
<td>don't know/no answer</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td><strong>BARBER OR BEAUTY SHOPS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>separate/segregated anywhere</td>
<td>51</td>
<td>39</td>
<td>52</td>
<td>62</td>
</tr>
<tr>
<td>anywhere</td>
<td>43</td>
<td>56</td>
<td>42</td>
<td>32</td>
</tr>
<tr>
<td>don't know/no answer</td>
<td>6</td>
<td>5</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td><strong>TRAIN/PLANE/BUS STATIONS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>separate/segregated anywhere</td>
<td>61</td>
<td>48</td>
<td>63</td>
<td>71</td>
</tr>
<tr>
<td>anywhere</td>
<td>30</td>
<td>48</td>
<td>35</td>
<td>25</td>
</tr>
<tr>
<td>don't know/no answer</td>
<td>4</td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
</tbody>
</table>

Source: The Roper Organization Inc. Study 4644, 1976
As will be seen, absolute majorities of the public questioned in this survey, smokers and non-smokers alike, supported policies which would limit cigarette smoking in a majority of the locations cited. Numerous other surveys have shown that people exposed to cigarette smoke report eye irritation, headache, nasal symptoms, and cough. Obviously, passive smoking is annoying and discomforting to many people.

There are other, perhaps more compelling reasons, however, why passive smoking is an important issue.

The Public Health Service reviewed the health significance of passive smoking in detail in its 1979 Report on the health consequences of smoking, and this and more recent information establish the following conclusions:

1. Some of the potentially dangerous ingredients in cigarette smoke are present in greater quantities in sidestream smoke than in mainstream smoke. Levels of cigarette smoke constituents, including carbon monoxide, can be reached in cigarette-smoke-filled rooms which may adversely affect the very large numbers of men and women, many of them elderly, who suffer from chronic lung disease or cardiovascular diseases.

2. Passive smoking can exacerbate the symptoms of asthma and chronic bronchitis. It is unclear whether a tobacco smoke allergy exists as a disease entity, but tobacco smoke can trigger symptoms in people with existing allergic conditions.

3. In addition to the effects of involuntary smoking on people with existing disease, recent studies suggest that passive smoking may contribute to disease in healthy nonsmokers. A 1980 study reported that chronic exposure of nonsmokers to cigarette smoke in the workplace significantly reduced small airways function. While we do not know at this time whether such an effect results in disease in healthy people, we cannot assume that it is an innocent effect. In 1981, three
studies reported an increased risk for lung cancer in nonsmoking wives of smoking husbands, although the results of one of the three studies were not statistically significant. In reviewing these studies, which are described in detail in the Public Health Service's 1982 Report on the relationship between smoking and cancer, Dr. Edward N. Brandt, Jr., the Assistant Secretary for Health, stated that "while the nature of this association is unresolved, it does raise the concern that involuntary smoking may pose a carcinogenic risk to the nonsmoker... For the purpose of preventive medicine, prudence dictates that nonsmokers avoid exposure to second-hand tobacco smoke to the extent possible."

An aspect of passive smoking which is not generally recognized is the effect of such smoking on infants and children. A recent analysis of data from the National Health Interview Survey has shown, for example, that children under 17 who live with smoking parents had more days of restricted activity and bed disability due to acute respiratory disease than did children in families with no smokers. Other studies have shown an increased incidence of pneumonia and bronchitis in the first year of life among children of parents who smoked as well as increased prevalence of childhood asthma in children of smoking parents.

It would appear, as a former Surgeon General stated several years ago, that henceforth, when we speak of the rights of nonsmokers, we must extend our concern into the nursery.
Senator Hawkins. We would also like to recognize the fact that the American Cancer Society and the American Heart Association have worked closely with the Lung Association in putting this hearing together.

The third panel will consist of three distinguished physicians who will cover the issues pertaining to health-related effects of smoking on women and children and a nationally known television personality who is an expert in children's education.

Senator Riegle. Madam Chairman, I wonder, while the panel is coming, if I could just be recognized for a brief comment.


Senator Riegle. We are meeting up in the Banking Committee, as you well know, Madam Chairman, on housing issues, where I am the ranking member. Therefore, I have been detained up there and must go back. However, I just want to make one point for the record.

As you know, the Commerce Committee also has jurisdiction on the issue of advertising as it relates not only to cigarette advertising and warning labels in that regard, but other related kinds of issues. From strictly a jurisdictional point of view, many of us who serve on the Commerce Committee, as I do, would hope that when this committee finishes its work on any legislative product, that we would agree to have it considered on a joint basis by the Commerce Committee, so that we tie the two jurisdictions together and be in a position to have that kind of joint consideration.

I say that as one who personally has a concern about the health effects associated with smoking. I say it as a person who is a non-smoker. In any event, I think from a jurisdictional point of view, it is very important to recognize that the Commerce Committee does have some longstanding jurisdictional standing in this matter, and we ought to take some account of that. I raise it as a member of both committees.

I thank the chairperson.

Senator Hawkins. I am informed that the Parliamentarian ruled under the Preponderance ruling that it should be referred to this committee singly.

Senator Riegle. I understand that, but I still want to make the point that I think we would be well advised as a committee, when we finish our work, to consider asking the Commerce Committee as well to weigh what we have suggested, so that we not have a jurisdictional conflict at a later point on the floor. I think it is better that the committees harmonize, if they can. That is the point I would make.

I thank you for yielding to me.

Senator Hawkins. Thank you, Senator Riegle.

Our third panel today consists of distinguished physicians and a nationally known television personality who is an expert on children's education.

Our first witness, Dr. Virginia Ernst, is associate professor of epidemiology at the University of California, San Francisco.

Our second witness is Dr. Lawrence Longo, head of the division of perinatal biology and professor of physiology, obstetrics and gynecology at Loma Linda University in California. Dr. Longo will be
the spokesman for the American Academy of Pediatrics and the American College of Obstetricians and Gynecologists.

Our third speaker is Dr. Robert Sandhaus, the senior staff physician and associate professor of medicine at the University of Colorado Health Sciences Center in Denver. He is accompanied by Dr. Michael Shaubraum, president of the National Jewish Hospital in Denver. Dr. Sandhaus will be a spokesman for the National Jewish Hospital and Research Center-National Asthma Center in Denver, Colo.

Our last speaker, Mr. Robert Keeshan, professionally known to millions as Captain Kangaroo, is a highly successful television producer and renowned advocate for children's education.

We welcome you all here.

We will ask Dr. Ernster to begin.

STATEMENT OF DR. VIRGINIA ERNSTER, ASSOCIATE PROFESSOR OF EPIDEMIOLOGY, UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

Dr. Ernster. Thank you, Madam Chairwoman and members of the committee, for the opportunity to speak on behalf of S.772, the Comprehensive Smoking Prevention and Education Act. I hope you share my concern about the importance of this bill for the health of American women and children.

I am a cancer epidemiologist on the faculty of the school of medicine at the University of California, San Francisco. Through my work, I am familiar with the statistics that indicate, given current trends, that lung cancer will surpass breast cancer as the No. 1 cause of cancer death among American women by the mid-1980's. This is an epidemic that could have been prevented if it were not for cigarette smoking.

Cancers of the larynx, oral cavity, esophagus, bladder, kidney, and pancreas are also increased among smokers. About one-fifth of new cases of cancer and over a quarter of cancer deaths among American women are presently attributed to cigarette smoking. Other nonmalignant but frequently fatal conditions are more common in smokers, including emphysema and heart disease. Women smokers share these disease risks with men but they face additional unique risks related to oral contraceptive use and pregnancy.

Cigarette smoking acts in combination with oral contraceptive use to markedly increase a woman's risk of cardiovascular disease. Among women 35 years of age and older, oral contraceptive use is medically contraindicated in smokers based on their excessive mortality.

Cigarette smoking during pregnancy has been associated with increased risks of spontaneous abortion, premature labor, complications of pregnancy, retarded fetal growth, and perinatal mortality. According to a 1980 Roper poll, over half of women who smoke are unaware that smoking during pregnancy increases the risk of miscarriage and stillbirth. It is estimated that 30 percent of pregnant women are smokers. One-quarter to one-third quit smoking during pregnancy, but the fact that the majority do not must reflect the...
combined effects of ignorance and the difficulty that confirmed smokers face in attempting to quit.

There are additional risks to young children exposed involuntarily to tobacco smoke, including respiratory illnesses, such as bronchitis, pneumonia, and allergies, and possibly middle ear disease. We know, too, that children of parents who smoke are more likely to become smokers themselves than are children of nonsmoking parents.

Smoking prevalence data show that the average age of beginning to smoke has lowered considerably over time. Now the average girl who begins regular smoking is about 14 or 15 years of age. This raises a couple of important concerns. One relates to the longer cumulative exposure to cigarette smoking that will accrue over the lifetime from earlier initiation. The other has to do with the timing of initiation in relation to the key reproductive events. Most young women who smoke start to do so before making the decision to use oral contraceptives or to become pregnant, and it is obviously difficult for a woman to quit after she is “hooked.”

Sadly, the increase in medical knowledge of the health hazards of smoking for women has been paralleled by sharp increases in cigarette advertising directed at women in the United States. Willingly or not, American women encounter in their daily lives many sources of positive messages regarding cigarettes—attractive female models, purportedly smokers, on billboards, newspaper and magazine ads, on taxi, bus, and subway posters, on coupons for free packs. They may be offered cigarette sample giveaways on city streets or be exposed to more subtle forms of promotion such as fashion, sport, and other social events sponsored by cigarette companies. Most of the major American women’s magazines have become quite dependent on cigarette advertising during the past decade, with up to 15 percent of total advertising revenues in those publications coming from cigarettes. Unfortunately, the images of glamorous, healthy, and athletic women in the ads are completely at odds with what really happens to many women who smoke.

The tobacco industry publicly acknowledges that it is directing much of its contemporary cigarette advertising to the female market. In a front page article entitled, “Women Top Cig Target,” Advertising Age of September 28, 1981, quotes the president and chief executive officer of R. J. Reynolds, who described women’s market as “probably the largest opportunity for Reynolds.” The article cites industry sources who view the working woman, under stress, as the ideal candidate for their product. In January of this year, a long piece in the same publication was titled, “Marketers Clamour to Offer Lady a Cigarette.”

In light of the overwhelming evidence regarding the health hazards of cigarette smoking, it is of paramount importance that people who choose to smoke are fully informed of the risks they undertake and that they are continually reminded of those risks. The present imbalance between pro-cigarette advertising and antismoking educational campaigns is revealing: more than $1 billion was spent by the 6 leading U.S. tobacco companies to promote cigarettes in 1980, compared to less than $10 million in antismoking educational activities through the combined efforts of the American Cancer Society, the American Lung Association, and the
American Heart Association, and only $750,000 by the Federal Office on Smoking and Health for the same purpose. The money expended yearly for cigarette advertising alone—now estimated to be about $1.4 billion—exceeds the entire budget of the National Cancer Institute, and the costs that were associated with promoting a single new brand of cigarettes this year directed exclusively to women was far in excess of the annual research budget of the American Cancer Society. It is indeed ironic that the product that is the No. 1 cause of mortality in this country is also No. 1 in advertising.

The present warning on cigarette packages that reads, "Warning: the Surgeon General has determined that smoking is dangerous to your health" is abstract and does not mention any specific diseases caused by smoking; it is therefore unlikely to be taken as a concrete warning that individuals can apply to themselves. Moreover, the "sameness" of the warning has made it all but invisible. Therefore it is not surprising that recent national surveys show that 31 percent of cigarette smokers are unaware that smoking greatly increases one's risk of cancer. S. 772 proposes to inform those actually at risk by authorizing that the package warning labels refer to specific diseases and that they be varied so that smokers of a particular brand will read about different health hazards on different packages.

Because the warning labels would identify specific diseases such as lung cancer, emphysema, and heart disease, people might be more likely to personalize the risk.

The bill is of particular importance to the health of American women because one of the proposed labels deals with the special dangers cigarette smoking poses to women of reproductive age, that is, "Warning: The Surgeon General has determined that cigarette smoking by pregnant women may result in miscarriage, premature births, and low birthweight babies."

In the face of the bold and colorful positive imagery ubiquitously conveyed in cigarette advertising, the bill is a step to provide information to make the decision to smoke truly informed.

Another positive provision of the bill relates to the requirement for manufacturers to disclose the additives contained in cigarettes. Several hundred additives are used to enhance flavor, moisten, or slow the burn of cigarettes. Exactly which of these is contained in any given brand is presently not reported, but cigarette additives are thought to include cocoa, licorice, coumarin, glycerol, triethylene glycol, and catechol, among others. Based on laboratory tests, there is reason to believe that the named substances may play a role in carcinogenesis. Tar and nicotine levels in cigarettes have been reduced in response to health concerns, but the unknown compensating additives may have their own adverse health effects that will be impossible to investigate in the laboratory if those chemical components remain secret.

Rarely, if ever, have the data on any medical issue been so unequivalent, but revenue comparable to that invested by the cigarette industry is not available to health agencies to present a balanced view to the public. My 8-year-old daughter refuses to use cigarette ads for any school projects that require magazine cutouts. She ex-
They want to make you think that cigarettes will make you beautiful, but really they just want to make money. Those ads are dumb because cigarettes make you die." I wish that all children—and adults—were similarly aware.

The Comprehensive Smoking-Prevention and Education Act is a step in that direction. It is an idea whose time has long since come and one whose importance should be obvious to political leaders who are truly concerned with the health of the American public, women and men alike.

Thank you.

Senator Hawkins. Thank you, Dr. Ernster.

Dr. Longo.

STATEMENT OF LAWRENCE D. LONGO, M.D., HEAD OF THE DIVISION OF PERINATAL BIOLOGY, LOMA LINDA UNIVERSITY SCHOOL OF MEDICINE, LOMA LINDA, CALIF., REPRESENTING THE AMERICAN ACADEMY OF PEDIATRICS AND THE AMERICAN COLLEGE OF OBSTETRICIANS AND GYNECOLOGISTS

Dr. Longo. Madam Chairman, Senator East, I appreciate this opportunity to offer comments on behalf of the American College of Obstetricians and Gynecologists and the American Academy of Pediatrics on the harmful effects of cigarette smoking on the pregnant woman, her unborn fetus, and the newborn infant and child. Our organizations encourage the concept of rotational warning labels on cigarette packages which will improve public awareness of the health consequences of smoking during pregnancy. In my testimony I would like to review the short-term and long-term effects of smoking and will conclude with an estimate of the numbers of pregnant women and infants at risk who could benefit from this legislation.

I have published numerous papers on maternal smoking by pregnant women and coauthored the chapters on pregnancy and infant health in the 1979, 1980, 1981, Surgeon General's reports on smoking and health. Since the first report 25 years ago that the newborn infants of women who smoked during gestation were of significantly lower birthweight than the infants of comparable non-smokers, the adverse effects of maternal smoking on pregnancy have been extensively documented and acknowledged by all health care professionals. The original Surgeon General's report on smoking and health in 1964 contained only one brief paragraph and a few references on smoking and pregnancy.

In contrast, the 1979 report consisted of almost 100 pages and several hundred references documenting the considerable body of epidemiological, clinical, and laboratory evidence concerning the role of cigarette smoking in complications for the pregnant woman and her child.

The complications of pregnancy ascribed to cigarette smoking may be divided into those which affect the mother, the embryo and fetus, the placenta, and the newborn infant and child. The mother, fetus, and placenta constitute an integrated organic unit rather than separate systems or organs. Thus, although separation of these effects is convenient, it is somewhat arbitrary. Although we do not understand all of the mechanisms underlying the effects of
smoking on pregnancy outcome, the epidemiologic evidence is clear.

Women who smoke cigarettes show a greater incidence of placenta previa, that is, blocking of the opening of the uterus by the placenta; abruptio placentae or premature separation of the normally implanted placenta from the uterus; vaginal bleeding during pregnancy; and premature rupture of the membranes. These complications increase the risk of fetal and neonatal death and their occurrence appears to increase with the number of cigarettes smoked.

For instance, the incidence of placenta previa and abruptio placentae is about 25 percent greater for those mothers who smoke less than 1 pack of cigarettes per day while it is about 90 percent greater for those who smoke 1 or more packs of cigarettes per day.

We know that tobacco smoke contains several thousand compounds. For the pregnant woman and fetus, the most important of these appear to be nicotine, carbon monoxide, and the polycyclic aromatic hydrocarbons. It is known that nicotine rapidly crosses the placenta and acts as both a stimulant and depressant on the fetal nervous system. Nicotine is also found in the breast milk of those mothers who smoke.

Carbon monoxide, another component of tobacco smoke, decreases the capacity of blood to transport oxygen by competing with it for hemoglobin. Carbon monoxide binds with the hemoglobin in place of oxygen, therefore, depriving the developing fetal tissues of oxygen, which they critically need.

The requirement of disclosure of cigarette additives to the Secretary of the Department of Health and Human Services would aid researchers to further determine the effects of these substances on the developing fetus.

As you have heard from previous witnesses, the risk of spontaneous abortion is 30 to 70 percent higher among women who smoke than nonsmokers and again increases with the number of cigarettes smoked.

The rate of premature deliveries also increases significantly with the level of maternal smoking. About 13 percent of all preterm births can be attributed to this factor. Perhaps most importantly, for women who smoke more than 1 pack of cigarettes a day, perinatal mortality increases about 37 percent. Smoking less than 1 pack of cigarettes a day is still associated with a 24 percent increase in fetal mortality. Cigarette smoking has been shown to be the single most important identifiable contributor to perinatal mortality in our society.

Virtually all of the studies published, from many countries and ethnic groups, have been consistent in showing that maternal smoking has an adverse effect on the birthweight. These newborn infants weigh on the average 200 grams, or about a half a pound, less than the babies born to comparable women who do not smoke, and this birthweight is inversely related to the number of cigarettes smoked.

Several abnormalities of infancy and childhood occur more frequently among the offspring of mothers who smoke. These children experience a higher rate of morbidity and mortality up to 5 years of age. Smokers' children have more hospitalizations, more visits to the doctor, and more use of specialized medical services. More in-
fants of smoking parents are hospitalized for pneumonia and bronchitis. Sudden infant death syndrome occurs about 2½ times more frequently among the children of women who smoke.

Furthermore, long-term sequelae or consequences of maternal smoking during pregnancy are now becoming apparent. Several studies suggest that older children of mothers who smoke have slight but measurable deficits in physical growth, intellectual ability, emotional development, and behavior.

For instance, the British perinatal study showed that the physical growth of smokers' children remained less than that of non-smokers' offspring, at least until age 11. Associations have also been reported between maternal smoking and deficits in neurological and intellectual development of the child. These include minimal cerebral dysfunction and abnormal or borderline electroencephalograms, hyperkinesia, and abnormal infant behavior patterns. The potential seriousness of these effects requires increased research and public health attention and the attention of this committee.

The question arises whether smoking low tar and nicotine cigarettes improves these adverse outcomes. To date, the evidence indicates no difference in the health of the mothers or infants of women who smoke these cigarettes.

Amazingly, despite all that we know about the negative effects of smoking by pregnant women, presently one-third of all women and about two-thirds of teenagers continue to smoke during pregnancy. Thus, for the United States with about 3.5 million live births each year, about 1.2 million pregnant women are at risk as smokers. Of the 68,600 perinatal deaths per year in this country, it is estimated that a fifth, or 13,720 infant deaths, could be prevented by mothers not smoking.

Additionally, perinatal morbidity is dramatically increased by maternal smoking. Various forms of brain damage such as hyperkinesia, cerebral palsy, and other nervous and behavioral disorders which are the result of maternal smoking could be avoided if we are successful in educating mothers to the hazards of smoking during pregnancy.

In conclusion, cigarette smoking has been shown to have numerous adverse effects on the mother, fetus, newborn infant, and the child in later years. Smoking is now recognized to be the single most important known cause of perinatal mortality. Pregnant women as well as mothers of small children are well advised to cease smoking, not only for their own health and well-being, but for that of their children.

We support S. 772 and urge the Congress to enact this legislation which will increase public health awareness of the hazards of smoking. The provisions of the legislation—rotational warning labels for cigarette packages, additional research efforts, and a requirement for the disclosure of additives to the Secretary of the Department of HHS—are an important step in this process. We, as health professionals, are ready and willing to work with you.

Thank you.

[The prepared statement of Dr. Longo follows:]
STATEMENT
of the
AMERICAN COLLEGE OF OBSTETRICIANS AND GYNECOLOGISTS
and
AMERICAN ACADEMY OF PEDIATRICS

Presented by
Lawrence D. Longo, M.D., FACOG

on

S. 772 Smoking Prevention Health and
Education Act of 1983

before the
Committee on Labor and Human Resources
United States Senate

May 5, 1983
Mr. Chairman, members of the Committee, my name is Lawrence D. Longo, M.D. I am Head of the Division of Perinatal Biology and hold Professorships in physiology and obstetrics and gynecology at Loma Linda University School of Medicine. I have published numerous papers on maternal smoking, and co-authored the chapters on pregnancy and infant health in the 1979, 1980, and 1981 Surgeon General's report on "The Health Consequences of Smoking".

I appreciate this opportunity to offer comments on behalf of the American College of Obstetricians and Gynecologists and the American Academy of Pediatrics on the harmful effects of cigarette smoking on the pregnant woman, her unborn fetus, and the newborn infant and child. Our organizations encourage the concept of rotational warning labels on cigarette packages which will improve public awareness of the health consequences of smoking during pregnancy. In my testimony, I will review the short-term and long-term effects of smoking and will conclude with an estimate of the numbers of pregnant women and infants at risk who could benefit from this legislation.

Since the first report 25 years ago that the newborn infants of women who smoked during gestation were of significantly lower birth weight than the infants of comparable nonsmokers, the adverse effects of maternal smoking on pregnancy have been extensively documented and acknowledged by health care professionals. The original Surgeon General's report on smoking and health in 1964 contained only one brief paragraph and a few references on smoking and pregnancy. In contrast, the 1979 report consisted of almost 100 pages and several hundred references documenting the considerable body of epidemiological, clinical, and laboratory evidence concerning the role of cigarette smoking in complications for the
The complications of pregnancy ascribed to cigarette smoking may be divided into those which affect (1) the mother, (2) the embryo and fetus, (3) the placenta, and (4) the newborn infant and child. The mother, fetus, and placenta constitute an integrated organic unit rather than separate systems or organs. Thus, although separation of effects into these categories is convenient, it is also somewhat arbitrary. Although we do not understand all of the mechanisms underlying the effects of smoking on pregnancy outcome, the epidemiologic evidence is clear.

Women who smoke cigarettes show a greater incidence of placenta previa (placental blocking of the opening of the uterus), abruptio placentae (premature separation of the placenta from the uterus), vaginal bleeding during pregnancy, and premature rupture of the membranes. These complications increase the risk of fetal and neonatal death and their occurrence appears to increase with the number of cigarettes smoked. For instance, the incidence of placenta previa and abruptio placentae is about 25 percent greater for mothers who smoke less than one pack per day than that of nonsmoking women, and it is about 90 percent greater for those who smoke one or more packs of cigarettes per day.

Tobacco smoke contains more than 2,000 compounds including nicotine, carbon monoxide, oxides of nitrogen, ammonia, polycyclic aromatic hydrocarbons, hydrogen cyanide, and vinyl chloride. For the pregnant woman and fetus the most important of these appear to be nicotine, carbon monoxide, and the polycyclic aromatic hydrocarbons. Although the effects of nicotine on the fetus are not well established, it is known that nicotine rapidly crosses the placenta and in animal studies has been found to act as a stimulant on the fetal nervous system. Nicotine is also found in the...
breast milk of mothers who smoke. Carbon monoxide, another component of tobacco smoke, decreases the capacity of blood to transport oxygen by competing with it for hemoglobin. Carbon monoxide binds with the hemoglobin in place of oxygen therefore depriving the developing fetal tissues of oxygen. The polycyclic aromatic hydrocarbons, components of tobacco smoke produced by incomplete combustion, are potent carcinogens. Although they pass through the placenta, their effect on the fetus is unknown. Requiring disclosure of cigarette additives to the Secretary of DHHS would aid researchers to further determine the effects of these substances on the fetus.

The risk of spontaneous abortion is 30 to 70 percent higher among pregnant smokers than among nonsmokers and again increases with the number of cigarettes smoked. The rate of premature deliveries also increases significantly with the level of maternal smoking. About 13 percent of all preterm births can be attributed to this factor. Perhaps most importantly, for women who smoke more than one pack a day, perinatal mortality increases about 37 percent. Smoking less than one pack of cigarettes a day is still associated with a 24 percent increase in mortality. Cigarette smoking has been shown to be the single most important identifiable contributor to perinatal mortality in our society.

Virtually all of the more than 60 studies published, involving about a million births from many countries and ethnic groups, have been consistent in that maternal smoking has an adverse effect on birth weight. These newborn infants weigh on the average 200 grams less than babies born to comparable women who do not smoke, and the birth weight is inversely related to the number of cigarettes smoked. In an analysis of
data from Ontario, Canada, the number of newborns weighing less than 2,500 grams was 52 percent greater among women smoking less than one pack or more per day, compared with the pregnancies of non-smoking women. The relationship between smoking and low birth weight is independent of all other factors influencing birth weight such as race, parity, maternal size, socioeconomic status, and sex of the child.

Several abnormalities of infancy and childhood occur more frequently among the offspring of mothers who smoke. These children experience a higher rate of morbidity and mortality up to 5 years of age. Smokers' children have more hospitalizations, more visits to the doctor, and more use of specialized services. More infants of smoking parents are hospitalized for pneumonia and bronchitis. Sudden Infant Death Syndrome (SIDS) occurs two and one-half times more frequently among the children of women who smoke.

Furthermore, long-term sequelae or consequences of maternal smoking during pregnancy are becoming apparent. Several studies suggest that older children of mothers who smoke have slight but measurable deficits in physical growth, intellectual ability, emotional development, and behavior. For instance, the British Perinatal Study showed that the physical growth of smokers' children remained less than that of non-smokers' offspring, at least until age eleven. Associations have been reported between maternal smoking and deficits in neurological and intellectual development of the child. These include minimal cerebral dysfunction and abnormal or borderline electroencephalograms, hyperkinesia, and abnormal infant behavior patterns. The potential seriousness of these effects requires increased research and public health attention.
The question arises whether smoking "low tar and nicotine" cigarettes improves these adverse outcomes. The evidence to date indicates no difference to the health of mother and infants.29

Amazingly, despite all that we know about the negative effects of smoking by pregnant women, presently one-third of all women and two-thirds of teenagers continue to smoke during pregnancy.30 Thus, for the United States with about 3.5 million live births each year (1979 figure) about 1.2 million pregnant women are not at risk as smokers. Of the 68,600 perinatal deaths per year in this country (19.6 perinatal deaths/1000 live births, 1979 figure), it is estimated that a fifth, or 13,720 infant deaths, could be prevented by mothers not smoking. Additionally, perinatal morbidity is dramatically increased by maternal smoking. Various forms of brain damage such as hyperkinesis, cerebral palsy, and behavioral disorders which are the result of maternal smoking, could be avoided if we are successful in educating mothers to the hazards of smoking during pregnancy.

In conclusion, cigarette smoking has been shown to have numerous adverse effects on the mother, fetus, newborn infant, and the child in later years. Smoking is now recognized to be the single most important known cause of perinatal mortality. Pregnant women as well as mothers of small children are well advised to cease smoking not only for their own health and well-being, but for that of their children.

We support S. 772, and urge the Congress to enact this legislation which will increase public health awareness of the hazards of smoking. The provisions of the legislation, rotational warning labels for cigarette packages, additional research efforts, and a requirement for the disclosure of additives to the Secretary of DHHS, are an important step in this process. We, as health professionals, are ready and willing to work with you.
REFERENCES


12. Schlede E., Merker H.J.: Effect of benzo(a)pyrene treatment on the benzo(a)pyrene hydroxylase activity in maternal liver plasma, and fetus of the rat during day 13 to day 18 of gestation. Naunyn-Schmiedebergs Archives of Pharmacology 272:89-100, 1972.


16. Ibid.


Senator HAWKINS. Thank you.

Dr. Sandhaus.

STATEMENT OF ROBERT A. SANDHAUS, M.D., PH. D., SENIOR STAFF PHYSICIAN, NATIONAL JEWISH HOSPITAL AND RESEARCH CENTER/NATIONAL ASTHMA CENTER, DENVER, COLO.

Dr. SANDHAUS. Madam Chairperson, my name is Robert A. Sandhaus, M.D., Ph. D. I am a senior staff physician at the National Jewish Hospital and Research Center/National Asthma Center in Denver. It is on behalf of this institution that I present this testimony today. I am also an assistant professor, of medicine at the University of Colorado School of Medicine. I would like to thank the committee for my promotion to associate professor, but I do not think they will buy it back home.

I am an investigator with particular interests in the causes and biochemistry of pulmonary emphysema.

I appreciate your having afforded me the opportunity to present my medical and scientific views which I believe are pertinent to S 772, a bill which I and the National Jewish Hospital/National Asthma Center strongly support.

NJH/NAC is an internationally recognized medical center for the study and treatment of respiratory diseases and immunologic disorders. The hospital is unique in its exhaustive, multidisciplinary approaches to these problems. Clinical and research emphasis is placed on emphysema, chronic bronchitis, asthma and other lung diseases, exclusive of lung cancer, and various immunological disorders.

We are keenly interested in the devastating effects of cigarette smoking on the lungs. This panel is concerned with the issue of increased smoking among women and children, a situation that concerns and distresses me greatly, as does smoking by men. The important fact here is that smoking causes chronic bronchitis and emphysema, diseases for which there are, as yet, no cures. This bill, in part, seeks to educate women, children, and men that smoking and

ERIC
Emphysema are directly linked. I applaud it, and all efforts which will prevent children and adults from taking up the habit or will encourage those who smoke to quit.

What I would like to do is present to this committee the current state of the art, if you will, regarding biomedical evidence linking cigarette smoke inhalation to the evolution of pulmonary emphysema. You have heard the epidemiologic arguments linking cigarette smoking to emphysema. These were the first and strongest arguments—strong enough to cause the cigarette labeling laws that are currently in existence. Now the cause-and-effect relationship can be based on more than epidemiologic data. A brief history will help.

In the mid-1960’s, scientists demonstrated that it was possible to create an animal model of emphysema by exposing lungs of laboratory animals to an enzyme called elastase. Elastases are a class of enzymes that have the ability to degrade or destroy the protein called elastin. Now elastin, as the name implies, is the protein that imparts the elastic properties to the lung, that is, its ability to expand and contract during breathing. These early studies showed that animal emphysema can be caused by elastase.

At the same time this work was progressing, it was found that there was a rare inherited form of emphysema not related to cigarette smoking necessarily, and that this hereditary emphysema was due to a lack of an enzyme inhibitor in the blood called alpha-1 antitrypsin.

These two areas of investigation—experimental emphysema in animals and human inherited emphysema—began to meet with the discovery of a potent elastase enzyme in human white blood cells, and the discovery that the action of this enzyme is blocked by alpha-1 antitrypsin, the protein which is deficient in the blood of individuals with hereditary emphysema.

Now we have some very useful information. On the one hand, we have elastase enzymes in white blood cells of humans which can destroy lung tissues. At the same time, there is another protein in the blood called alpha-1 antitrypsin which blocks the destruction of the tissue by the elastase. In other words, there is a stalemate between the opposing forces in the lungs, and normally no damage takes place.

How does this relate to smoking? The crucial investigations from several laboratories have shown that cigarette smoke is capable of destroying the ability of alpha-1 antitrypsin to block the action of white blood cell elastase. In other words, cigarette smoke blocks the protective protein from doing its job, and the white blood cell elastase then is free to destroy lung tissues, and results in emphysema. The normal protective effect of alpha-1 antitrypsin is blocked by oxidizing agents in cigarette smoke.

Thus, we believe that virtually all of the emphysema we see is due to a deficiency of alpha-1 antitrypsin action. In rare instances, this deficiency of action is due to a genetic defect which results in a lack of the protein, alpha-1 antitrypsin. In the vast majority of emphysema cases, however, it is a functional deficiency—alpha-1 antitrypsin has been inactivated by cigarette smoke oxidants.

In recent research we have shown that white blood cells are constantly migrating in and out of lung tissue and, because of their
content of elastase, are capable of degrading lung elastin in small quantities during this migration. However, when alpha-1 antitrypsin is inactivated by cigarette smoke, this normal migration then causes significantly greater destruction of lung elastin.

In summary, the body’s own white blood cells contain an enzyme capable of causing the lung deterioration known as emphysema, an irreversible, relentlessly fatal lung disease. The lungs’ defense relies on an enzyme inhibitor called alpha-1 antitrypsin to balance the action of the destructive enzyme. When this balance is upset by oxidants in cigarette smoke, the white blood cell enzyme can chew away at the lung, unbridled, and cause disease.

Thank you very much.

The CHAIRMAN. Our last speaker, Mr. Robert Keeshan, affectionately known to millions as Captain Kangaroo, is a highly successful television producer and is a reknown advocate of children’s education.

We are very happy to have you here, Mr. Keeshan.

STATEMENT OF ROBERT KEESHAN, EXECUTIVE PRODUCER OF ROBERT KEESHAN ASSOCIATES [CAPTAIN KANGAROO]

KEESHAN. Thank you very much, Mr. Chairman, Senator Hawkins, Senator East. I am delighted to be with you today and have the opportunity that you present to me to express my views on what I feel is a very serious health problem threatening the well-being of the Nation’s greatest resource, its young people.

Most of you are aware that I have spent 35 years on television and radio and elsewhere educating young Americans, helping them to develop intellectually, physically, and emotionally, so that they may become healthy, well-educated, stable citizens in American society.

The human learning process begins at birth and of our learning in our early years is a result of observation. We speak with the same accent as our parents, display their mannerisms because they are, in our early years, our principal models. As we grow older, observation remains a principal source of education. By preteen and early teen years, many of the most influential models for young people are their peers, and it is at this time that that terrible demon, peer pressure, whips young people into conformity.

It is very difficult to be an independent, nonconforming teenager. It is a time when we are desperately attempting an escape from childhood. It is at this time that our teens are being given the message that cigarette smoking is grown up, mature, it is “being cool,” “liberated,” “macho,” the “in thing to do.” The magazine ads tell us that “Camel is where a man belongs,” if you “want to be successful, you should smoke Vantage,” and “Kool is the only way to play it.”

It is not easy to give up cigarette smoking once you start, but it is not easy to start, either. It is not a natural thing for us humans to do. In fact, it is quite unnatural for a living thing to breathe cigarette smoke. Try blowing smoke in the face of your dog or cat and note the reaction. But peer pressure in preteen and teens overcomes the offensive and unnatural aspects of cigarette smoking, and they are hooked.
I remember clearly as a 17-year-old U.S. marine being told by my buddies that if I did not smoke cigarettes I was not a real marine. I wanted to be just like John Wayne, and I started smoking cigarettes. It took me 25 years to grow up and break the habit, and I don't think I am any less a man than I was at Camp Lejeune, N.C.

Cigarette companies sponsor special events which appeal to old and young alike, sporting events, musical and art events featuring some of the Nation's greatest heroes and superstars. It is understandable why young people come to accept the sponsor's product and fail to appreciate the dangers associated with use of that product.

We must not be complacent. Cigarette smoking by children and adolescents is on the rise. It is estimated that each day, including this day, 5,000 children light up for the first time. Some of these children are only 7 or 8 years of age, members of our hurried generation confusing smoking with maturity, modeling the behavior of the Marlboro man, the Camel man, the Virginia Slims woman.

Dr. Mortimer Lipsett, who was here earlier today, director of the National Institute of Child Health and Human Development, recently told the House Subcommittee on Health and the Environment that approximately 3 million—3 million—American adolescents are now regular smokers. Teen girls are smoking more than teen boys. Fifteen percent of teenage girls smoke cigarettes. Cigarette brands aimed at women are many, "More," the beige, the slender, the "More You" cigarette, "Satin," for women who deserve the satin moment, "Now, I assume for the liberated women, and "Eve." "Eve" is not designed for Adam, it is the cigarette that tastes as good as it looks.

Smoking is not just a habit, it is an addiction, and young people must be told this. It has now been established that it is, in fact, the most widespread example of drug dependence in our Nation.

I commend the sponsors of this legislation for including a warning label that reads, "Teenagers. Smoking is addictive, never starting means never having to try to quit."

We all know the pains suffered by the long-time adult smoker who decides to quit the habit and of the pains suffered by the smoker who contracts cancer, emphysema, or heart disease. Would it not be better to apply an ounce of prevention to arrest the development of America's most preventable cause of death?

This bill is attractive because it calls for the organization and the marshaling of forces by the Federal Government and the private sector to work together to educate all Americans as to the dangers associated with cigarette smoking. We must reach young Americans with the message. Cigarette smoking, feeling good and living long do not go together.

On the "Captain Kangaroo" program we have recently initiated a campaign against smoking, which we call, "Smoking YUCK!" and a press kit is attached for your information. The campaign is designed with the idea that our very young must begin developing attitudes about smoking at a very early age.

Yes, let us help those Americans who are victims of the habit to give it up, but let us turn our greater efforts to our children who have the most to gain and the longest to live. If we can, indeed, reach young men and women successfully through comprehensive
efforts, then I think we can all say with conviction, "You have come a long way, Baby!"

The CHAIRMAN. Thank you, Mr. Keeshan. I appreciate that.

I notice that they have come up with a little badge here that can be worn that has your Captain Kangaroo antismoking campaign—"Smoking YUCK!" that is. I got a note from the press corps that they would all like to have one of these buttons. I do not know what they want to use it for, but I think that is pretty good.

Did you bring some extra ones with you?

Mr. KEESHAN. We shall fulfill that request, yes.

The CHAIRMAN. OK. That is great.

Let me turn to Dr. Ernster and just ask a couple questions.

Knowing what we know today and realizing that deaths from several diseases can be prevented, could you comment on the current evidence that quitting smoking can reduce an individual's risk of disease even if he or she has smoked for several years?

Dr. ERNST. There are a number of studies that demonstrate that people who quit smoking, as time goes on, have lower and lower relative risks of lung cancer compared to people who continue to smoke, and that is true for other diseases as well.

The CHAIRMAN. Dr. Ernster, it has been said that nonsmokers can be affected by the smoking of others. I think we have had it referred to today as "passive smoking." What evidence is there that household tobacco smoke may affect the health of children and other nonsmoking persons?

Dr. ERNST. Some of that has been mentioned this morning. I would emphasize that people with preexisting heart conditions and allergies are particularly susceptible. That obviously includes many people in the population. Young children seem to be more prone to a variety of respiratory ailments, such as pneumonia and bronchitis and their allergies can be triggered. There are more bed days lost to school among children in the households of smoking parents than nonsmoking parents due to acute respiratory conditions.

The CHAIRMAN. Thank you.

Let me turn to Dr. Longo and I will try to hurry because we have a vote that just went off and I want to allow other Senators an opportunity to ask some questions.

Dr. LONGO. Senator Hatch, I believe it is very important. I want to establish that point. I believe it is important because the present legislation to disclose the level of nicotine is not enough. Several years ago I served on a committee for the National Academy of Sciences in which we looked at carbon monoxide toxicity. We identified pregnant women and children as one of the most vulnerable groups to carbon monoxide toxicity.

Now carbon monoxide is particularly invidious because it combines with hemoglobin very avidly, replacing the oxygen that should normally be there and, therefore, depriving the developing organism and the sensitive brain and other nerve tissue of oxygen. I think it is imperative that this be disclosed.
It has been shown that in even these so-called low tar and low nicotine cigarettes they are not low in carbon monoxide.

The CHAIRMAN. Dr. Longo, it has been shown that women who smoke have smaller babies with birth rates inversely related to the number of cigarettes smoked. In your experience could this be reversed if women smokers stopped smoking during their pregnancies?

Dr. LONGO. Yes, Senator. In fact, this has been shown in several studies, that if women stopped before the third trimester of pregnancy, apparently these effects can be reversed.

The CHAIRMAN. Thank you.

Dr. Sandhaus, it has been said that smoking destroys the lung tissue. At what point does the destruction of lung tissue begin after a person smokes a cigarette? I might add, in other words, do people have to smoke often for the destruction of lung tissue to occur?

Dr. SANDHAUS. Presumably, the lung tissue destruction takes place almost instantaneously with the smoking of a cigarette.

The CHAIRMAN. A single cigarette?

Dr. SANDHAUS. A single cigarette can be shown to inactivate all of the available alpha-1 antitrypsin present in the lung. The advantage that humans have is that they have a great pulmonary reserve and it takes a lot of destruction before symptoms occur.

The unfortunate event is that most people do not smoke a single cigarette. They smoke multiple cigarettes multiple times a day throughout their lives.

The CHAIRMAN. There have been numerous studies which show that some forms of emphysema are due to hereditary genetic defects. How much emphysema in this country is due to heredity and how much would you attribute to cigarette smoking?

Dr. SANDHAUS. Well, this hereditary form of emphysema that I spoke of probably accounts for, at most, about 3 percent of all the emphysema that is seen in the clinical centers around the country. Even those estimates are from Scandinavian studies where there appears to be a higher incidence of the hereditary form of emphysema than we have in the United States.

The CHAIRMAN. I see.

Let me go to you, Bob, and ask you this question. If you could send one message to the young people in this country or throughout the world regarding the hazards of smoking, what would that message be?

Mr. KEESHAN. Well, it was the message, Senator, that you don't start. The problem with young people is you cannot send one message; it takes more than one to get through to them.

I was interested before in the discussion about the multiple labels on cigarette packages. What we are really attempting to do is to repeat the message, the single message, and then repeat the multiple message. Young people are in an educational environment at all times. Many people want to know what is the difference between education and entertainment, for example. For young people—very young children, particularly—I draw no line. They are being educated by everything they see in their environment every day. Therefore, it is not simply enough to say to a child, for example, in something more obvious, "Don't cross the street. You
might become injured.” That is something that has to be a redundant message, constantly an educational process.

I think smoking is no different, except perhaps that peer pressure may be a factor with cigarette smoking where it might not be with traffic safety and other safety messages.

The CHAIRMAN. Thank you so much.

We will turn to Senator Hawkins at this point.

Senator HAWKINS. Dr. Ernster, I was interested in your statistics that said that 50 percent of the women were unaware that smoking would harm an unborn child. How do you relate that to the figures that Senator East had, that 90 percent of the people are aware of the warning label on cigarettes?

Dr. ERNST. I think that there is an important distinction. People have a general awareness, just as the current warning label is couched in general terms, that “smoking ain’t good.” However, many people have difficulty identifying specific health effects that they might, in turn, apply to themselves.

It was interesting to me yesterday that my cab driver asked if he could smoke, and I said that I preferred that he didn’t and began to discuss some of the many health consequences of smoking. At the end he said, “I’m going to quit tomorrow,” adding, “I’ve known that it was bad all along, but I really didn’t know the exact reasons for that.” I think that that anecdote can be generalized.

In the case of pregnant women, people seem to be aware that it is not good to smoke during pregnancy but cannot really cite the specific outcomes. If they think it might just result in a little bit smaller baby but not know why that is a serious problem or not be aware of the actual serious risks of death, whether through an enhanced risk of spontaneous abortion or miscarriage or stillbirth or even death shortly after birth—if they cannot really specify those outcomes, it is going to be very hard for them to be motivated to quit.

Senator HAWKINS. Therefore, you would support the specific multilabeling?

Dr. ERNST. Absolutely. I think that if we had only one label, it would have to be a very long label, indeed, because it would have to cite so many specific health outcomes. With the proposed rotational label, people will read a short and readable label when they pick up a cigarette package and a different one at different points in time, and, hopefully, they will come to appreciate the specific risks involved.

Senator HAWKINS. I am reminded of the story that a major accident is something that happens to me, and a minor accident is something that happens to you. Maybe this is what you are saying, that it has to be targeted to the specific smoker—

Dr. ERNST. I agree.

Senator HAWKINS (continuing). Woman, teenager, children, etcetera.

I always call you “Captain Kangaroo.” I grew up with you. My children and I grew up with you. I wondered where our grandchildren were getting this “yuck” stuff, and now I know.

I appreciate all of you on this panel. You have really been of great value to us.
Since you do understand advertising probably more than others, what values can you assign to that of role models such as the sitcom 30-minute shows that everybody has to watch in the evening, where they, without advertising, smoking—the mother, father, whoever, the glamorous role that is assigned to it silently, such as the soap operas all afternoon involving alcohol and tobacco?

Mr. Keeshan. I think that the programs that reach a family audience where there are likely to be juveniles involved, there is, I think, a serious effort on the part particularly of the crafts—writers, producers, and directors—not to use alcohol, drugs, cigarettes, and the like unless there is a clear reason for the character to use them. That was not always true. Up until a few short years ago, the hero would say to the heroine, "Goodness, that was a terrible experience. Here, have a cigarette?" or he would say, "Jennie, you don't look well. Come on, have a drink." Of course, all of these behaviors were being modeled for our young people at a very early age.

However, it certainly is true that that does still exist in programs that are designed for adults, like soap operas and so on, which are watched by an enormous juvenile audience, even though they are not designed for them. I do not suggest that there is any way that we can prohibit the use of cigarettes and alcohol where it is dramatically called for in scripts, but I think unions, and particularly the craft unions, want to not harbor those practices where they might affect young people.

I think in advertising and in other modeling and in the home this behavior is being modeled for young people all the time. We somehow or other, through an educational process, have to counteract this modeling process.

Senator Hawkins. Therefore, you would say the amount of spent—I believe Dr. Ernst said a billion dollars is spent by the industry promoting cigarette smoking, and we spend $100 million in counter, educational programs, the Government trying to counteract that—you would say that is out of kilter?

Mr. Keeshan. I think that it is somewhat out of balance. There is no question that the see-saw is way out of balance from what it should be, if we are to be effective in reaching young people with that message.

Senator Hawkins. You support the multilabel, I gather from your testimony?

Mr. Keeshan. Oh, I certainly do. I think it is a very, very important aspect of this legislation.

Senator Hawkins. Thank you.

The Chairman. Let us thank you for coming.

At this time we will recess so that we can go and vote. We will finish with the last panel of voluntary agencies as soon as we return. We will recess until that time.

[Recess taken.]

The Chairman. Finally, our last panel also consists of three distinguished professionals representing three of the largest and most effective voluntary health organizations.
Our first speaker will be Dr. William Cahan, who is an attending surgeon at Memorial Sloan-Kettering Cancer Center in New York and will be representing the American Cancer Society.

Our second speaker will be Dr. John Oaths. Dr. Oates is a professor of medicine and pharmacology at Vanderbilt University and chairman of the smoking subcommittee of the American Heart Association. He is appearing on behalf of the American Heart Association today.

Our last speaker will be Dr. Edwin Fisher. He is an associate professor of psychology at Washington University in St Louis. He is a board member of the American Lung Association on whose behalf he is appearing today.

We are going to turn first to you, Dr. Cahan, and we will go from there.

STATEMENT OF WILLIAM CAHAN, M.D., PROFESSOR OF SURGERY, CORNELL UNIVERSITY MEDICAL COLLEGE, ATTENDING SURGEON, THORACIC SERVICE, MEMORIAL HOSPITAL, THE MEMORIAL SLOAN-KETTERING CANCER CENTER, NEW YORK CITY, N.Y., REPRESENTING THE AMERICAN CANCER SOCIETY

Dr. CAHAN. Thank you, Senator.

I am not only here as a representative of the American Cancer Society, but also for the Coalition for Smoking and Health—that rare combination of the heart, lung, and cancer problems that is working for specific legislation.

The CHAIRMAN. If I could just interrupt you for a second, I would like to recognize the efforts of the Coalition for Smoking and Health which you have just mentioned, representing of course the American Cancer Society, the American Heart Association, and the American Lung Association.

Dr. CAHAN. Right.

The CHAIRMAN. We really appreciate your representation here today.

Dr. CAHAN. This is the third time I have testified on this type of bill. The first two times were in the House of Representatives, Congressman Waxman's subcommittee. Each time I come down here I just think in advance, of course, of what to tell you; what to really express to you about the subject of cancer.

I have seen the enemy. I see it every day of my life. I have seen it for the 41 years I have been at Memorial Sloan-Kettering Cancer Center in New York. It is quite awful and awesome, and I try, as best I am able with limited vocal reserves, to give you an impression of the awfulness of it.

Need I tell you it has probably touched people perhaps in your family, certainly your friends. When it does, there is that moment, of course, when the moment of catastrophe, the moment of sense of awe is caved in, "Where do I go from here," and so forth and so on.

When you see that and you see it in a situation as we have with lung cancer, in which I happen to be very much interested and I am a specialist in, and you add to that the concept that it is a preventable disease for a majority of these people, it really gives one a sense of frustration. It gives one a sense, if you will, of outrage to a
certain extent that we should even think of countenancing something that should go on and do these things.

It is particularly—of course, the evidence, I don't have to tell you, is overwhelming. If you talk about the weight of the evidence, we were even going to try to get from the Library of Congress all of the bibliography on the subject of tobacco and disease. Literally, it would have taken a van to have carted it over here. Therefore, we decided against that concept.

In addition, we were going to also collect, in all honesty, the "pro," if you will, tobacco, the prosmoking literature, which of course is relatively miniscule and rather confined in its journal expressions, largely to the Journal of the tobacco industry.

There is no question about the relationship in any of our minds at the Memorial Cancer Center. We think it is unquestioned that this is a cause. It comes into our life daily. In our history-taking of patients we ask, "When did you smoke," and they say when they do. "How long?" "How many packs a day." This is a litany that we repeat over and over again with these patients.

It, therefore, comes almost to a sense of disbelief when recently I read some of the testimony of a congressional subcommittee in which a man of impeccable credentials still raises certain questions about the cause and effect of tobacco and cancer, particularly lung cancer. The man in question is Professor Langston of Chicago.

There are three or four subjects that he raises which are old, old, shall we say, questions about the subject. I would like to take them up because it is possible that his testimony may come across into your committee.

First of all, he asks these questions, which are rather logical. There is no question there is a certain logic to them.

"Why does lung cancer appear only rarely in both lungs if, after all, both lungs are subjected to cigarette smoke?"

First of all, let me just begin by saying there are large areas of ignorance of our understanding of cancer in general, but it is not at all unique in our bodies to have one of paired organs take on the cancer, if you will. We rarely see simultaneous cancers of the kidneys. We rarely see simultaneous breast cancers. We rarely see simultaneous ovarian cancers, and we rarely see simultaneous lung cancers but we do see them. I think this is one of the interesting questions yet to be resolved, but it is not an unusual effect and it does not, therefore, count against smoking in that regard.

"Why doesn't cancer develop in the trachea?" The trachea, as you well know, is the windpipe. After all, the same smoke going down this organ goes to both lungs. With the traffic in nicotine, coal tars, and so forth, why doesn't it pick up and do the same thing?

Again, it is mystery to a large extent, but the trachea is a vertical organ. It is the most sensitive part of the bronchial tree, and it is also a place in which the smoke passes readily both in and out rapidly. It is in the bronchiae, the branches on either side, where it puddles, and in so doing goes into solution in the mucous lining and there in solution becomes in more chronic contact, if you will, with the lining of the bronchus. Therefore, it has a better chance of causing all changes.
"Why hasn’t the rate of cancer of the larynx increased?" That is another question that has been asked. After all, the smoke goes through the larynx, into the lung. Why hasn’t the larynx as well? Well, actually there is an increase in the morbidity of cancer of the larynx. If you look at the mortality statistics, I am glad to say they are rather static and perhaps falling off, because we are getting it earlier and can cure it more. However, in the end there is a definite increase in larynx cancer, for example, in women, where one would expect it would occur concurrent with the rise in cancer of the lung.

There is also, of course, organ specificity for this. You might say that it goes through the mouth, then why aren’t there more “this’s” and more “that’s”? The fact remains that there are certain target areas that seem to be more susceptible than other target areas. We do not know all the reasons for that.

The question has also been raised as to whether or not lung cancer keeps changing and there is a natural decline in it, just as there has been with stomach cancer. However, we do not think there is anything natural about it. We think that there are, indeed, waves when cigarette smoking was at its peak, during World War I, World War II, and we see the expected peak and decline after those incredible periods. At this time, however, we can hardly talk about a natural decline in terms of the women, as you have heard today.

In the past 30 years there has been a 1,0010-percent increase in the instance of lung cancer in women. You have heard it is about to pass breast cancer as the leading cause of cancer in women. In the last 20 years there has been a 300- or 400-percent increase in the instance of lung cancer.

Our practice at Memorial Hospital used to be 8-to-1 10 years ago male to female. It is now even within a 10-year period, and we know well that it is going to pass that very shortly and be women to men in larger and larger proportions.

What occurred to me, of course, in dealing with this as a lifetime project was that habits are, indeed, like—as somebody once said, they begin as slender threads and wind up as cable. Never was this more true than it is at the present time.

As Bob Keeshan said, there is nothing like stopping them before they start, impressing them with that.

Can a label be of effect? This is, of course, what we are considering here today.

In thinking about this, in thinking of a question that Congressman Billey asked me, he said, "How can you be sure a label can be effective?" It took me a little while to try to document such a thing. It occurred to me at one time that, “Ah,”—and perhaps there are enough gray beards on the panel and in the senatorial group that you are to remember a thing called tincture of iodine. As I said that phrase, can you see the label on a tincture of iodine bottle? I think you will remember it as being skull and crossbones and the word, "Poison.” In case you want to be reminded of that, I have here a bottle of tincture of iodine. That will be quite reminiscent to you, the point being that with a label that is strong enough, it is a remembered thing.
Each one talks about the possibility that labels might be of some commercial detriment, if you will, or might stop people from taking or buying a product. I have the current label right on the front of the box of St. Joseph's aspirin for children saying, “Warning. Keep this and all medicines out of children's reach.” People are labeling on the front of these various packages.

Yesterday, believe me, with my coat collar turned up and dark glasses and a slouch hat, I slinked into a tobacco shop to buy a couple packs of cigarettes. I bought two just to see what the label looks like. It has been some time since I have looked at it.

I will offer this in testimony because I think those of you who wear glasses, I would defy you to read these labels at this particular time at any distance, particularly as they happen to be where they are located, which is not exactly a prominent position. I would even call them closet labels, if you want to use that label. They are off here. I will submit these to you.

Here is a newer cigarette, and you can barely discern it against the background. There it is. It is conforming to the law, but it certainly hasn't got any prominence on that package.

What is even more important is that you are talking about informing the citizens about the hazards. The phrase that is used here is not a specific phrase. It could say, “The Surgeon General has determined that jogging is bad for your health,” or that shoveling snow on a weekend could be bad for your health.

What I think we really need here, in light of all of this, is a specificity of a message, just as that which has been discussed often today. People should be told of specific problems that could arise. In so doing, you will really bring it across to them. I think that is terribly important.

Now, speaking of that message, I took the liberty of bringing two X-rays with me of two patients. The first patient was a 59-year-old woman who had been begged and pleaded with by her family, her physicians, her children to stop smoking her two packs a day since her younger days and could not succeed until one day she coughed up a little blood. Her physician took a chest X-ray, which is here, and I think, if I may point out to you, that this is shadow on her right side. This lung, the left side, is OK. This, of course, proved to be cancer. The day she saw this she found herself capable of stopping. By this time, it was of course too late and she went on and died, this mother, this wife, this grandmother, of a preventable disease.

The other X-ray is of interest. It occurs also in a woman in her late fifties from West Virginia who noticed that she had shortness of breath. A chest X-ray was done. This shadow was shown in her chest. We operated on it. It was the day before I testified this past March before the Waxman committee. I went to see her the morning after the operation to tell her that all was well and that I would be covered in my absence in Washington.

She said, “What are you going to Washington for?” I said, “I'm going there to testify,” and so forth. She said, “You know, that is quite something. Do you know who my father was?” I said no. She said, “He was the leading distributor of tobacco and tobacco products in America.” I said, “Oh, really?”
She said, "Yes. You know what happened to him, don't you?" I said no. She said, "He had lung cancer and he died of it."

This woman not only had lung cancer but she had emphysema, so that in our operating on her we were limited in what we could do for her because she had a coexistence of two diseases. We could only take a little pie-shaped piece out.

Needless to say, she has gone back home and she no longer smokes, but she still has the residual emphysema, and we cannot possibly operate on her lung cancer again. This is sort of a sin.

I will say that the other phase of smoking that perhaps does not come to you but comes to us as surgeons when we open chests to see if we can get out the culprit is a picture that I am about to show of two types of lungs, both autopsy specimens, one of which is in a nonsmoker, which. I will have here in a second, and the other is in a very severe case of smoking.

I think the first picture is of a relatively normal lung. Although you see it rather relatively pink and gray, there are little dark areas scattered though the surface, which are the collection of granules of carbon, dust, and so forth, that occur in a normal urban life. We can expect those. That is not any great harm to the lung itself.

In distinction from that, I want to show you the lung of a patient who was a heavy smoker. This, I think, will show a difference to you.

Here we see the deposit of tar of a very heavy smoker, three packs a day, in addition to which there are big, large blisters, so-called bulbous emphysema, which are evidences also of the fibrosis, the trapping that you have heard about in emphysema here. We see this frequently in heavy smokers.

Somebody has said that these two pictures should be in every classroom in America as somewhat of a deterrent to the young from smoking.

The CHAIRMAN. Maybe we can help you to get them. I think they will be on every television set in America by tonight.

That is hard to believe.

Dr. CAHAN. One more word in talking of labels, if I may. We go to a great deal of extent to put the letter "R" on motion pictures to decide that this is restricted from children, or "PG" for parental guidance. It seems to me if we do that, that there is a reason to do this labeling that I think has some importance in things that are as lethal as cigarettes have been.

Cancer is not a sentimental disease. It has no respect for beauty, wealth, or position. A beautiful woman called Mrs. William Paley smoked herself into death. She had all three.

Ed Murrow, the great announcer, he smoked himself into a cancer lung. More close to home are those Senators who died of lung cancer, all of whom smoked: The Vice President's father, former Senator Prescott Bush, Richard Neuberger; Senator Richard Russell and his brother, Judge Robert Russell; Senator Case; Senator Everett Dirksen, after whom this building was named. All smoked heavily, all died of lung cancer. They are but a few. I think you should know about that, too.
I think that time is short and the hour is late. Thirty-three million Americans will have their lives shortened by some cigarette-connected disease. Whom are we coddling in this? Whom are we trying to prevent from having trouble? The American citizens or a multibillion-dollar organization whose sole object is its perpetuation and its survival?

I do not think there is a time any longer for either tip-toeing about this problem. We must come directly on it. For those of us who see these tragedies daily, we are terribly, terribly anxious to move fast and find there is an urgency factor.

Indeed, cancer is not a sheik disease by any stretch of the imagination. It is an awful one. For those who see it daily, it is something that one must do something about.

It takes courage to live with cancer and it takes courage to fight it. I think that the citizens look to you for this particular kind of action.

In the time it has taken me to testify, 10 children have lit up for the first time in America and 4 American citizens have died of a smoking-related disease.

The CHAIRMAN. Thank you, Doctor.

[The prepared statement of Dr. Cahan follows:]
TESTIMONY
On Behalf of
THE AMERICAN CANCER SOCIETY
Before
The Committee on Labor & Human Resources
United States Senate

WILLIAM G. CARAN, M.D.
Professor of Surgery, Cornell University Medical College; Attending Surgeon, Thoracic Service, Memorial Hospital, The Memorial Sloan-Kettering Cancer Center, New York City
Member, American Cancer Society
Ad Hoc Committee on Tobacco Habituation

May 5, 1983

For further information, contact:
Alan C. Davis
The American Cancer Society
777 Third Avenue
New York, New York 10017
(212) 371-2900
MR. CHAIRMAN, MEMBERS OF THE COMMITTEE, it is an honor to be here today to testify on behalf of the American Cancer Society, a voluntary health agency with over 2-1/2 million active volunteers.

I am Professor of Surgery at the Cornell University Medical College, and serve as attending surgeon, the Thoracic Service of Memorial Hospital, in the Memorial Sloan-Kettering Cancer Center. I am a long-time, active volunteer of the American Cancer Society. I have served as a Member of the Committee on Tobacco and Cancer of the Society's National Board of Directors, and currently serve on the Society's Ad Hoc Committee on Tobacco Habitation. As a clinical researcher, I conducted some of the early research experiments in animals which helped to establish the causal relationships between smoking and cancer.

I would like to start out by commending the Chairman and the members of his Committee who have worked for the passage of this legislation in the last year.

I sincerely hoped last year when I testified before the House that I would not have to testify on this legislation again, not because I believe it to be anything but a privilege to appear before this Committee but, because I had hoped the last Congress would understand the importance of this legislation and pass it. Since they did not, I appreciate the opportunity to, once again, express my views and those of my fellow members of the Cancer Society, or S. 772.

I am here today to express the strong support of the American Cancer Society for this legislation and for education on smoking. 51 million Americans light up cigarettes every day and we
know that that act will contribute to the death of over 300,000 of them this year. 440,000 Americans will die of cancer this year; 129,000 will die of smoking-related cancers.

Despite Tobacco Institute claims to the contrary, the Surgeon General says that 86% of the 111,000 lung cancer deaths this year will be smoking related. We know from the Surgeon General’s report that the overall cancer death rates of male smokers are approximately double those of non-smokers and for female smokers the death rate is approximately 30% higher.

The higher cancer rates for men reflect the fact that in the past, more men than women smoked, and smoked more heavily. In recent years, however, the gap between male and female smoking has been narrowing. 25 million women now smoke and because of that before the end of this decade it is estimated that lung cancer will surpass breast cancer as the number one cancer killer of women. The only possible way to avoid this eventuality is to drastically reduce the number of women smoking.

The Surgeon General found that cigarette smoking was a major cause of lung, laryngeal, oral cavity, and esophageal cancer. It was also found to be a contributory factor to bladder, kidney and pancreatic cancer. In addition, he noted that epidemiological studies suggest an association between cigarette smoking and stomach cancer and a possible association between smoking and uterine and cervical cancer.

What is even worse, those cancers most closely associated with cigarette smoking (lung, esophageal, laryngeal, oral cavity and pancreatic cancer), can be the most difficult ones to treat.
the cancers with the least hope for survival. For example, the overall five-year survival rate for lung cancer is only 10%; for cancer of the esophagus, 4% and for cancer of the pancreas, 2%.

There are those who would say that none of this is true, that cigarette smoking does not cause cancer. This is nonsense. Every surgeon general for the last twenty years has said that cigarette smoking causes lung and other cancers and has backed that statement up with considerable data. As I said earlier, I myself conducted some smoking studies and I know from firsthand experience what the findings are. Cigarette smoking is the leading cause of lung cancer and that simply cannot be disputed.

If our opponents say that we do not know exactly what triggers a cancerous condition in the cell I can only say that they are right about that. What is important, however, is that we know that whatever the mechanism is for turning a normal cell into a malignant cell, cigarette smoking triggers that mechanism in around 129,000 Americans every year.

If our opponents say this knowledge is based on epidemiological studies I must admit that is partially true and I must also say I am not an epidemiologist. I know that the studies done on smoking are not that different than the studies done on asbestos and other substances which have been widely acceptable as being carcinogens. Furthermore, could cigarettes survive the Delaney test if they were covered under the Food, Drug and Cosmetic Act? I submit they could not.
The plain and simple truth is smoking causes cancer. I know it, my fellow Thoracic surgeons know it, and I believe those in the industry knew it too.

The only question that should be of interest to this panel then is whether this bill will be effective. I am no advertising expert. However, I have seen some information on the European experience with labeling that I think is pretty convincing. For example in Sweden, which adopted a rotational warning system with 16 different specific health warnings in 1977, the percentage of adult male smokers dropped from 43% in 1976 to 31% in 1980. The percentage of female adult smokers decreased from 34% to 25%. Of even greater interest, the percentage of 13 year-old children who smoked was nearly cut in half between 1977 and 1980. A major study done for the Swedish government after the implementation of this rotational warning system concluded that not only had the warning labels been noticed and understood, they had had a direct impact on both the public’s knowledge of the health hazards of smoking and the reduction in the percentage of people who smoke. Similar experiences have occurred in Finland and Norway.

Our own label which says nothing about how cigarette smoking is hazardous, what diseases it causes, is far from conspicuous. It is no wonder it has ceased to be effective. During almost that same period cigarette smoking among young females age 17-18 in our own country increased. As a doctor I cannot express how strongly I believe that if these labels accompanied by vigorous public education can stop just 1% of our children from starting to smoke.
or 10% of our adults from lighting up they are worth every penny of
cost to the industry or to any of us asked to foot the bill.

As for the provision of this legislation on cigarette additives, the Cancer Society feels very strongly that these sections
must remain in this bill. As a scientist I am outraged that an
industry that produces a product that kills is permitted to keep
secret the additives of that product, any of the additives, even
ces, from responsible public health officials. We know that
ingredients used to flavor cigarettes such as deertongue and cocoa
barks contain substances which are carcinogenic. That we do not
know what else in cigarettes is a carcinogen causes me great dis-
tress. That we may never know leaves me discouraged and fearful
for all the patients I will have in future years who will
needlessly die.

I have had to watch the misery of cancer in thousands of
patients, in recent years, increasingly in women. Dr. Langston,
testifying before the House said that lung cancer may have created
as a disease. I know from personal experience this is not true.

Mr. Chairman, I am at your disposal when it comes to this
legislation. To be here today I left many patients in New York who
are in serious trouble. Most have lung cancer. This disease in
women is becoming epidemic in my experience and the experience
of my fellow Thoracic Surgeons. It makes us heartsick and angry.
Whatever I can do to help the women of younger generations to
understand what they are doing to themselves when they smoke,
I will do. I believe this bill is important in that process.
If the members of your Committee could spend one week with me, one week with my patients, lung cancer patients of which only one in ten have any hope of surviving for five years or more. I believe you would have a unanimous vote for this bill. Since you cannot, I can only urge you to try to understand my frustration and the pain and misery of my patients.

Help us to help these people avoid having to seek the kind of help I can provide, which is all too often not enough to save their lives.

The CHAIRMAN. I have listened to a lot of testimony, marvelous testimony, on this subject. I do not know of anybody who has hit the nail on the head as well as you have.

Dr. CAHAN. Thank you.

The CHAIRMAN. I just personally very much appreciate it. Would you be kind enough to let me have those two pictures? They look like they are expensive, so I would be happy to pay for them.

Dr. CAHAN. I would be glad to.

The CHAIRMAN. I would appreciate that. We will try to disseminate those because I think people really need to see things like that.

I am personally very appreciative of your testimony here. All of the testimony has been excellent today, from what I have been able to observe.

If you will wait, we would like to ask some more questions of you. I would just like to ask one right now, just to emphasize your point.

I happen to feel that one of the biggest problems we have in America today is, of course, the health of our people. I also believe that it is becoming a more and more expensive process to have the Federal Government and any and all financing approaches pay for the health of our people. You have made a point here today that one of the best things we can do to help the health of our people is through preventive medicine, and one of the best things we can do in preventive medicine is to help people to realize the evils of smoking and how harmful and deleterious it is to the human being and to the human mechanism.

Do you agree with me that we would save billions of dollars if we quit, just in health costs alone from the Federal Government, if we could get people to quit smoking?

Dr. CAHAN. The cost—well, it would be enormous. I don’t know the figures. I am poor at such things.

Do you mean the cost of Government—

The CHAIRMAN. Yes.

Dr. CAHAN. I am told it is in the billions.

Balance is often talked about in terms of the cigarette industry saying how much they would lose by all these things, and of course what you have just said is that it is way overweighted on the side
of how much would be saved and how many lives would be saved, not only that, but how many tragedies would be averted.

The CHAIRMAN. Well, if you add lives, tragedies, costs of medical care and treatment, and all of the other aspects to the cost of health care needed because of smoking, it amounts to billions of dollars every year. That is one of the points we are trying to bring out.

We appreciate it.

Dr. Oates, let's turn to you.

STATEMENT OF JOHN A. OATES, M.D., PROFESSOR OF MEDICINE AND PHARMACOLOGY, VANDERBILT UNIVERSITY, AND CHAIRMAN OF THE SUBCOMMITTEE ON SMOKING OF THE AMERICAN HEART ASSOCIATION, REPRESENTING THE AMERICAN HEART ASSOCIATION

Dr. Oates Mr. Chairman and members of the committee, I am Dr. John Oates, professor of Medicine and Pharmacology at Vanderbilt University and chairman of the Subcommittee on Smoking of the American Heart Association.

I appreciate the opportunity to testify on behalf of the Heart Association, a nonprofit, voluntary health organization with over 120,000 members and almost 2 million other volunteers who are dedicated to the reduction of premature death and disability from cardiovascular disease.

Cardiovascular disease kills nearly 1 million Americans each year. This year about 1.5 million Americans will have a heart attack, and about 550,000 of them will die. Another 174,000 people will develop severe peripheral vascular disease.

These figures have a special significance because cigarette smoking has been firmly established as a major contributor to the occurrence of myocardial infarction, sudden coronary death, and peripheral vascular disease. Cigarette smoking is the most preventable cause of death and disability from these cardiovascular diseases.

The total of coronary deaths from cigarettes is exacted largely in mid-life, ages 35 to 65. Compared to nonsmokers, cigarette smokers are more likely to die from myocardial infarction and to die suddenly from coronary artery disease.

Fortunately, quitting smoking reduces the risk toward that of nonsmokers.

The American Heart Association and the Coalition on Smoking or Health are committed to helping smokers who want to quit and preventing children from starting to smoke. Accordingly, we wholeheartedly support the passage of S. 772. A congressional mandate for a program to inform the public on the health risks of smoking can be instrumental in combining and coordinating the efforts of the public and private sectors to address the problem of cigarette smoking.

Thus, a relatively small Federal effort can be used to mobilize larger private resources to address a major health problem.

Requiring the rotation of new warning statements on cigarette packages is needed to better inform the public since the current warning statement is overexposed and worn out. This should come
as no surprise, since any message presented exactly in the same way will soon become so familiar that it will lose its effectiveness. Clearly, there is a need for varied warning statements in a visible format. Yet, the tobacco industry argues that the warning label should remain the same. This is contradicted by the practices of the industry itself, which changes advertising copy frequently to sell cigarettes. It is obvious that the health message also must be changed to attract attention.

Cigarette advertising uses a multitude of images to appeal to different markets. A logical extension of this is that the warning must also be tailored to appeal to differing consumers. For example, smokers with a history of heart disease in their families may find a warning on smoking and heart attack more personally relevant than a general message.

The Heart Association has insufficient access to the media to inform the public adequately on the hazards of smoking. The number of antismoking public service announcements aired on television since the ban on cigarette commercials has been drastically reduced, with hardly any being shown during prime time.

Information in the print media has been equally scarce. It has been suggested by some that heavy advertising by the tobacco industry serves to discourage coverage of the hazards of smoking in the print media. This potential for intimidation was reinforced when a cigarette advertising account of 500,000 pounds sterling was abruptly withdrawn from London's Sunday Times after a report on heart transplant patients which named the brands of cigarettes smoked by all five of the patients.

For whatever reason, the broadcast and print media do not provide the public with full information on the dangers of smoking. Accordingly, the warning labels would provide an assurance that all citizens have access at least to a minimal level of information. They would provide an important means by which the Federal Government, at no cost, can effectively assist the private sector in educating the public about the hazards of smoking.

The Heart Association affirms that citizens should have a freedom of choice regarding risks that they wish to take. This important freedom carries with it the responsibility that decisions to incur substantial risk from commercial products be informed choices. The decision to smoke is usually made before the age of 21, usually without full awareness of the odds against escape from the smoking habit.

This legislation would become an important step toward assuring that it is a more informed decision.

Thank you.

[The prepared statement of Dr. Oates follows:]

161
TESTIMONY
OF
JOHN A. OATES, M.D.

CHAIRMAN, SUBCOMMITTEE ON SMOKING
AMERICAN HEART ASSOCIATION

Before the
COMMITTEE ON LABOR AND HUMAN RESOURCES
UNITED STATES SENATE
CHAIRMAN ORRIN G. HATCH

ON

S.772, "THE COMPREHENSIVE SMOKING PREVENTION
EDUCATION ACT"

MAY 5, 1983
Mr. Chairman and members of the Committee on Labor and Human Resources
my name is John A. Oates, M.D. I am Professor of Medicine and Pharmacology
at Vanderbilt University and Chairman of the Subcommittee on Smoking of the
American Heart Association.

I appreciate the opportunity to appear before this Committee on behalf of the
American Heart Association to testify in strong support of the "Comprehensive
Smoking Prevention Education Act of 1983." The American Heart Association is
a nonprofit voluntary health organization with over 120,000 members and almost
2 million volunteers. The primary objective of the Association is the reduction
of premature death and disability from cardiovascular diseases.

Cardiovascular diseases kill nearly one million Americans each year. This is
more than all other causes combined. Heart attack, the nation's number one
killer, claims most of these lives. This year, as many as 1.5 million Americans
can be expected to have a heart attack and about 550,000 of them will die.
The survivors will join over 4 million Americans who have a history of coro-
nary disease.

These figures have special significance because cigarette smoking has been
firmly implicated as a major risk factor for heart attack, sudden death and
peripheral vascular disease and it greatly aggravates other forms of cardio-
vascular diseases. Cigarette smokers are more likely than non-smokers to
suffer a heart attack, more likely to die from these attacks and more likely to
die suddenly. This effect is directly related to the amount smoked, with heavy smokers being at three times the risk of non-smokers. Cigarette smoking is a particularly powerful contribution to heart attacks and sudden death that occur at younger ages. Based on data from the Framingham Heart Study, we estimate that over 174,000 Americans will develop peripheral vascular disease this year. While twenty percent of these people will be diabetics, 70% of the remainder will be cigarette smokers. Moreover, diabetics who also smoke are at even greater risk. The link of cigarette smoking to peripheral vascular disease is consistent and independent of diabetes and other risk factors and related directly to the number of cigarettes smoked.

Perhaps most significant is the fact that when people stop smoking, their risk from these cardiovascular diseases gradually returns to normal. The risk of fatal and non-fatal heart attack among ex-smokers gradually returns to that of non-smokers and the risk of peripheral vascular disease is similarly reduced. As one of the labels of the bill states, “Quitting now greatly reduces the risk to one’s health.”

The evidence incriminating cigarette smoking as a major risk factor for heart attack and peripheral vascular disease has been reviewed by an expert panel of the American Heart Association and has been judged to be conclusive. Furthermore, the panel concluded “Theoretically, cigarette smoking is the most preventable cause of these cardiovascular diseases and mortality therefrom.”

In spite of the overwhelming evidence linking cigarette smoking to cardiovascular and other diseases, over 54 million Americans are still smoking. However, two thirds of all smokers would like to quit and most have tried to
do so. Following an unsuccessful attempt to quit, many of these smokers tend to switch to low tar and nicotine cigarettes. This is evidenced by the continuous increase in the market share of these cigarettes to 60.9% of all sales in 1981.

This development is alarming because available evidence suggests that many people switch to low tar and nicotine in an effort to lower their risk of adverse health effects. While switching to these cigarettes may lower the risk for some diseases, there is no evidence of a reduction in risk for cardiovascular diseases. Any public perception that low tar and nicotine cigarettes are safe could be dangerous since most cigarette-related deaths are from heart attacks.

The tobacco industry has attempted to use the findings of the recent CHNRFIT study to show that cigarette smoking is an insignificant risk factor in cardiovascular disease. The industry avoids the conclusions of the study which showed that both groups, the "special intervention" group and the "usual care" group reduced their risk factors over the term of the study. As Dr. Brandt noted at the release of the findings of the study, "It emphasizes that the "usual care" afforded to American results in reduced risks for American men. This study again shows the value of reducing one's risk of coronary heart disease. For example, the death rate for those who quit smoking for one year was about half that of those who did not."

There can be no doubt that the Congress of the United States -- and this Committee in particular -- has the authority to ensure that we have adequate laws enacted to protect the safety and health of the American public. The Food, Drug and Cosmetic Act ensures that our foods, drugs and cosmetics are
safe and effective and that where a potential hazard exists they are regulated and labeled to give adequate warning to the users. The Consumer Product Safety Act ensures that consumer products do not present unreasonable risks to consumers and that if risks exist, the products are labeled to inform and warn the users of risks. And there are many other health and safety laws which the Congress has enacted to ensure the protection of the American public. These include the Fair Packaging and Labeling Act, the Toxic Substances Act and the Federal Hazardous Substances Act.

The tobacco industry has made the claim that "no other product lawfully manufactured and sold in the U.S. is subject to such burdensome requirements as those proposed... in the Comprehensive Smoking Prevention Education Act." (Ironically, however, tobacco and tobacco products are probably the least regulated products in the U.S. when it comes to health.) Actually, tobacco and tobacco products are neither foods nor drugs under the Food, Drug and Cosmetic Act although many of the additives of cigarettes should be considered as drugs and tobacco continues to be shipped overseas under the Food for Peace Program. Tobacco and tobacco products are also specifically exempt from statute under all of the acts I just mentioned, the Consumer Product Safety Act, the Fair Packaging and Labeling Act, the Toxic Substances Act, and the Federal Hazardous Substances Act.

A few years ago AHA presented testimony before the House Subcommittee on Health and the Environment on the issue of whether saccharin should be removed from the market (as required under the Food, Drug and Cosmetic Act) because it had been determined that saccharin causes cancer in animals.
It took an act of Congress to continue to allow saccharin to stay on the market. With saccharin we were confronted with limited studies and it was determined that the scientific evidence was sparse and inconclusive. It was the position of the AHA that the science implicating saccharin as a threat to health was insufficient. This is not the case for tobacco. The case against cigarettes is clear and scientifically substantiated. Because a handful of scientists do not accept these conclusions, does not make it a controversy. Unanimity of medical opinion on complex issues is rare. The relationship of cigarette smoking to disease is not controversial. Since 1964 some 35,000 scientific studies have amassed overwhelming evidence that is widely accepted by the scientific community. The tobacco industry's claim is purely and simply a public relations tactic to create doubt and confusion in the minds of the public. In fact, if tobacco and tobacco products were regulated under the Food, Drug and Cosmetic Act, the Delaney Clause would have required their removal from the market long ago. It is interesting to note that for a "regulated" product such as saccharin the Congress requires a disease specific warning, but in the case of cigarettes nothing specific is required.

The legislation before us today does not attempt to remove a product from the market nor does it infringe upon the advertising formats which the tobacco companies may use in selling their products. What the legislation does is more adequately warn the public of the harmful effects of cigarette smoking. What the legislation does also is to replace the present worn out unspecific warning label with a set of rotating warning labels. These new disease specific labels would be much more informative than the present label.
The provisions requiring rotation of the four new warning statements on cigarette packages are an essential element of any comprehensive educational effort if the public is to be better informed of the specific dangers of cigarette smoking. The rationale for the requirement of a warning statement in the first place was to inform consumers of the health hazard. However, the FTC has recently concluded that the current warning statement, which has been used on packages and advertisements since 1972, is overexposed and its effectiveness is very limited. This should come as no surprise since any message presented exactly the same way will soon become so-familiar that it will lose its effectiveness.

The American Heart Association is committed to helping smokers who want to quit and preventing children from starting to smoke. Accordingly, we enthusiastically support the passage of S. 772 the "Comprehensive Smoking Prevention Education Act of 1993". The provision of statutory standing for the functions of the Office on Smoking and Health will be instrumental in combining and coordinating the efforts of the public and private sectors to address the problem of cigarette smoking. This is a clear case where a relatively small federal effort can be used to mobilize enormous private sector resources to address a major health problem.

In the 1981 Staff Report of the Federal Trade Commission on Cigarette Advertising, it was concluded after assessing numerous surveys that "there appear to be significant gaps in consumer knowledge that a substantial portion of the population does not know how dangerous smoking is, or whether the dangers apply to them." The Report went on to conclude that...
(a) a large number of consumers do not know what diseases are related to smoking.

(b) a substantial number seriously misunderstand and underestimate the extent of the increased risk of suffering these health problems.

(c) many seriously underestimate or are unaware of severity of these health consequences and the increased risk of premature death from these illnesses; and

(d) a significant number of consumers incorrectly do not believe that these health consequences have any personal relevance to themselves.

Meanwhile, the so-called "brand advertising" goes on at the tune of over one billion dollars per year. Regardless of the intent, non-smokers and children are heavily exposed to the promotion of cigarettes. It would be very difficult to argue convincingly that some non-smokers, especially children and teenagers are not affected by the constant portrayal of smokers as being young, attractive, healthy and enjoying an adventurous, pleasurable lifestyle. The industry continues to portray smokers in this image even though it maintains that smoking is an "adult" custom. Cigarettes are available in practically every drug store, supermarket, gas station, restaurant, and convenience store in this country. Free packages are handed out by the thousands on the streets and through the mails. Cigarette companies have gone out of their way to relate their products to foods (KENTS), healthy activities, success,
social prominence, distinction, and sexual attraction. Ironically they supposedly adhere to voluntary codes which specifically prohibit such practices.

Although there are effective messages that counter this kind of bombardment, comparable resources available are insufficient. It is not difficult to understand why 52% of smokers do not know or believe that smoking is a major risk factor for cardiovascular diseases. All of this coupled with the industry's continued denial of the health risks makes it all the more important that we remind people, and remind them often of the dangers.

Last year this Committee heard testimony from both the Administration and other public witnesses about the addictive nature of cigarette smoking. In early March, the Public Health Service issued a pamphlet entitled "Why People Smoke." In the opening paragraphs of the pamphlet, cigarette smoking was cited as being the "most widespread example of drug dependence in our country." It was stated that cigarettes are:

- psychoactive
- create dependence and lead to compulsive use
- that when one gives up the use, one may experience physiological and psychological distress and
- that there is a strong tendency among former users to relapse sometimes months or years after quitting.

The addictive nature of cigarette smoking makes it all the more reason to ensure that the Public receive adequate and specific warnings about the
health hazards. Keeping people from starting is therefore all the more important.

We are pleased to see that the legislation includes as part of one of the proposed new warning labels a specific reference to the addictiveness of cigarettes. As the FTC report noted, "evidence indicates that the majority of people do not think smoking is addictive." What is particularly important to note is that most teens believe that cigarette smoking is "okay if they quit before it becomes a habit." But the data is clear that a vast majority of smokers who smoke as an experiment become regular users.

The warning label becomes a very important medium to get more information to the public. The labels proposed are short, concise and specific. The American Heart Association has made and will continue to make efforts to better inform the public about the hazards of smoking. However, we do not believe that we have adequate access to the media to fully inform the public. Our efforts will have greater chance of success if the clearly deceptive intention of cigarette advertising is attenuated by including an effective warning. The warning is yet another way, at no cost, in which the federal government can effectively assist the private sector in educating the public about the hazards of cigarette smoking. It is not the only means of education or the sole answer to the problem, but it is a critical part of the effort and a part which only the federal government can play.

Therefore, the American Heart Association strongly endorses S. 772 and hopes that this committee will move expeditiously in ensuring its enactment.
The CHAIRMAN. Thank you, Dr. Oates.

Let's now turn to Dr. Edwin Fisher, associate professor of psychology at the Washington University in St. Louis, Mo.

STATEMENT OF EDWIN B. FISHER, JR., PH. D., ASSOCIATE PROFESSOR OF PSYCHOLOGY, WASHINGTON UNIVERSITY, ST. LOUIS, MO., REPRESENTING THE AMERICAN LUNG ASSOCIATION

Dr. Fisher. Thank you.

Along with that, I am also on the board of the American Lung Association and chair of its Smoking and Health Committee.

I am delighted to appear before you, along with my colleagues in the Coalition for Smoking or Health.

I also direct the Behavior Therapy Clinic at Washington University in St. Louis and, as such, have been directly responsible for a variety of programs on smoking cessation, adolescent smoking prevention, weight loss, diabetes, nutrition management, and related issues over the past 5 years. As such, I feel I have a fair amount of direct clinical experience in dealing with the problems of people trying to change their behavior in ways similar to or including not starting to smoke and ceasing to smoke.

Now I would like to discuss a little bit the current warning label. As a behavioral scientist, it struck me this morning in listening to some of the testimony and some of the questioning that the current warning label does exactly what we would expect it to do. It is a general warning label. It provides general, vague information about the risks of smoking. The result of that warning label, after over a decade, is that the public at large has a general, vague understanding of the risks of smoking. Unfortunately, what research and behavioral science, psychology, education, and health education indicate is that vague, general understandings of issues do not really assist people in taking helpful or specific or directive action.

As an example of some of the really disturbing deficits in specific knowledge about the risks of smoking, I draw you to just one from a compendium developed by the Federal Trade Commission in 1981. Based on reviewing a variety of surveys and interview studies and focus panel studies, they came up with data that, although 10 percent of chronic obstructive pulmonary disease is caused by smoking, 34 percent of smokers do not know that smoking causes emphysema. Fifty-five percent of smokers do not know that smoking causes bronchitis.

While many of these people know that smoking is somehow bad for them, they are painfully unaware of the real specific risks in this area.

Dr. Cahn has discussed and presented very effectively the risks of smoking for cancer. I think it is important to keep in mind that, as strong as that association is, the prevalence of death associated with smoking is much more in the area of heart disease. Additionally some of the links between smoking and disease are stronger in the area of lung disease.

Yet, when people come to our smoking clinics, they quit, they stay off for a week or two, and then they start relapsing. They say, "Well, I guess you've got to go some way, and I guess lung cancer is as good as any." They are shockingly unaware of the fact that
there are many, many risks that they encounter by returning to smoking that are independent of and additive to the risks of lung cancer.

The knowledge is not widely understood in the land. To speak of knowledge of the risk of smoking as being generally accomplished I think is really a misreading and misdescription of what we do know about severe deficits in knowledge of the specific, actual links between smoking, disease, and death.

I would like to speak a little bit about the rotating warning label. There are three principles in psychology and education that I think are particularly pertinent to this.

The first is habituation. As psychologists use the word, we mean that when you repeat a stimulus many times, it loses its impact; it loses its value. The warning label that has been repeated on the sides of cigarette packages for over a decade now has no doubt lost much of its impact. We know that this is a common finding in a variety of areas of information processing, and it appears to be happening with that label.

Putting things more positively, we know that in order for people to learn material, they learn best when the material is presented in concrete, meaningful terms, such as are proposed in the bill—terms like "lung cancer", "emphysema", "heart disease", "miscarriage", "addictive". These are concepts that the average American understands very clearly. These are the sorts of concepts that get learned.

Senator Quayle was proposing this morning that perhaps we get into a voluntary, negotiated agreement with the tobacco companies. I am concerned that the language of bureaucratic compromise that such an agreement would likely manifest would hardly be in the concrete terms that people need. If we are interested in educating the public, we need to educate them in real terms, not in the terms that negotiators agree upon as a way to satisfactorily "vague up" an issue.

Third, there is substantial evidence that specific information is the sort of information that best allows people to guide their behavior. The specific information in the proposed warning is a vast improvement over the vague, general information in the past warning.

I would also like to commend the proposed warning C, which not only gives specific information, but gives a specific thing one can do with a specific address right there on the pack. The way to help people act is to give them specific information as to why they might and a specific path to take. I think these warning labels are an admirable example of cost efficiency in both these areas.

Some experience has been mentioned with these labels over the past decade or so. Comprehensive health education related to smoking, such as proposed in this bill, has been tried in Norway, Finland, and Sweden, and has achieved rather remarkable successes. Between 1977 and 1980 within Sweden there were 9 percent decreases in the percentages of adult males and adult females smoking, following the implementation of a comprehensive health education program on smoking, including labels such as proposed here.

In Finland, the percentage of smokers among secondary school students was decreased by 50 percent following the enactment of a
similarly comprehensive program between 1977 and 1979—a 50 percent decrease in only 2 years in the group that perhaps we are most concerned about, adolescents.

Now several people discussing legislation like this have proposed that the parallel with alcohol be considered. You all, of course, are concerned about alcohol, but it occurs to me that considering such a parallel, one also needs to consider the track records of the companies, of the industries, the producers, manufacturers, distributors involved.

I remember when I was in high school that a spokesperson came around, sponsored by the alcohol distributors, and he told us about the risks of drinking and the importance of responsible drinking. As far as I am aware, no spokesperson from the tobacco companies has ever appeared at American high schools informing adolescent smokers about the risks of smoking.

I think that the relative levels of good citizenship of the two industries needs to be weighed in comparing potential Government dealings with them.

The case of smoking—I think needs to be seen as singular. The risks justify it. Low levels of public knowledge justify it. The track records of the industry in reporting about the nature and effects of its products justify it.

My last concern is with freedom. Some may say that this bill constitutes yet another attempt by Government to interfere with the freedom of individuals in this country. I would like to put that claim in a special perspective. Every year the voluntary associations and the Government together spend somewhere between $50 and $100 million informing people about risks from smoking. One hundred million is certainly a very optimistic estimate. The exact figure is, no doubt, closer to the $50 million. In contrast, every year the tobacco companies spend over $1.2 billion in this land marketing what the Public Health Service recently called the most widespread example of drug dependence in our country which causes more illness and death than all the other drugs. That $1.2 billion buys a lot of advertising. That $1.2 billion pays for a lot of free samples being handed out to people on the street corners in towns across this land. That $1.2 billion pays for a lot of giveaways at professional sports events to young fans and their families. These things influence people. They cause smoking.

What does freedom mean in this context? I would like our children to be free to grow up without those seeking the widespread consumption of such deadly drugs outspending those trying to prevent this consumption by a ratio of 24 to 1.

I think this bill is admirable, positive, and constructive and should be an effective attempt to begin to redress this imbalance.

I thank you for your attention.

[The prepared statement of Dr. Fisher follows:]
THE Comprehensive Smoking Prevention Education ACT
S. 772

Testimony of the
American Lung Association

Presented by
Edwin J. Fisher, Jr., Ph.D.
Chair, Smoking and Health Committee and
Member, Board of Directors of the
American Lung Association

Committee on Labor and Human Resources
United States Senate

Washington, D.C.
May 5, 1983
Testimony of Dr. Edwin E. Fisher, Jr.

It is a pleasure to testify before this committee.

My name is Edwin E. Fisher, Jr. I am here representing the American Lung Association. I am Chairman of the Lung Association's Smoking and Health Committee and also a Board Member of the Lung Association. I hold similar positions in the American Lung Association of Eastern Missouri in St. Louis, where I live.

In my personal life, I am an associate professor of psychology at Washington University in St. Louis. I am also Director of the Behavior Therapy Clinic in the Department of Psychology there. I am also Associate Director of our NIH funded Diabetes Research and Training Center in our medical school.

In the Behavior Therapy Clinic, which I direct, we have run a number of programs in health education over the past five years for weight loss, nutrition management of diabetes, smoking cessation, and a variety of other problems. Thus, I have substantial clinical experience in helping people to change problems such as smoking.

Let me start by reminding the Committee that the American Lung Association has never supported a ban or prohibition on smoking or cigarettes. We seek to inform the public and to help individuals act in accord with decisions they make for their own welfare.

I would like to focus on several aspects of the bill before us today.

First of all, as background, I would like to review briefly the Federal Trade Commission report on advertising of cigarettes and public knowledge of health effects of smoking. This report was published in May 1981.

As a behavioral scientist, I am impressed by a particular aspect of this report: that is, the findings in the report hold up against repeat testing by different researchers with different samples. The report is based on a number of surveys,
Interview studies and focus panels conducted by a variety of researchers in the private sector, university-based departments, and government circles. All of these researchers come to a similar set of conclusions about severe knowledge deficits in the public regarding the specific health risks associated with smoking.

This sort of replication of findings across studies is the hallmark of high scientific responsibility. Therefore, I think the report is particularly deserving of our attention.

The FTC report showed that there is a general awareness in the public that there is something wrong with cigarette smoking. However, there is a low level of awareness of risks of smoking associated with diseases other than lung cancer. There is also generally poor awareness of who may be more at risk.

I am painfully shown this every time I run a smoking cessation clinic. The people come in, join, and quit. As some begin to relapse, they often say things like, "I guess I have to go' somehow. It may as well be lung cancer." They are not even aware of the fact that they are running many risks besides lung cancer.

I would like to highlight just a couple of areas that seem especially important.

It is a fact that heart disease accounts for most of the excess deaths among smokers in this country. Yet 32 percent of the public and 37 percent of smokers do not know that smoking causes disease.

Similarly, smoking causes 70 percent of cases of "chronic obstructive pulmonary disease." This is fancy talk for emphysema and bronchitis and a few other lung diseases. Yet 34 percent of the public does not know that smoking causes many cases of emphysema. Thirty-nine percent of smokers do not know this. Similarly with bronchitis, 55 percent of the public at large and 60 percent of smokers do not know that smoking causes many cases of this disease.
Together, cancer, heart disease, and chronic obstructive pulmonary disease account for 62 percent of the deaths in the United States each year according to figures from the National Center for Health Statistics.

With this background, I would like to look for a moment at the provision in this bill which would establish an area within the Department of Health and Human Services to carry out a program to inform the public in a systematic manner about the risks of smoking.

It seems to me the knowledge deficits which I have sampled from the FTC report demonstrate the need for vigorous action in this area. The American Lung Association, as well as the American Cancer Society and the American Heart Association are certainly eager to cooperate with the Federal government in a broad-based program to inform the public. However, we need the government to take its rightful role in this. Owing to the health impact of smoking, that role should be a major one.

A critical consideration for you on this may be whether the public really wants to know. Is there a public interest to be served by the sort of information to be provided?

Two months ago, the Public Health Service made an announcement on the addictive characteristics of smoking. This received front-page coverage in many U.S. newspapers. Colleagues of mine running smoking cessation programs all over the country have mentioned to me substantial interest among their program participants in this statement. We all were deluged by questions about it in our clinic meetings following the PHS announcement.

Note that the PHS statement did not give news of a "new miracle cure" for a dreaded disease. It merely gave the public useful information about the nature of a problem which it faces. Yet, it was widely and eagerly read. This helps us understand that the public has a deep and serious interest in learning more about the things which affect its health.
Perhaps the most controversial aspect of the proposed bill is the retention of labels. I would like to refer to that with respect to well respected and well documented psychological and educational principles.

The word is known in the field as "habituation." To avoid confusion, psychologists use this word in a slightly different form than used to refer to additive processes regarding smoking in several of the Surgeon General's reports and other government documents.

As used here, "habituation" refers to the fact that the frequent repetition of a stimulus causes it to lose its effects. Thousands of subjects from Pavlov's dogs to numerous participants in countless studies of human information processing have shown this aspect.

The current warning label has been on cigarette packs for over a decade. It certainly has suffered from habituation, causing the impact of the label to be diminished considerably.

A second consideration is that accurate, meaningful concepts are much better remembered, much better learned, than vague concepts. The current warning label is a vague, general warning. Such language of bureaucratic compromise will not be well-remembered by most consumers.

As opposed to the current language, a clear, crisp warning should be in terms that are meaningful, such as, "death," "premature birth," "addiction," "suffering now," and "miscarriage." These are terms that are well-understood by the majority of the public, and will cause the labels to be meaningful to them. There is an absolute need in fulfilling our duty to inform the public if we inform the public in vague language which it does not understand.

Finally, I would like to mention a well-understood principle of health education. This is that specific information regarding risks and ways in which they may be remedied is more effective than vague information. Again, the proposed warning labels put in specific terms the risks of smoking, matching the specific knowledge deficits noted earlier.
Experience with informative warning labels as this bill proposes has been gathered in a number of countries. But it is important to understand that this is not just a bill for warning labels. It is a comprehensive smoking education bill. I think that is exactly what is needed. Norway, Sweden, and Finland have carried out comprehensive smoking education and realized substantial reductions in the percentages of smokers among adults and teenagers. In Sweden, this led to a nine percent decrease in the rate of smoking among adult males and females. The percentage of 13-year-olds who smoked in Sweden was cut by nearly 50 percent between 1977 and 1980. In Finland, the percentage of secondary school students smoking dropped from 27 to 13 percent between 1973 and 1979. In Norway, the percentage of male smokers between the ages of 15 and 21 dropped from 82 to 30 percent between 1978 and 1980.

I would like to discuss briefly the issue of additives to cigarettes. Currently, cigarette companies, at least one or two of them, can put almost anything they like in a cigarette without informing the consumer about it. Given the documented risks of smoking, this stands as a glaring exception to the fine American tradition of free flow of information which is helpful to people living their lives.

In planning government policy and actions, there has to be some consideration of the track record of the particular industry or product in question. Several defenders of the cigarette companies point out the lack of smoking warnings, etc., on alcohol, for instance. But, I remember when I was a high school student that a spokesperson came around sponsored by the alcohol distributors. He told us about the risk of drinking and the importance of responsible drinking. As far as I am aware, no spokesperson has ever appeared in American high schools for the tobacco industry informing adolescent smokers about the risk of smoking. I think that the relative good citizenship of the two industries needs to be weighed in comparing government dealings with them.
The case of smoking needs to be seen as singular. The risks justify it. Low levels of public knowledge justify it. The track record of the industry in reporting about the nature and effects of its products justify it.

I would like to address two further points. One draws again on the NIC report on knowledge of risks of smoking. From the behavioral science perspective, one of the striking parts of the data in the report is the difference between the public at large and smokers regarding the awareness of smoking-related risks. Across almost every issue, every health risk surveyed, there is a reliable difference indicating a greater portion of the public at large than of smokers knows of the health risk in question. The differences range from 4 to 11 percent between nonsmokers and smokers.

Now, this looks like a pattern of which psychologists have long been aware. It is the human being's tendency to avoid learning what she or he would prefer not to know. We have done a fair amount of research on how to help people learn what they need to know. This research has taught us that it is important that people receive consistent information from many sources when their tendency not to want to know needs to be counteracted.

Given the importance of consistent, broad-based information on emotional issues such as health effects of smoking, it is especially important that the task of informing the public in these matters not be left merely to the voluntary agencies or to the TV and radio stations willing to donate a little bit of time. We need to have a concerted, coordinated effort in which many credible sources of information in the field come together to inform the public. Certainly, the government belongs in a prominent role in this effort.

My last concern is with freedom. Some may say that this bill constitutes yet another attempt of government to interfere with the freedom of individuals in this country. I would like to put that claim in a special perspective.
Every year, the voluntary associations and the government together spend somewhere between $50 and $100 million informing people about risks from smoking. The $100 million is certainly a very optimistic estimate; the exact figure is no doubt much closer to the $50 million.

In contrast, every year the tobacco companies spend over $1.2 billion in this area marketing what the PBS recently called "the most widespread example of drug dependence in our country" which "causes more illness and death than all the other drugs."

Our children should not grow up in a country in which those seeking the consumption of such a widespread, deadly drug outspend those trying to prevent this consumption by a ratio of twenty-four to one.

Thank you very much.

The CHAIRMAN: Thank you so much. I think the testimony of all three of you gentlemen has been excellent. We really appreciate it.

I am going to turn to Senator East at this time and let him ask his questions. Then I will turn to Senator Pell, and then come back with some questions I have.

Senator East: Thank you, Mr. Chairman. I know you are anxious to wind this up, and I am, too. Therefore, I will be as quick as I can.

Senator Pell: I have several questions.

The CHAIRMAN: As soon as Senator East is through, I will call on you, and then I will wind it up.

Senator Pell: All right.

Senator East: I would like, if I could, Mr. Chairman, to make one comment on what Dr. Fisher has said for the record.

Dr. Cahan: Would you mind talking a little louder? I am sorry, I cannot hear that well.

Senator East: I would just like to make one observation for the record in response to a matter Dr. Fisher has discussed, and then I would like to put a question to Dr. Oates, and then I shall cease and desist because I realize the pressure of time.

I do not wish to sound overly defensive about it, but when you mentioned, for example, this question of advertising of the tobacco industry, not to dispute your concern about any form of advertising, but I would like to point out for the record on the question of balance and proportion and the problems of emotion in this issue. It is to be remembered that there is no cigarette advertising on television. Yet, the beer ads run daily with athletes showing the merits of light beer and this beer versus that beer, and we also know, as a matter of fact, as I was noting earlier in these hearings, the direct cause and relationship between drinking and teenage death in automobiles.

All I am saying for is a little proportion and balance here. The idea seems to be that somewhere the tobacco industry is the great
offender—they have been continually restricted and constricted in what they can do. The notion that some way or another they remain the big offender in this area in appealing to youth is unfounded, and I have not seen any ads on television of prominent athletes pushing cigarettes of late, but I see it with beer, for example, on a daily basis.

It is not to say that two wrongs make a right. I am not arguing that. I am merely asking, as I had at the outset of this hearing, that as we move through this legislation that emotion on any particular element of this not reach such a high decibel level that we lose sight of proportion, balance, and scientific causation.

Also in that context, I was a bit troubled with Dr. Pollin's suggestion, that cigarette smoking is the gateway to other drugs, I think, again, this has an emotional ring to it that may end up discrediting the very cause that he hopes to serve.

Having made that observation, Mr. Chairman, if I might just address this question to Dr. Oates. Dr. Oates, there was an article in the May 3 New York Times in which I would like to quote briefly from and, with the permission of the chairman, make it a part of the record here. This is Tuesday, May 3, 1983, New York Times. "Sex hormone linked to heart disease, new study finds," by Lawrence K. Altman. I just wish to read, Mr. Chairman, the first three paragraphs from it.

I would like Dr. Oates' comment on this study—

A new study, with major implications, has found evidence that men who have suffered heart attacks, the Nation's leading killer, have more of a hormone called estradiol than men without heart disease.

The new report from Columbia University, the National Institutes of Health and the Framingham Heart Study surprised even its authors. The research found no evidence that such commonly suspected risk factors as cholesterol, high density lipoproteins, high blood pressure and cigarette smoking, differed among those who did and did not have heart attacks.

They go on to say,

These findings suggest that heart disease could be primarily a hormonal disorder which, if true, would affect treatment and prevention.

Now I am suggesting that this must eclipse all other studies I would simply like to get your opinion of what you think of this—-not—and I just ask as a layman—suggest the imperfect state of scientific findings today?

I am back to my concern about scientific causation. If, for example, we made the mistake of saying the only rabbit to track here was the smoking rabbit, we might end up being counterproductive to the very cause we hope to serve, namely, a comprehensive and balanced understanding of the cause of heart disease?

Here we have a study, I presume, from a very responsible source, suggesting the problem may be due to hormonal imbalance as opposed to diet, cholesterol, and any other matters.

Again, I repeat that I am not suggesting that this pre-empts all other studies. I am simply suggesting it does indicate the somewhat imperfect world of scientific causation in which we live, and move, and have our being. I would appreciate your comment upon this study.

Dr. OATES, I am delighted that you raise this particular point because I think it takes us into a major misconception about the
impact of cigarettes on heart attacks and sudden death. That is the misconception that these are effects that occur late in life when one is in their eighties and that this may be a convenient way to end a full life. Indeed, this is not the case.

The toll of coronary deaths from cigarettes is exacted largely in midlife, in ages 35 to 65. This study is of note in that it dealt with individuals who were over 61. This is at a point in time in which the risk of cigarette smoking in terms of increasing coronary mortality has largely passed. These risks are primarily directed toward moving the coronary mortality toward the young person.

For example, in women under 50 cigarette smoking increases the risk of myocardial infarction. In one study it is shown to be increased by sevenfold. If they also receive oral contraceptives, the risk goes up to twentyfold; again young women.

In males under 45 cigarette smoking is the dominant risk factor for mortality from myocardial infarction. Dahl and Hjert have estimated a fifteenfold increase in risk in this age group.

If one takes the age of myocardial infarction and sudden death and looks at the contribution from cigarette smoking, this is highest in the thirties and declines later in life, so that cigarette smoking is pushing these coronary complications to a young age, an age in life that is a productive age, an age in which men and women have family responsibilities, and is not one that comes largely in the elderly, to whom this particular study is addressed.

Dealing with individuals who are in these younger age groups, there have been eight major prospective studies documenting 12 million person years of experience in the United States, in Great Britain, in Canada, and in Sweden. Coronary disease mortality has been shown to be increased by cigarette smoking in all of these studies addressing males in midlife. Smoking is an independent risk factor, and it further increases the risk from coronary mortality in individuals who have hypertension and who have hypercholesterolemia. It can act alone but it greatly increases the risk to individuals who carry additional risk factors, whatever they be, and they could indeed, to some extent, be hormonal in the elderly.

I think it is fair to say that since the initial studies linking cigarette smoking to myocardial infarction, the power of the evidence has increased in strength and the evidence has been further elaborated, particularly as it deals with the incidence of myocardial infarction in midlife.

Senator East. I appreciate your comment, Dr. Oates.

I would like to request that the committee staff be directed to contact Dr. Castelli, who made a study. Again, it looks extremely responsible. It is in the New York Times. They could see if he would be willing to give us a written report on this study, so that we could consider it along with the others.

My whole point is, with all respect to Dr. Oates, to suggest the division of scientific opinion on many of these things and the relative merit of a particular causation factor.

Now, as a layman, I am not in a position to argue against what you are saying. What you are showing is the ability of a man, who obviously is very knowledgeable on this, to begin to sort and refine even further. You may be correct, but clearly the implication, at least in the New York Times, of this article is that this may be a
new horizon that would dwarf the others. Again, I repeat, it may
not be.

However, I am back to my concern about whether we do not
overreact in isolating this variable of smoking and focusing upon it
as well nigh the exclusive causation element here, when in fact it
is a part of a whole mosaic of variables which even within a specif-
ic context could vary significantly.

Obviously, this would imply an infinite potential of other factors
to be weighed.

I do not wish to belabor the point, and the chairman has been
overly generous and kind to me already. I would like to make that
request of the committee staff.

The CHAIRMAN. We will be happy to do that.

[The information referred to follows:]}
Sex Hormone Linked To Heart Disease, New Study Finds

Estrogens correlated with higher incidence

By LAWRENCE K. ALTMAN

A NEW study with major implications has found evidence that men who have suffered heart attacks, the nation's leading killer, have more of a hormone called estradiol than men without heart disease.

The new report from Columbia University, the National Institutes of Health and the Framingham Heart Study surprised even its authors. The research found no evidence that such commonly suspected risk factors as cholesterol, high density lipoproteins, high blood pressure and cigarette smoking differed among those who did and did not have heart attacks.

These findings suggest that heart disease could be primarily a hormonal disorder, which, if true, would affect treatment and prevention.

An extra supply of estradiol — one of the female sex hormones known as estrogens that normally develop in men as well as women though in less amounts — correlated with the incidence of heart attacks in the study in the May issue of The American Journal of Medicine.

The new study examined 112 men ranging in age from 21 to 63. Their average age was 45, thereby extending earlier findings made among younger men, aged 34 to 43, by Dr. Gerald E. Phillips of St. Luke's-Roosevelt Hospital Center and Columbia.

"Of the 15 subjects with the highest estradiol levels, 13 had coronary heart disease, whereas of the 15 subjects with the highest cholesterol levels, only three had coronary heart disease," Dr. Phillips, the chief author of the study, said in an interview.

Doctors do not know what causes the increased levels of estradiol, nor do they know how to prevent it or how to treat it. It may turn out that the higher estradiol levels stem from environmental factors, such as diet, genetic factors or both, Dr. Phillips said.

For example, it may be a result of a hereditary biochemical abnormality in the natural process by which the body converts testosterone, the male sex hormone, to estradiol.

Dr. William P. Castelli, who heads the Framingham Heart Study, said the new finding "adds to our knowledge but it will take some doing to figure out the mechanism." He added, "It raises many more questions than it answers, but it will encour..."
Hormones and Heart Disease

Continued from Page 31

eve to start looking in other directions.

The incidence of fatal heart attacks has dropped dramatically in recent years. Why this has happened has not been determined. Nor, for that matter, has the basic cause of heart attacks. Many researchers have focused attention on the amount of cholesterol and fats people eat. But others, such as Dr. John McMican, one of Britain's leading cardiologists, have challenged the idea that changing diets, reducing the fats and cholesterol, reduces the incidence of heart attacks.

"An immense effort has been devoted to the reduction of cholesterol levels in the blood by diet and by drugs," he said. "In most cases I have concluded that these efforts have had no detectable influence on the course of development of coronary heart disease."

Dr. McMican proposed that researchers have neglected Dr. McMican's theory.

It was only in 1962 that Dr. McMican started to look for alternate causes. He noticed that heart attacks were more common in men than in women, and that men were more likely to have heart attacks than women who were menopausal. He also noticed that heart attacks were more common in men who had a history of heart disease in their family than in men who did not.

Dr. McMican's theory is that heart attacks are caused by a lack of hormones, specifically estrogen. He believes that estrogen helps to prevent heart attacks by reducing the amount of cholesterol in the blood and by reducing the amount of fat in the arteries.

Dr. McMican has conducted several studies to support his theory. In one study, he found that women who had had a hysterectomy had a higher risk of heart attack than women who had not. He also found that women who used estrogen replacement therapy had a lower risk of heart attack than women who did not.

Dr. McMican believes that estrogen replacement therapy can help to prevent heart attacks. He recommends that women who are at risk of heart attack should be prescribed estrogen replacement therapy.

Dr. McMican's theory is supported by other researchers. Dr. Philip's recent study has also shown that estrogen replacement therapy can help to prevent heart attacks.

In conclusion, Dr. McMican's theory that heart attacks are caused by a lack of hormones, specifically estrogen, is supported by other researchers. Dr. Phillips's recent study has also shown that estrogen replacement therapy can help to prevent heart attacks. Therefore, it is important for women who are at risk of heart attack to be prescribed estrogen replacement therapy.
CURRENT CLIPS
from the printed media

Office of Communications / 834-51/2810/495-4253

MAY 3, 1983 A.M.

SEX BISEXUALS LINKED TO HEART DISEASE. NEW STUDY FINDS
Estrogens correlated with higher incidence

By Lawrence A. Altmann

A new study of older heart patients has found that those who have had recent heart attacks, the male patients' leading killer, have more of a hormone called estrogens than men with heart disease.

The new study started at Columbia University, the Long Island Jewish Hospital and the New York Presbyterian Medical Center. The research was based on evidence that certain hormones are known to affect the heart. Estrogens, for example, have been linked to the heart disease in women. But the study was designed to explore whether estrogens might also be a factor in men.

Theestrogens suggest that heart disease could be a specific hormone disorder, or even, with other treatment procedures.

The study examined 125 men ranging in age from 65 to 90. About 70 had recent heart attacks, 35 had coronary heart disease, and 20 had normal hearts. The study was conducted in the laboratory of Dr. Philip Robinson of Columbia University.

Dr. Robinson found that the highest estrogens levels, and not coronary heart disease, were found in the men with recent heart attacks. The study showed that those who had coronary heart disease, only those who had coronary heart disease, were the patients who showed high levels of estrogens.

Doctors do not know what causes the increased levels of estrogens, but they have been able to show that it is linked to the disease. In the study, the men with the highest estrogens levels were found to have more heart attacks and to be at greater risk of developing heart disease.

Dr. Lawrence W. Costello of the University of California, San Diego, who directed the study, said in a news release:

"We have found that the incidence of heart attacks is highest in the men with the highest estrogens levels. This suggests that estrogens may be involved in the development of heart disease."

The study was supported by the National Institutes of Health and the American Heart Association.

Source: Current Affairs

PHILADELPHIA'S PENNSYLVANIA HOSPITAL REPORTS
SEX BISEXUALS LINKED TO HEART DISEASE. NEW STUDY FINDS
Estrogens correlated with higher incidence

By Lawrence A. Altmann

A new study of older heart patients has found that those who have had recent heart attacks, the male patients' leading killer, have more of a hormone called estrogens than men with heart disease.

The new study started at Columbia University, the Long Island Jewish Hospital and the New York Presbyterian Medical Center. The research was based on evidence that certain hormones are known to affect the heart. Estrogens, for example, have been linked to the heart disease in women. But the study was designed to explore whether estrogens might also be a factor in men.

Theestrogens suggest that heart disease could be a specific hormone disorder, or even, with other treatment procedures.

The study examined 125 men ranging in age from 65 to 90. About 70 had recent heart attacks, 35 had coronary heart disease, and 20 had normal hearts. The study was conducted in the laboratory of Dr. Philip Robinson of Columbia University.

Dr. Robinson found that the highest estrogens levels, and not coronary heart disease, were found in the men with recent heart attacks. The study showed that those who had coronary heart disease, only those who had coronary heart disease, were the patients who showed high levels of estrogens.

Doctors do not know what causes the increased levels of estrogens, but they have been able to show that it is linked to the disease. In the study, the men with the highest estrogens levels were found to have more heart attacks and to be at greater risk of developing heart disease.

Dr. Lawrence W. Costello of the University of California, San Diego, who directed the study, said in a news release:

"We have found that the incidence of heart attacks is highest in the men with the highest estrogens levels. This suggests that estrogens may be involved in the development of heart disease."

The study was supported by the National Institutes of Health and the American Heart Association.
Association of Hyperestrogenemia and Coronary Heart Disease in Men in the Framingham Cohort

GERALD B. PHILLIPS, M.D.
New York, New York
WILLIAM P. CASTELLI, M.D.
Framingham, Massachusetts
ROBERT D. ABBOTT, PH.D.
Bethesda, Maryland
PATRICIA M. MACAMARA, A.B.
Framingham, Massachusetts

The serum levels of estradiol and testosterone as well as established risk factors for coronary heart disease were estimated in 61 men (mean age 70.0 ± 6.4 [SD] years) with coronary heart disease and in 61 matched control subjects enrolled in the Framingham Heart Study. The mean serum estradiol level was significantly higher in the subjects with coronary disease (p = 0.015). This difference in estradiol level increased with the exclusion of subjects older than 75 years (p < 0.001). The mean serum testosterone level was not significantly different. None of the established risk factors for coronary heart disease was different between subjects with coronary disease and control subjects except blood glucose level, which was higher in the subjects with coronary disease (p = 0.025). We conclude that hyperestrogenemia is an important correlate of coronary heart disease in men.

An increase in the mean serum concentrations of estradiol and estrogens in a group of men aged 54 to 63 years who had had a myocardial infarction has been previously reported [1]. Evidence that clinical signs of feminization preceded the myocardial infarction [1] suggested that the hyperestrogenemia may also have preceded the myocardial infarction. Further analysis of the data in the patients and control subjects revealed a correlation between the ratio of the serum concentrations of estradiol to testosterone and the areas under the glucose and insulin curves, respectively, in the glucose tolerance test [2]. This correlation along with other evidence suggested that these and perhaps other risk factors for myocardial infarction might be secondary to an increase in the serum estradiol-to-testosterone ratio or some closely related function [3]. It was further hypothesized that hyperestrogenemia might be the major predisposing factor for myocardial infarction and that the glucose-insulin-lipid defects and hypertension may be of secondary importance or even incidental to the development of myocardial infarction [2,3]. In order to evaluate these hypotheses, a matched-pair study was carried out in which the serum estradiol and testosterone levels, as well as established risk factors for coronary heart disease, were measured in a sample of male participants in the Framingham Heart Study. The results of this study, which was carried out in an older, larger, and less selected population than in the previous study [1], and in which the hormones were measured without knowledge of the diagnosis, are consistent with the hypotheses.

SUBJECTS AND METHODS

From the Department of Medicine, Columbia University College of Physicians and Surgeons and St. Luke's-Roosevelt Hospital Center, New York, New York; the Framingham Heart Study, National Heart, Lung, and Blood Institute, Framingham, Massachusetts; and the Biomatrix Research Branch, National Heart, Lung, and Blood Institute, Bethesda, Maryland. This work was supported in part by Grant NHL 28644 from National Institutes of Health, U.S. Public Health Service. Requests for reprints should be addressed to Dr. Gerald B. Phillips, Parnassus Hospital, 436 West Glens Drive, New York, New York 10025. Manuscript accepted March 14, 1983.

May 1983 The American Journal of Medicine Volume 74 283
Subjects enrolled in the study were aged 30 to 62 years at the first examination, occurring between 1948 and 1962. Sampling procedures and analytical techniques have been described previously [1].

The present study was carried out during the 19th examination of the Framingham Heart Study. The participants included in the analysis consisted of 181 males with a Coronary Heart Disease diagnosis, which included myocardial infarction, coronary insufficiency (OPE), or angina pectoris (15 patients), and 181 females with the same diagnosis (15 patients), with a Coronary Heart Disease diagnosis of 41% in the male group and 53% in the female group.

All cardiovascular events were confirmed by two physicians using the Duke criteria for diagnosis of acute myocardial infarction. The data included in the analysis were those that met the criteria established by the investigators.

Various blood samples were drawn from the subjects at random times in the morning, and the serum was separated and stored at -20°C. The serum samples were drawn and coded in Framingham, were sent to the investigator in New York City (G.W.B.) for hormone analysis. The results of the estradiol and testosterone measurements were related to the age and menopause status before the study of the subjects with coronary disease was revealed to the investigator. Although unknown to him, about two months after the study, the number of samples from subjects with coronary disease and control subjects were analyzed simultaneously in each assay. Measurement of the levels of adrenal hormones [2], insulin-like hormone [7], and follicle-stimulating hormone were carried out. The results were analyzed using the U.S. National Institute of Health ERD (endocrine research department) methods for the measurement of 17β-estradiol and testosterone were measured in 61 and 62 consecutive serum samples (CHEAS) in 50 matched pairs. The serum samples were analyzed using the ERD method [7], which is a standard technique for the measurement of estradiol and testosterone.

The results were analyzed using the U.S. National Institute of Health ERD (endocrine research department) methods for the measurement of 17β-estradiol and testosterone were measured in 61 and 62 consecutive serum samples (CHEAS) in 50 matched pairs. The serum samples were analyzed using the ERD method [7], which is a standard technique for the measurement of estradiol and testosterone.

The results were analyzed using the U.S. National Institute of Health ERD (endocrine research department) methods for the measurement of 17β-estradiol and testosterone were measured in 61 and 62 consecutive serum samples (CHEAS) in 50 matched pairs. The serum samples were analyzed using the ERD method [7], which is a standard technique for the measurement of estradiol and testosterone.

The results were analyzed using the U.S. National Institute of Health ERD (endocrine research department) methods for the measurement of 17β-estradiol and testosterone were measured in 61 and 62 consecutive serum samples (CHEAS) in 50 matched pairs. The serum samples were analyzed using the ERD method [7], which is a standard technique for the measurement of estradiol and testosterone.

The results were analyzed using the U.S. National Institute of Health ERD (endocrine research department) methods for the measurement of 17β-estradiol and testosterone were measured in 61 and 62 consecutive serum samples (CHEAS) in 50 matched pairs. The serum samples were analyzed using the ERD method [7], which is a standard technique for the measurement of estradiol and testosterone.

The results were analyzed using the U.S. National Institute of Health ERD (endocrine research department) methods for the measurement of 17β-estradiol and testosterone were measured in 61 and 62 consecutive serum samples (CHEAS) in 50 matched pairs. The serum samples were analyzed using the ERD method [7], which is a standard technique for the measurement of estradiol and testosterone.

The results were analyzed using the U.S. National Institute of Health ERD (endocrine research department) methods for the measurement of 17β-estradiol and testosterone were measured in 61 and 62 consecutive serum samples (CHEAS) in 50 matched pairs. The serum samples were analyzed using the ERD method [7], which is a standard technique for the measurement of estradiol and testosterone.

The results were analyzed using the U.S. National Institute of Health ERD (endocrine research department) methods for the measurement of 17β-estradiol and testosterone were measured in 61 and 62 consecutive serum samples (CHEAS) in 50 matched pairs. The serum samples were analyzed using the ERD method [7], which is a standard technique for the measurement of estradiol and testosterone.

The results were analyzed using the U.S. National Institute of Health ERD (endocrine research department) methods for the measurement of 17β-estradiol and testosterone were measured in 61 and 62 consecutive serum samples (CHEAS) in 50 matched pairs. The serum samples were analyzed using the ERD method [7], which is a standard technique for the measurement of estradiol and testosterone.

The results were analyzed using the U.S. National Institute of Health ERD (endocrine research department) methods for the measurement of 17β-estradiol and testosterone were measured in 61 and 62 consecutive serum samples (CHEAS) in 50 matched pairs. The serum samples were analyzed using the ERD method [7], which is a standard technique for the measurement of estradiol and testosterone.
TABLE I  Risk Factor Data for Subjects with Coronary Disease and Control Subjects*

<table>
<thead>
<tr>
<th></th>
<th>Control Subjects</th>
<th>Subjects with Coronary Disease</th>
<th></th>
<th>Control Subjects</th>
<th>Subjects with Coronary Disease</th>
<th></th>
<th>Control Subjects</th>
<th>Subjects with Coronary Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>50.0 ± 9.9</td>
<td>50.0 ± 9.9</td>
<td>50.0 ± 9.9</td>
<td>50.0 ± 9.9</td>
<td>47.2 ± 4.1</td>
<td>47.2 ± 4.1</td>
<td>47.2 ± 4.1</td>
<td>47.2 ± 4.1</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>139 ± 20.7</td>
<td>142 ± 22.2</td>
<td>138 ± 25.1</td>
<td>143 ± 22.8</td>
<td>139 ± 22.8</td>
<td>139 ± 22.8</td>
<td>139 ± 22.8</td>
<td>139 ± 22.8</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>216 ± 41.8</td>
<td>228 ± 43.6</td>
<td>224 ± 41.5</td>
<td>219 ± 44.9</td>
<td>219 ± 44.9</td>
<td>219 ± 44.9</td>
<td>219 ± 44.9</td>
<td>219 ± 44.9</td>
</tr>
<tr>
<td>Cigarettes (per day)</td>
<td>6.3 ± 17.2</td>
<td>7.2 ± 16.2</td>
<td>7.1 ± 15.9</td>
<td>7.0 ± 14.5</td>
<td>6.7 ± 11.9</td>
<td>6.7 ± 11.9</td>
<td>6.7 ± 11.9</td>
<td>6.7 ± 11.9</td>
</tr>
<tr>
<td>Ethanol intake (g/d)</td>
<td>7.7 ± 3.7</td>
<td>8.2 ± 7.9</td>
<td>8.1 ± 7.6</td>
<td>8.0 ± 6.9</td>
<td>7.9 ± 6.8</td>
<td>7.9 ± 6.8</td>
<td>7.9 ± 6.8</td>
<td>7.9 ± 6.8</td>
</tr>
<tr>
<td>Blood glucose (mg/dL)</td>
<td>117 ± 20.1</td>
<td>123 ± 25.0</td>
<td>121 ± 23.9</td>
<td>123 ± 24.9</td>
<td>123 ± 24.9</td>
<td>123 ± 24.9</td>
<td>123 ± 24.9</td>
<td>123 ± 24.9</td>
</tr>
<tr>
<td>Quotient's index (g/m²)</td>
<td>27.7 ± 4.0</td>
<td>27.7 ± 4.0</td>
<td>27.7 ± 4.0</td>
<td>27.7 ± 4.0</td>
<td>27.7 ± 4.0</td>
<td>27.7 ± 4.0</td>
<td>27.7 ± 4.0</td>
<td>27.7 ± 4.0</td>
</tr>
<tr>
<td>Alcohol intake (g/day)</td>
<td>0.6 ± 0.6</td>
<td>0.7 ± 0.8</td>
<td>0.7 ± 0.7</td>
<td>0.7 ± 0.8</td>
<td>0.7 ± 0.8</td>
<td>0.7 ± 0.8</td>
<td>0.7 ± 0.8</td>
<td>0.7 ± 0.8</td>
</tr>
</tbody>
</table>

* Based on 81 subjects with coronary disease and 81 control subjects. Values are means ± standard deviation.

Excludes subjects with smoking only and those with coronary disease and 49 controls.

Excludes subjects with coronary disease older than 75 years and their matched control subjects, leaving 48 subjects with coronary disease and 48 control subjects significantly different from matched controls by paired t test. * p < 0.01, ** p < 0.001.
Table 2: Mean Hormone Levels for Subjects with Coronary Disease and Control Subjects

<table>
<thead>
<tr>
<th></th>
<th>Coronary Disease</th>
<th>Control Subjects</th>
<th>Coronary Disease</th>
<th>Control Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>32.75 ± 7.64</td>
<td>38.54 ± 9.37</td>
<td>32.64 ± 7.49</td>
<td>37.60 ± 10.18</td>
</tr>
<tr>
<td>Testosterone (ng/dL)</td>
<td>3.80 ± 1.23</td>
<td>4.13 ± 1.45</td>
<td>3.98 ± 1.24</td>
<td>4.20 ± 1.58</td>
</tr>
<tr>
<td>Estradiol-to-testosterone ratio</td>
<td>3.95 ± 2.70</td>
<td>4.65 ± 3.24</td>
<td>4.97 ± 2.12</td>
<td>6.00 ± 4.06</td>
</tr>
<tr>
<td>Age 75 Years or Less</td>
<td>32.05 ± 7.13</td>
<td>37.61 ± 10.43</td>
<td>32.43 ± 7.10</td>
<td>38.53 ± 10.18</td>
</tr>
<tr>
<td>Testosterone (ng/dL)</td>
<td>3.88 ± 1.17</td>
<td>4.26 ± 1.41</td>
<td>3.96 ± 1.15</td>
<td>4.48 ± 1.42</td>
</tr>
<tr>
<td>Estradiol-to-testosterone ratio</td>
<td>3.90 ± 2.85</td>
<td>4.52 ± 3.26</td>
<td>4.44 ± 2.26</td>
<td>5.95 ± 4.13</td>
</tr>
<tr>
<td>Age 75 Years or Less</td>
<td>36.09 ± 8.24</td>
<td>31.96 ± 8.24</td>
<td>34.48 ± 8.26</td>
<td>35.05 ± 10.08</td>
</tr>
<tr>
<td>Testosterone (ng/dL)</td>
<td>4.04 ± 1.48</td>
<td>3.28 ± 0.99</td>
<td>4.02 ± 1.28</td>
<td>3.66 ± 1.28</td>
</tr>
<tr>
<td>Estradiol-to-testosterone ratio</td>
<td>9.25 ± 2.19</td>
<td>10.15 ± 2.76</td>
<td>9.25 ± 2.44</td>
<td>9.75 ± 2.90</td>
</tr>
</tbody>
</table>

Table 3: Correlation Coefficients between Hormone Levels and Risk Factors for Coronary Heart Disease

<table>
<thead>
<tr>
<th>Correlated Variables</th>
<th>Subjects with Coronary Disease</th>
<th>Subjects with Control Disease</th>
<th>NS Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (bpm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High-density lipoprotein cholesterol (mg/dL)</td>
<td>0.27*</td>
<td>0.23</td>
<td>-0.10</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>0.02</td>
<td>0.08</td>
<td>-0.18</td>
</tr>
<tr>
<td>Blood glucose (mg/dL)</td>
<td>-0.02</td>
<td>-0.24</td>
<td>-0.15</td>
</tr>
</tbody>
</table>

*Significantly different from zero: p < 0.01.

Table 4: Mean Hormone Levels for Subjects with Coronary Disease and Control Subjects

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Coronary Disease</th>
<th>Control Subjects</th>
<th>Coronary Disease</th>
<th>Control Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estradiol (pg/mL)</td>
<td>32.75 ± 7.64</td>
<td>38.54 ± 9.37</td>
<td>32.64 ± 7.49</td>
<td>37.60 ± 10.18</td>
</tr>
<tr>
<td>Testosterone (ng/dL)</td>
<td>3.80 ± 1.23</td>
<td>4.13 ± 1.45</td>
<td>3.98 ± 1.24</td>
<td>4.20 ± 1.58</td>
</tr>
<tr>
<td>Estradiol-to-testosterone ratio</td>
<td>3.95 ± 2.70</td>
<td>4.65 ± 3.24</td>
<td>4.97 ± 2.12</td>
<td>6.00 ± 4.06</td>
</tr>
<tr>
<td>Age 75 Years or Less</td>
<td>32.05 ± 7.13</td>
<td>37.61 ± 10.43</td>
<td>32.43 ± 7.10</td>
<td>38.53 ± 10.18</td>
</tr>
<tr>
<td>Testosterone (ng/dL)</td>
<td>3.88 ± 1.17</td>
<td>4.26 ± 1.41</td>
<td>3.96 ± 1.15</td>
<td>4.48 ± 1.42</td>
</tr>
<tr>
<td>Estradiol-to-testosterone ratio</td>
<td>3.90 ± 2.85</td>
<td>4.52 ± 3.26</td>
<td>4.44 ± 2.26</td>
<td>5.95 ± 4.13</td>
</tr>
<tr>
<td>Age 75 Years or Less</td>
<td>36.09 ± 8.24</td>
<td>31.96 ± 8.24</td>
<td>34.48 ± 8.26</td>
<td>35.05 ± 10.08</td>
</tr>
<tr>
<td>Testosterone (ng/dL)</td>
<td>4.04 ± 1.48</td>
<td>3.28 ± 0.99</td>
<td>4.02 ± 1.28</td>
<td>3.66 ± 1.28</td>
</tr>
<tr>
<td>Estradiol-to-testosterone ratio</td>
<td>9.25 ± 2.19</td>
<td>10.15 ± 2.76</td>
<td>9.25 ± 2.44</td>
<td>9.75 ± 2.90</td>
</tr>
</tbody>
</table>

Significantly different from zero: p < 0.01.
HYPERTENSION AND CORONARY DISEASE·PHILLIPS ET AL

afternoon. In the previous study [1], the subjects with coronary disease were men aged 34 to 64 years, were selected not only by a diagnosis of myocardial infarction, but also to exclude other disorders, drug intake, and alcoholism, and had samples obtained in the morning while fasting. Despite these differences, the results of the two studies are similar. The mean serum estradiol level was significantly higher in the subjects with coronary disease, whereas neither the estrone level nor the estradiol-to-estrone ratio was significantly different in the subjects with coronary disease and control subjects. The estrone level in serum estradiol level was persistent when the analysis was controlled for risk factors or coronary heart disease. The same results in two groups at the different ends of the age spectrum for coronary heart disease and control subjects support the validity of the finding of hyperestrinemia in men with a history of coronary heart disease and suggest its importance.

In the previous study [2], furthermore, the area under the ROC curve curve in the glucose tolerance test correlated with the serum estradiol level but not with the estrone level. In the present study, serum estradiol levels were observed between a single, random blood glucose determination from a sample obtained in the afternoon and the serum estradiol and estrone levels and the estradiol-to-estrone ratio. The relationships persisted after analysis controlled for age and obesity. Factors known to affect the blood glucose level [11, 12]. Thus, as both studies, the estradiol level, but not the estradiol-to-estrone ratio, correlated with coronary heart disease, whereas the estrone level did not correlate with blood glucose. The same results in the two studies support the hypothesis that an elevation in the estradiol-to-estrone ratio leads to the glucosuria/lipid defect and an elevation in the estradiol level predisposes to coronary heart disease [2, 3]. The correlation between the estradiol level and the estradiol-to-estrone ratio might explain the association of risk factors such as high blood glucose levels, lipids, and coronary heart disease. If this is the case, an elevation in the estradiol level without an accompanying elevation in the estradiol-to-estrone ratio might predispose to coronary heart disease without risk factors. Conversely, an elevation in the estradiol-to-estrone ratio without an elevation in the estradiol level might lead to risk factors without coronary heart disease. Thus, the risk factors may not be required for or even contribute to the development of coronary heart disease, it is still possible. Allowing, that some closely related function of estradiol and testosterone other than the estradiol-to-estrone ratio, possibly including the levels of free estradiol and testosterone or of another hormone(s) such as dehydroepiandrosterone (4), might correlate both with coronary heart disease and risk factors.

In the previous study, the only established risk factor for coronary heart disease was that was significantly different between subjects with coronary disease and control subjects was the blood glucose level. Except for dehydroepiandrosterone, however, other risk factors, i.e., the levels of systolic blood pressure, high-density lipoprotein cholesterol, and cigarette smoking, were in the expected direction. That cholesterol was not in the expected direction is consistent with the observation that the cholesterol level is a major risk factor after age 50 [13, 4]. The elevation of smoking as a risk factor also diminishes with age [16]. The effect of systolic blood pressure [16] may have been masked in this group by the treatment some were receiving. The difference in the mean high-density lipoprotein cholesterol level between subjects with coronary disease and control subjects was 38 mg/dl (p < 0.01), a difference similar to that of 41 found in larger population over age 50 [17]. In addition, risk factors may have been affected by a change in patient habits after the diagnosis of coronary heart disease. The correlation between estradiol and high-density lipoprotein cholesterol levels of the control subjects in the present study was unexplained but could reflect a different response to this hormone in the subjects with coronary disease. Administration of estradiol to men does cause an increase in high-density lipoprotein levels [18], but this does not mean that an increase in the natural estradiol will have a similar effect. A correlation between estradiol and high-density lipoprotein cholesterol levels has been reported in women [19, 20], however, administration of testosterone to men has not been reported to have the same effect.

The results of measurements of serum estradiol and testosterone in men with coronary heart disease in other laboratories have been conflicting. Asai et al. [21] reported a correlation in estradiol levels in men with myocardial infarction or peripheral vascular disease, however, these investigators measured serum estradiol, growth hormone, luteinizing hormone, follicle-stimulating hormone, cortisol, total androstenedione, and total testosterone levels and found all of these hormones significantly elevated except for testosterone and total thyroxine, which they found significantly reduced. Pharrow, et al. [22] similarly measured a spectrum of hormones in men with acute myocardial infarction and reported no changes in serum estradiol, androstenedione, or total testosterone levels. These results in men are consistent with the results in women [21], however, they are different from the results in men with coronary heart disease. The results in women are also consistent with the results in men [21].

May 1992 The American Journal of Medicine Volume 72
of estradiol in these same patients, however, showed no significant difference from levels in control subjects [35]. Heller et al. [36] found no difference between the estradiol levels in men who had had a myocardial infarction and those in age-matched control subjects. Luna et al. [34] observed an elevation in serum estradiol levels of female patients within two weeks of a myocardial infarction and in men with both angiography and coronary artery disease or arteriography. Zamoff et al. [32] reported normal estradiol levels in men with a history of myocardial infarction and in men with coronary artery disease on arteriography without a history of infarction. However, their reported estradiol, DHEAS, and DHEAS levels were elevated in the former group, whereas those levels were normal in the latter. DHEAS levels of subjects with coronary disease and control subjects were not significantly different in the present study. Klatzer et al. [37] found a decrease in estradiol levels in men studied on each of the first three days after a myocardial infarction and again three to nine months later, as well as in men with unstable angina. The reason for the differences in results between laboratories is not clear.

The cause of the hyperestrogenemia is not known. That it was the result of an exogenous factor in this study was considered. It could not be attributed to obesity [35, 36] since Queener's index was controlled for in the analysis of covariance. One possibility is that the hyperestrogenemia in the subjects with coronary disease resulted from a change in diet intended to lower serum lipid levels. However, a change to this type of diet appears to lower serum estrogen levels [37]. Another possibility is that more subjects with coronary disease than control subjects may have been consuming drugs that raise the serum estradiol level. For example, some drugs have been reported to increase serum estradiol and decrease serum testosterone [38]. However, no pattern relating a particular drug to estradiol level could be discerned in this study. For example, of the subjects with the 15 highest estradiol levels, only two were taking a digitalis preparation. Serum testosterone, furthermore, was not decreased in the subjects with coronary disease. In the previous study, showing hyperestrogenemia in men who had had a myocardial infarction, none of the patients or control subjects was taking any drugs [1]. In two other studies reporting hyperestrogenemia in men with myocardial infarction, the elevation in serum estradiol levels could not be attributed to drugs [28, 31]. No other exogenous factor to account for the hyperestrogenemia could be identified.

The mechanism for the hyperestrogenemia is not known. It could not be explained by an elevation inlutestosterone since the serum testosterone and follicle-stimulating hormone levels are the subjects with coronary disease and control subjects were not significantly different in these and other studies [27, 32]. More than half of the serum estradiol produced in men appears to come from the peripheral conversion of serum testosterone [39]. This relationship probably accounts for the high degree of correlation observed between estradiol and testosterone in subjects with coronary disease and control subjects, compared to (r = 0.54, p < 0.001) or separately (p < 0.001). Of interest is that in this and the previous study [1], as well as in the reports of others [28, 30–32], the testosterone level of the subjects with coronary disease was higher than that of the control subjects, although the difference did not reach statistical significance in any of these studies. Thus it is possible that a significantly increased testosterone level might be found in patients with coronary heart disease. In another series of subjects who were studied and that part of the hyperestrogenemia in patients with coronary heart disease might be secondary to a hyperandrogenemia. No other study as well as the mechanism for the hyperestrogenemia were identified in this study.

Whether the hyperestrogenemia preceded the coronary heart disease is not known. Evidence of a significant association between myocardial infarction [1] and reports of hyperestrogenemia in men with angina [31, 32] and coronary artery disease [31] support this possibility. That administration of estrogen to men leads to myocardial infarction [40] suggests that hyperestrogenemia may also predispose to myocardial infarction. If this is the case, the highly significant association of hyperestrogenemia with coronary heart disease in men both in an earlier age group [1] and in this study as an older age group in which established risk factors become weak or nonexistent [12–15] would suggest the importance of hyperestrogenemia as a predisposing factor.

REFERENCES


194

23-5X 0-85–12
wheezing, coughing, and pneumothorax. 

Hypertension and Coronary Disease—PHILIPS ET AL.

188

1974

5 Scalid-C. Cardiac valve disease and cause following myocardi-

6 athinfarction and valve perforation. Framingham Study, 20-

7 year follow-up. Section 53. CSH Creative (publication N


9 1977

8 Aldrich CE, Morrow 3L, Carta R. Radiopaque anatomy of.

10 hearts by CE, Batson and protocol of immunohaema-

11 tology. N.Y. J. Med (October 1977) 591

11 Megryris J. A. The problem for human different pneumo-

12 pathy and high intensity pneumo-phonoryography. Cardio-

13 ity 1977: 10

14 Megryris AL. Radiopaque anatomy for human lung-examining


16 Aizenstark IP, Poljanik RA. Wilson et al. For control heart

17 disease rat. The importance of heart relationships among.

18 non-occlusive lesions in individuals of different ages. Prev Med

19 1982: 11: 131

19 Hughes JR, Wunderlein LW, Franselle A. A cation micro-

20 search for measurement of arterial diameter. Brit Heart

21 1978: 9: 253

21 Davisson MB. The effect of aging on coronary microcirculation:

22 a review of the English literature and a practical approach to

23 the diagnosis of dilatation disease in the elderly. Amer-

24 J Cardiol 1979: 23: 686

24 Atiyeh AV, J. Kauffman ML. Influence of weight change on

25 changes in atherosclerotic level. The Framingham Study. N

26 Engl J Med 1978: 30 1942

26 Goldhill JW, Young M, Tandy R. Systolic heart disease.

27 atheroma. Circulation 1980: 54 579

27 Gordon T, Cassalini HP, Wurtz MC, et al. High density lipo-

28 protein as a low risk factor. Circulation 1977: 54 707

28 Dohle, Patrick M. Mortality in relation to surviving 20 years.


30 in the men and women: the Framingham Study. Br Heart J 1977: 54 573


31 other factors in coronary heart disease. Circulation 1977: 54 737


32 smoking, smoking and drinking alcohol on serum lipid.


33 Horbury A, Attwood A, Thistle D. Sex hormones and high.

34 density lipoproteins in health. Amer J Med 1977: 54 421


35 hyperlipoproteinemia in azoemics and other males. Amer.

36 J Med 1977: 54 423


37 hyperlipoproteinemia in elderly men with atherosclerotic.

38 disease. Amer J Med 1979: 91 553

38 Quddus DH, Adnams R, Garelick SL, et al. Severe as-

39 creation of angina and cerebrovascular disease in pregnant.


40 Sugarbaker DM, Fleiss J, Lane J, et al. Severe hyper-

41 tension and abnormal serum hormone levels. JAMA 1975 215: 843

41 Luxury M, Norton R. Conversion of blood androgen.

42 to estrogen in normal adult men and women. J Clin Invest

42 1959: 49 219

Senator East: I realize we cannot resolve it, and I do not mean to engage in interminable argument about it, but I would appreciate it if you might try to get that study.

The CHAIRMAN. We will surely do that. We also will be happy to ask the doctor any questions that we have.

Dr. Oates. Senator, may I respond to that?

The CHAIRMAN. Go ahead, Dr. Oates, and then I will turn to you, Dr. Cahan.

Dr. Oates. I would encourage you to request a written opinion from Dr. William Castelli, who has been involved in this study. I think the committee will be greatly enlightened by his communication on that.

The CHAIRMAN. We will be glad to do that.

Dr. Oates. I appreciate your request in that regard, Senator East. I think that will enhance the information in the hands of the committee. I think the evidence is unequivocal.

I think that in terms of this being "the only rabbit in the race", it is important to recognize that no one is advertising hypertension, urging one to go out and have high blood pressure, whereas we have over a billion dollars in cigarette advertising encouraging people of all ages to smoke.

The issue we are addressing here is not one of trying to outlaw cigarettes. We are simply urging that the public be provided with information about their risk. I think this is taking place in other areas. It cannot take place in the area of cigarette smoking without the assistance of the Federal Government.

The CHAIRMAN. Dr. Cahan?

Dr. Cahan. Lest you think there is no advertising on television, Senator, I may remind you of the Virginia Slims tennis tournament. There is a tale that lies therein because last time I testified we thought it would be wise, in light of the tremendous increase in women's lung cancer, perhaps to get a woman athlete, and a tennis athlete—of course, they are very famous, they are probably the most dominant type of woman athlete right now. Therefore, I called a very good friend who is highly connected in women's professional tennis and said, "By the way, could you please see if there are any women who would be glad to testify?" He said, "Well, they are all off on the Virginia Slims tournament." I said, "Oh, my goodness." He said, "Well, I know how you feel, but look what they've done for women's tennis." This is the remark made by this man.

I think that underlines that this is not exactly a static area of no advertising on television.

Senator East. However, it certainly, Doctor, is a greatly diminished one as opposed, for example, to beer advertisements where they show the husky athletes in the bar and they have just come off a playing field and are ordering a beer. We do not have that kind of grossness involved here. It may be sponsorship of an event.

I know what you are saying. I do not mean to be argumentative about it, but I see a difference in quantity here, what is being done, that approaches a very fundamental qualitative difference in the kinds of advertising. One is very blatant appeal, clearly a titillating ad which suggests the big athletes drink beer.
The fact that cigarette companies may sponsor a particular tennis tournament—I find a difference in degree that approaches to a point of difference in kind. However, I realize that reasonable minds could differ over that.

Thank you, Mr. Chairman.

The CHAIRMAN. Thank you.

Senator Pell.

Senator Pell. Thank you, Mr. Chairman.

I would like, first, to express my own personal affection and regard for Dr. Cahan, whom I have known for some years.

I have several specific questions.

Why is it that cigarette smoking is harmful and cigar and pipe smoking is not considered harmful?

Dr. Cahan. Well, I think that is an old wife's tale, Senator. Cigars and pipe smoking are, indeed, quite harmful. The difference is that usually cigar smokers and pipe smokers do not do as much inhaling as cigarette smokers. Therefore, their cancer, when it does occur, would seem to be much more in a local area such as within the oral cavity, esophagus, pharynx, and so forth.

Indeed, there are plenty of instances where pulmonary cancers have arisen in cigar and pipe smokers directly attributable to that. Also, I may remind you that cigarettes are very easy to smoke all the way down. Pipes are constantly going out, as are cigars. Therefore, the tendency is not to smoke quite as much. However, in those who smoke heavily, we see quite a lot of mouth cancer in particular.

Senator Pell. In that picture of the lung, the so-called clean lung which was still full of "cook" can that be cleaned out if you went out to live in Colorado where the climate is clean as opposed to New York City or some other place?

Dr. Cahan. No, I do not think it can be cleaned out. It just is one of the things that happens in the course of a lifetime. I think it is quite understandable. We all have those particles, more or less to some degree, depending on your exposure to the city dust, and so forth and so on. They are not harmful. They are taken and absorbed by the body as something that is quite inert.

Senator Pell. Dr. Fisher was talking about other diseases being caused by cigarette smoking. I was struck by the fact, when I went to visit a friend in the cardiac section of the George Washington University Hospital yesterday, that some of the people who have just been operated on who are recuperating, go down to the room at the end of the corridor and light up a cigarette, which would indicate the habit must be very strong, indeed.

Dr. Fisher. The habit is strong. Of course, not always is the information about the risks entailed in the habit as well presented, as it might be.

You mentioned Colorado. Perhaps a good reason for moving to Colorado is that there are now several units in the University of Colorado Medical Center in Denver, where I visited a couple weeks ago that do not allow smoking, so I think this is progressing.

Senator Pell. It has often been said that people should be allowed to kill themselves any way they like—wear helmets or not wear helmets when they are on motorcycles, wear safety belts, or
smoke cigarettes, but you come up against the fact that the taxpayer pays for their injuries in great part.

One device that has occurred to me—I do not know if any of you have ever thought about it—is to license the sale of tobacco or cigarettes. When anyone buys them, they would have to show a certificate saying, "I give up 10 percent of my medicare benefits for the future."

I was just curious, quite seriously, if that might have an inhibiting effect, if it were feasible. I thought that might amuse the chairman and might even stimulate him to incorporate an amendment in the bill.

The CHAIRMAN Senator Pell has always been very creative on this committee. [Laughter.]

Dr. Fisher, I do want to comment on that because I think it bears consideration.

Those sorts of measures often come up among those of us who are concerned about the issue, but I think reflection indicates that, as we "bleeding heart social scientists" refer to it, what you are proposing is a sort of "victim blaming," which we would rather stay away from.

The American Lung Association and our colleagues in the coalition have always been against a banning, a proscription, or a punitive approach to the individual who is addicted or habituated, or whatever you want to call it, to smoking.

We think that the proposal here for a comprehensive education and prevention act is much more in line with traditional American values, traditional ideas that people ought to be free to do what they please but they ought to have a lot of information by which to guide their actions. That is what we are interested in. That is the reason we support this bill.

Senator Pell. I know the tobacco industry objects strenuously to the labels on the packs. I can see that. It must be objectionable to them to pay for printing them and then have to print it, sort of like you do with iodine, have a skull and crossbones on it, or something of that sort.

Now this other approach of just having a certificate giving up a percentage of your potential medicare benefits would not be anything that the cigarette industry would have to do. Do you think that would be objectionable?

Dr. Fisher. I think people might feel that they were being unfairly singled out.

Once went to a meeting with some people who were hot about smoking. Just for example, I took exception to the fact that they were all drinking coffee out of styrofoam cups, spoiling our environment. Once we begin slashing at each other, singling out individuals and saying, "You are going to have to pay a $10 penalty for this," or "You are going to have to pay a $10 penalty," I think that begins to raise problems in terms of how we live together.

Our goal here is to encourage a good, educational, prevention approach.

Senator East. Would the gentleman yield?

Senator Pell. Certainly.

Senator East. Would you wish to extend that kind of provision to people, for example, who drank more than an established propor-
tion per day, for people who declined to use seat belts or people who were over a particular ideal weight or people who were excessive in their use of salt. What, again, I am asking for is some proportion and, balance here as to how far one could go in a federally-funded program for medical benefits singling out certain risk factors. Once we start down that road, I would simply remind the chairman there would be many other things we would want to add to that list. It might be an acceptable and appropriate approach, but I think Dr. Fisher suggested there are limits to how far Government can go in demanding certain behavioral forms. Certainly, the informational end of it is valuable. I am not arguing that.

I didn't know whether our distinguished Senator from Rhode Island would consider our adding more than merely a smoking certificate in terms of reducing medicare benefits.

Senator Pell. I think if it could be shown that perhaps a 25-percent increase in disease came from a particular illness or benefits were paid for a particular illness, an argument ought to be made along those lines. At this moment I am not making that argument. I am throwing this out as a suggestion.

Perhaps we ought to study it further and debate whether people who do not wear their seat belts would give up certain medical benefits.

The Chairman. I hate to say this, Senator Pell, but the Tobacco Institute might suddenly see that this particular bill is a much nicer bill than they thought originally.

Senator Pell. That thought occurred to me in making the suggestion.

The Chairman. That is what I figured.

Senator Pell. One final question: Near the Senate floor there is a little box of snuff that some of us take occasionally, made from tobacco. Is that also harmful or not?

Dr. Cahan. Snuff has its own problems. It is now lauded as a substitute for tobacco, but I assure you there is a fair amount of— I think it is called packing snuff, as I remember, in the South. A great deal of that is done. A great deal of oral cancer comes very much in those areas where they—

Senator Pell. I am not talking about chewing tobacco; I am talking about sniffing.

Dr. Cahan. You are talking about sniffing it? I do not know of any figures on that. I was thinking about slowing it away in the mouth the way good pitchers supposedly do all the time.

Senator Pell. I do not think many of my colleagues use that little box, which is just to the left as you come out the door.

Senator East. I was unaware of it, Senator.

Senator Pell. I would be glad to show it to you.

The Chairman. Dr. Fisher?

Dr. Fisher. I would like just to comment on two points.

Senator Pell, I am not expert in this particular area, but I know my colleagues in the Lung Association are increasingly concerned about the impact of smokeless tobacco, chewing as well as snuff. It is interesting to consider the fact that the snuff is efficient—it is not as efficient as inhaling cigarette smoke, but it is an efficient way of getting nicotine to the central nervous system. It is prob-
ably addictive, I hate to think of all of you Senators running around becoming junkies on snuff.

Also, there are increasing concerns because a lot of people, in response to knowledge of the risk of cigarette smoking, are turning to smokeless tobacco. Senator East, I am sure, is thinking we have soccer stars on TV talking about snuff and smokeless tobacco, etc.

We would be happy to furnish you some information on this, if you would desire it.

The other thing I did want to comment on was this. I really like your activist inclinations. I think perhaps a slight twist on what you were proposing perhaps gets more into the sorts of things that we can do, which would be to think in terms of positive incentives for people who live healthy lifestyles, whether it is using their seatbelts or controlling their weight, or whatever. If we can think, through health insurance and other sorts of mechanisms, of positive incentives for lifestyles—and a lot of the insurance companies are already doing this—I think legislation which encourages private sector forces to encourage health in their own ways would probably be quite feasible.

Senator Pell, I think you are right. I was remiss in closing that I did not acknowledge the good works that some of the cigarette companies are doing. I think Philip Morris, for example—I know they conduct the tennis tournaments in my home city. They do a lot in the arts and humanities. I think they use their profits, some of their profits, for very good causes, indeed.

The CHAIRMAN. Thank you, Senator Pell.

Let me ask you, Dr. Cahan. Your contributions, of course, are very well known in the field of lung cancer, and, as I have said, we have appreciated your testimony here today. In your experience, what other types of cancer are caused by cigarette smoking?

Dr. CAHAN. Cancer associated with the oral cavity, for one; cancer of the esophagus, usually in association with alcohol as co-carcinogens; cancer of the larynx, which we have mentioned, trachea; pancreas, you heard today, and the bladder, conceivably of the kidney. I think the pancreas is now becoming much more of a target than it used to be. Certainly the urinary bladder is.

The CHAIRMAN. In your experience, what is the typical course for a patient you diagnose as having lung cancer? What would be the typical course of treatment?

Dr. CAHAN. A patient dying of lung cancer?

The CHAIRMAN. Yes. What would he go through.

Dr. CAHAN. There are two things that happen. Either they die by local invasion within the chest cavity itself, invading important structures within the cavity, such as the heart sac or the windpipe or the bones. More frequently, it spreads beyond the thoracic cavity to the brain, to the liver, and to bone, and kills them that way.

The CHAIRMAN. I see.

Dr. Oates, your presentation, I think, has been enlightening to all of us here today. I want to thank you for it.

In your experience, do you find that the switch to low tar-and low nicotine cigarettes has lowered the risk for some of these diseases?
Dr. OATES. The data have addressed several aspects of that. Low tar, low nicotine cigarettes clearly do not reduce the risk for coronary-related events, myocardial infarction and sudden deaths. The reason for this is not entirely known, but there certainly is the issue of compensation by smokers to inhale more deeply and to smoke more in order to maintain the same levels of nicotine and other substances in the blood.

For whatever reason, there is no reduction in the risk for coronary mortality associated with low tar, low nicotine cigarettes. In the area of lung cancer, while the risk is less, it is still quite evident. The low tar, low nicotine cigarette is not safe.

With regard to the issue of how well the public is informed, it is of interest that of individuals surveyed regarding low tar, low nicotine cigarettes, 60 percent were not aware of the fact that low tar and low nicotine cigarettes are not safe. A tremendous educational effort is required which would be implemented by this bill.

The CHAIRMAN. You have said that cardiovascular diseases kill nearly 1 million Americans each year. How many of those would you attribute to being either caused in part or wholly by cigarette smoking?

Dr. OATES. I think it is difficult to be precise as to the exact number of those. We know that of those in total, about 550,000 are related to coronary events, myocardial infarction and sudden death. Cigarette smoking is a leading risk factor in that regard. I think that the general estimate is in the neighborhood of 200,000 of those deaths may be related to cigarette smoking. It is an enormous epidemic.

The CHAIRMAN. I see. How is a person's risk of heart disease diminished if they quit smoking even if they have smoked for a number of years?

Dr. OATES. Over a period of time after ceasing smoking the risk returns back close to that of nonsmokers. Exactly how this occurs is not understood, but it is hopeful for those individuals who choose to cease smoking. That is an attractive part of this bill, that it would inform individuals of the risk of heart attack.

The CHAIRMAN. Dr. Fisher, in your experience as a psychologist, what has been the most effective way in discouraging youngsters from entering into smoking?

Dr. FISHER. There have been a number of things that have been useful. What is very important is the effects of specific information. For instance, the project carried out by the New Hampshire Lung Association several years ago brought biofeedback equipment into the school room so that young students could see their hands shake more if they smoked a cigarette or see their pulse rate increase, see increased carbon monoxide in their breath. The ability for first-hand the short-term, very specific effects of smoking a cigarette was quite influential in changing attitudes toward smoking and, some evidence indicates, changing subsequent smoking behavior itself.

I think we can then look at that as being parallel to the provisions of this current bill. This is just one example with teenagers and smoking of the general principle that specific, concrete, real information is most helpful to people.
Additionally, we think the impact of people that children look up to is very, very important in this area. I was talking with some people just last evening about the fact that—I do not remember her name but one of the prominent female actresses was on the cover of Seventeen one month and was in a Virginia Slim's cigarette commercial shortly thereafter. This sort of linking of attractive role models with smoking is very important. That is one of the reasons the Lung Association sponsored the Brooks Shields poster several years ago. This is a woman that children look up to and see as an appropriate role model. Getting her to essentially say, “Hey, it can be cool not to smoke,” seems to be important.

Additionally, the consistency of information is important. Developmental psychology research indicates that children or adolescents are at an age where they pick apart all the information they get. If they can say, “Well, that is just those people in the Lung Association trying to act like my grandmother,” or if they think, “Well, of course your doctor tells you not to smoke. What do you expect from your doctor,” or if their schoolteacher tells them to sit in their seat and not to smoke, et cetera, then they can discredit the information and avoid its implications. If it is not presented to youngsters as consistent information, where the Government, the professional sector, the schools, TV personalities like Captain Kangaroo, more adult personalities like Brook Shields, et cetera, agree about what’s important, they may not take it in. However, if all of these people are together, then I think we have a chance of getting youngsters to see that this is really something they do not want to do.

That is why I think that the first provision of your bill is very, very important. I do not think it makes sense to say, “Well, the voluntary associations are already doing enough in this area.” Each of professional community, the voluntaries, as well as the Government have a very, very important role. The Government has a unique type of credibility which needs to be brought to bear in this issue more effectively than it currently is. I applaud the first provision for that reason.

The CHAIRMAN. From your experience as a psychologist, is there a smoker’s personality? Can you describe this personality? Do you think that S. 772 will actually help people to quit regardless of their peculiar personalities?

Dr. FISHER. I do not want to take you to a lecture, Senator, but I am a behaviorist. Our approach to psychology sort of eschews the study of personality. We find that not to be a most useful concept.

If I had to talk about personality of the smoker, I would say that the most striking aspect of the smoker’s personality is that on average 4 to 11 percent of smokers are less likely to know about the risks of health which their smoking constitutes from the non-smoker in our society. It is a reliable finding from the Federal Trade report that I mentioned earlier. That looks very familiar to a psychologist.

It is very common that people are not likely to want to find out about risks associated with behavior. They may not want to find out. They sometimes prefer not to know.

Getting through that sort of “defensiveness,” as psychologists call it, highlights again the importance of consistent information. If
the smoker can say, "Oh, that's those people, those hotheads about smoking, who are telling me that, but they would tell me anything to get me to quit," if they can discount the information source, they can discount the information. Again, it is important, therefore, that the information in informing the public in this area be broad based and include the Government as a very active participant.

The Chairman. Thank you.

I certainly feel that we have had interesting hearings today. We have all learned a lot about the effects of cigarette smoking and other types of smoking on the human being.

We appreciate the efforts that all of you have put forth.

With that, we will recess these hearings.

I might mention that we are going to have a full day of witnesses who may disagree with the conclusions of many of you who testified today. In any event, we will want to look at both sides of this issue.

We do appreciate the testimony you have given. I have no doubt in my mind that it is true.

With that, we will recess these hearings until the next session.

[Whereupon, at 1.30 p.m., the committee recessed, to reconvene at the call of the Chair.]
SMOKING PREVENTION HEALTH AND EDUCATION ACT OF 1983

THURSDAY, MAY 12, 1983

U.S. SENATE,
COMMITTEE ON LABOR AND HUMAN RESOURCES,
Washington, D.C.

The committee met, pursuant to recess, at 9:37 a.m., in room SD-430, Dirksen Senate Office Building, Senator Orrin Hatch (chairman) presiding.

Present: Senators Hatch, East, Dodd, Nickles, Quayle, and Humphrey.

OPENING STATEMENT OF SENATOR HATCH

The CHAIRMAN. I welcome you here today to these full committee hearings devoted to S. 772, the Smoking Prevention Health and Education Act. As chairman of the Labor and Human Resources Committee, I also held hearings 1 week ago on S. 772 and I learned a great deal about the health of our citizens. I learned that the health hazards of smoking are enormous. In fact, smoking has reportedly claimed more victims in this country than all the Americans killed in major wars and traffic accidents. These facts were presented by some of the most distinguished scientists in the field of medicine and affiliated specialties.

Similarly, the bill received impressive support from the American Cancer Society, American Heart Association, American Lung Association, and virtually every health-professional organization in the country. Likewise, we heard moving support from Mrs. Barney Clark who testified before this committee last week that her husband would probably still be alive if he had not been a pack-a-day smoker of cigarettes for 25 of his 62 years.

The legislation we are dealing with today represents a national public education effort designed to improve our citizens' awareness about what the Surgeon General has said is the No. 1 preventable cause of death—cigarette smoking. The issue at stake is not whether a person should or should not smoke, or whether the Government should tell people how to live their lives. I am not in favor of Government regulations just for the sake of regulating. The legislation we are considering today will not prevent any citizen from using tobacco products. It will, however, allow them to make a more informed choice.

Today we are here to listen to another panel of witnesses who have been invited to testify as representatives of the tobacco indu-
try and outdoor advertisers. I am very happy to welcome our witnesses today.

Our first panel consists of Mr. Curtis H. Judge, of Lorillard, chairman of the executive committee of the Tobacco Institute; Dr. Sheldon C. Sommers, who is a pathologist at Lenox Hill Hospital in New York; Dr. Edwin R. Fisher, director of laboratories at Shady-side Hospital in Pittsburgh, Pa.; Dr. Roger D. Blackwell, who is professor of marketing at the Ohio State University; and, finally, Dr. Theodore H. Blau, who holds a doctorate in psychology and is currently in independent practice of clinical psychology in Tampa, Fla.

We will begin with Mr. Curtis Judge.

Mr. Judge, please proceed.

STATEMENT OF CURTIS H. JUDGE, PRESIDENT OF LORILLARD AND CHAIRMAN OF THE EXECUTIVE COMMITTEE OF THE TOBACCO INSTITUTE, NEW YORK, N.Y., ACCOMPANIED BY ARTHUR J. STEVENS, GENERAL COUNSEL OF LORILLARD

Mr. Judge. Thank you very much, Mr. Chairman.

I am Curtis H. Judge, president of Lorillard, a division of Loews Corp. I am also chairman of the executive committee of the Tobacco Institute, an association of tobacco manufacturers with headquarters in Washington, D.C. I testify here today on behalf of our industry.

With me today on this panel are Prof. Roger D. Blackwell, Dr. Theodore H. Blau, Dr. Sheldon C. Sommers, and Dr. Edwin R. Fisher. I am also accompanied, on my left, by Arthur J. Stevens, general counsel of Lorillard.

While we appreciate the time you have provided for us to testify, Mr. Chairman, we want to point out, with all due respect, that additional time should also be provided to permit members of the committee to consider and deliberate on the testimony that we have been permitted to present.

The issues raised by this legislation are serious ones, and we believe—and others agree with us—that holding a hearing today and a markup tomorrow suggests an inordinate rush to judgment.

Before the present national policy regarding tobacco and the use of tobacco products is discarded, we would hope that this committee will not only hear a full presentation of the industry position, but also take a reasonable time to study the record and reflect on it.

My oral remarks will be confined to S. 772. However, my full statement will deal with the full range of legislative proposals that are reflected in S. 1113 and in H.R. 1824. I ask that my full written statement be printed in the record of these hearings.

Mr. Judge. Thank you, Mr. Chairman.

Senators, we are here to express our strongest opposition to S. 772. Despite the appearances of good intentions, this is bad legislation. It is, of course, bad for our business. More than that, it sets a
bad precedent for many other businesses. But most importantly, it is bad for our free society and its citizens.

We oppose this bill because of the fundamental issue it raises of whether this country will have a national policy of choice or of compulsion, of public information or of public conformity. The measure of effectiveness of S. 772 will not be the degree to which the public is aware of official warnings, but rather the extent of public compliance with them. The goal of S. 772 is not an increased level of awareness about tobacco and health, but a reduced level of smoking.

To ignore the present national policy, as this bill does, is unnecessary, unwise, and unfair. S. 772 is founded on the notion that people are informed if—and only if—they conform to the Government line and stop smoking. In fact, S. 772, in our judgment, is a not very subtle giant step down the road to prohibition.

The present policy of Congress and the purpose of the present law is to establish a Federal program whereby the public may be adequately informed that cigarette smoking may be hazardous to health.

We submit that this has already been accomplished. The evidence shows that over 90 percent of the American public is aware of the claim that smoking is harmful—an unprecedented level of awareness that has been lauded by a Secretary of HHS and by the Surgeon General. There is also widespread awareness of specific claims. More than 90 percent of the public, for example, is aware of the claimed association between smoking and heart disease. Equal percentages are reported for awareness of claims about smoking and lung cancer and smoking and pregnancy.

This high awareness is not recent nor limited to health professionals or adults. Nearly 7 years ago, the Gallup poll surveyed teenagers across the Nation. It found that "a huge majority of teenagers, 96 percent, believe smoking to be bad for one's health, while another 89 percent think that smoking is a cause of cancer, and 76 percent link smoking and heart disease."

On the basis of these findings, George Gallup concluded that "efforts to convince America's teenagers of the dangers of smoking apparently have been highly successful."

In spite of these facts, S. 772 "finds" that present Government and private efforts have been "insufficient" in conveying this information to the public.

Naturally, this legislation is aimed at the tobacco industry. But the precedent embodied in it could well be used against other industries—food, beer, wine, alcohol, confectionery, cosmetics are only a few of the obvious targets. The regulatory spirit that inspires this bill convinces me that the hit list will expand. Today, tobacco—tomorrow, who knows?

Let me detail the many ways that this bill inflicts its damage on our industry and our society.

One of the most insidious elements of the bill is found in the section on "findings." Resolving scientific questions by a majority vote of Congress is inappropriate and misguided, especially where complex scientific questions are involved. It freezes scientific research in its tracks. It stops the search for an answer, because Congress has decreed the answer. It has the potential for diverting funds
from the basic research needed to answer the questions of what causes many chronic diseases.

Mr. Chairman, I am not a scientist. But, as I understand the nature of science, it is not establishing one's own answer and then structuring research designed to prove that predetermined answer. Science is an evolving, changing process of discovering answers to scientific questions.

For example, public health officials have urged people to reduce their smoking, their high blood pressure, and their cholesterol levels in order to lower their risk of dying from heart disease. When this risk-factor-reduction hypothesis was put to the test by the Multiple Risk Factor Intervention Trial, or MR FIT as it's called, the results came as a surprise to the governmental scientific community. MR FIT had not proven out their carefully developed and often expounded hypothesis. Ten years, and $115 million tax dollars later, the question of coronary heart disease causation is still a question.

This giant project, sponsored by the National Institutes of Health and reported in the September 24, 1982, issue of the Journal of the American Medical Association had proved nothing. There was no significantly different reduction in heart disease death rate in the group that had significantly greater reductions in cholesterol, blood pressure, and smoking. It seems clear, that this Government research project casts grave doubt on the validity of one of the warning labels in S. 772 concerning smoking cessation, and emphasizes the need for continued research and open-mindedness.

As was mentioned at last week's hearing, The New York Times on May 3 reported on another study that also demonstrates the danger of legislating scientific findings. This new research, conducted by Columbia University, the National Institutes of Health, and the Framingham Heart Study, surprised even its authors. According to the New York Times, it found no evidence that such commonly suspected risk factors as cholesterol, high blood pressure, and cigarette smoking differed among those who did and did not have heart attacks. These researchers found that men who suffered heart attacks have more of a certain type of estrogen than men without heart disease.

"These findings," according to the Times, "suggest that heart disease could be primarily a hormonal disorder which, if true, would affect treatment and prevention." And I would add, Mr. Chairman, Government health policy and legislation as well.

There will be scientific testimony later with respect to these studies and the proposed findings of S. 772. In addition, many other expert witnesses will have available for the committee written statements on the scientific issues in the bill.

Turning to the section in the bill on "Smoking Research, Education, and Information," there is nothing in this provision that the Secretary of HHS cannot do now administratively. There is no authorization of funds in this legislation, but we are sure that the demand for dollars would swiftly follow its enactment.

Establishing a statutory Interagency Committee on Smoking and Health would establish an unnecessary and expensive antismoking bureaucracy and create another costly advisory committee, with the authority to coordinate, and thereby to control, research on
smoking and health at the National Institutes of Health and throughout the entire Federal Government. In fact, as Dr. Brandt made clear last week, HHS neither wants nor needs a statutory Interagency Committee on Smoking and Health.

One of the major innovations of the hill is to be found in section 6. Rotating a series of new warnings on packages is calculated to have a "shock effect," to deter smoking, and to force the industry to use its packages for Government-mandated antismoking campaign.

I must note at this point that while S. 772 contains four warnings, S. 1116 proposes four times that number. Can we anticipate that cigarette packages and advertisements are to become adjuncts to medical journals? Will a bill be proposed for a new warning label every time an article is published alleging an adverse health effect due to smoking?

It is interesting to note that in 1980, after a study, the Department of Health and Human Services opposed warning statements on alcoholic beverages. Indeed, HHS rejected a specific warning label for pregnant women because, it said, "singing out a specific health issue ... may cause consumers to discount the importance of other significant alcohol-related health hazards."

The Department further pointed out that rotating warning labels "would confuse the public" and "pose potentially difficult administrative and enforcement problems."

Finally, HHS expressed concern that "the public is becoming jaded over Government warnings" and that the Government should use such warnings with caution.

Senators, it is high time for these cogent concerns about health warnings to be applied evenhandedly. It is time to stop singling out tobacco and to stop using smoking as a scapegoat for all of the many factors associated with disease.

Testimony offered to both House and Senate committees during the 97th Congress clearly demonstrated that this proposal for rotating warnings is an attempt to solve a problem that doesn't exist by a method that doesn't work.

The problem does not exist because, as I pointed out earlier, virtually everyone is aware of the Government's claims about smoking and health. The contrary premise on which this bill is based is supported by nothing more than the tortured arguments contained in the 1981 FTC Staff Report on Cigarette Advertising, which, incidentally, the Commission has never adopted. In that report, the staff erroneously concluded that anyone who does not believe the official line on smoking and health must be unaware of the alleged risks.

At the conclusion of my statement, Professor Blackwell, who is an expert on consumer behavior, will testify in more detail concerning the flaws in the premises underlying the FTC staff report and S. 772. In addition, because the proponents of this bill have placed such emphasis on that report, I would like to submit for committee review and for the record the Tobacco Institute's comments on the FTC staff report, which were submitted to the FTC in December 1981. Chapter 1 of those comments rebuts both the reasoning and conclusions about awareness set forth in the report.
Due to its voluminous content and in the interest of economy, the report "Comments of the Tobacco Institute on the FTC Staff Report on the Cigarette Advertising Investigation," was retained in the files of the committee where it may be researched upon request.

Mr. Judge. The distinction between belief and awareness is as crucial as it is overlooked. The fact is that a poll of people’s beliefs about smoking and health says nothing about their awareness of claims that smoking is harmful. And the fact that people do not believe these claims does not mean that they are unaware of them, but rather that they are not prepared to conform to Government views on smoking and health. That result may frustrate those who want to prevent people from smoking, but it surely provides no support for the claim that people need to know more about the Government’s position on this issue.

There also is no reason to expect that the proposed rotating warning system will accomplish its sponsors’ goal. There is, in fact, ample experience to demonstrate that such a system doesn’t work.

Despite claims to the contrary, the fact is that in Sweden, the country that originated rotating labels, per capita cigarette consumption increased after the rotating warnings and a host of other antismoking measures went into effect.

Elsewhere in Europe there are signs that the idea of multiple warning statements is one whose time has come and gone. England, after several years of tinkering with three rotating warnings, has cut back to one general warning which is much like the statement required in this country today and little different from the statement England started out with in 1971.

There may be a better lesson for this committee in the experience of the British, a people whose traditions of government and individual freedom are more akin to ours than in that of the Swedish, whose Government mandates social engineering and behavioral modification.

Although rotating labeling has not worked where it has been tried, it will most certainly create a regulatory nightmare of confusion, conflict, and a host of logistical problems.

For example, the bill would require each of four warning statements to appear "a substantially similar number of times on each brand of cigarettes." Today there are more than 200 cigarette brand packings produced domestically. New brands are introduced and old ones withdrawn. In these circumstances, equalizing the appearance of four different warning statements on every brand would be a logistical impossibility.

The bill’s requirement for disclosure of tar, nicotine, and carbon monoxide levels on cigarette packages also is unnecessary and unjustified. The public is continually made aware of tar and nicotine levels, which the cigarette manufacturers have included in their advertising since 1971.

Disclosure of carbon monoxide levels would serve no legitimate purpose. There is not sufficient scientific evidence for the conclusion that exposure to carbon monoxide from cigarette smoking causes disease. There is also no generally accepted method of measuring carbon monoxide in cigarette smoke.
Many of the attacks on this industry are based on the false premise that cigarette advertising encourages young people and nonsmokers to smoke. The fact is that cigarette advertising is brand advertising, intended to encourage smokers to switch from competitive brands. In the past decade, the percentage of the population who smoke has declined, while during the same period the number of cigarette brands and brand styles has substantially increased. Cigarette manufacturers must advertise to gain or maintain share of current smokers for each of their competitive brands. Clearly, such advertising neither is designed for, nor has the effect of, increasing the number of people who smoke.

In light of these facts, it is not surprising that in testimony before the House Committee on Commerce in 1969, the then Chairman of the FTC acknowledged that cigarette advertising is brand advertising. And in his 1979 Report on Smoking and Health, the Surgeon General said the following:

As the cigarette industry has asserted, the major action of cigarette advertising now seems to be to shift brand preferences, to alter market shares for a particular brand.

The provisions of section 5 of the bill, requiring all companies to report "a complete list of each chemical additive used in the manufacture" of cigarettes to the Secretary of HHS, and the Secretary in turn to report that list to Congress, completely ignore the existing disclosure agreement worked out last year in good faith between the cigarette manufacturers and HHS, which agreement Secretary Brandt praised during his testimony in the House in March and which he called an "opportunity to learn a great deal," just last week.

Pursuant to that agreement, the manufacturers have made available to HHS, with suitable safeguards to protect this highly sensitive commercial information, a list of their commonly used ingredients and, as Dr. Brandt pointed out to this committee, those ingredients used in large quantities by any single manufacturer.

This industry's contribution to our Nation's economy is enormous, more than $57.6 billion to the 1980 gross national product, according to a study by the Wharton Applied Research Center at the University of Pennsylvania. This amount was 2.4 percent of the gross national product for that year. That contribution generated the equivalent of over 2 million jobs, and some $21 billion in taxes of all kinds to Federal, State, and local governments.

I know that I don't need to remind you, Senators, that since the Federal excise tax was doubled on January 1 of this year, tobacco's tax contribution is now even more.

In addition to being an economically strong segment of the private sector, this industry has also been a responsible member of our society. Since 1954, the industry has committed some $111 million for unrestricted, independent research seeking answers to the continuing questions regarding smoking and health. In fact, during the last 10 years, our contribution has been far greater than the combined expenditures for similar research by all the voluntary agencies who oppose smoking.

For almost 20 years, this industry has worked to avoid having its brand advertising reach young people. Most recently, we have con
ducted an advertising campaign which has already reached 122 million Americans. One of the ads asks, "Do cigarette companies want kids to smoke?" And it gives our answer, "No. As a matter of policy, no. As a matter of practice, no. As a matter of fact, no."

Two other ads also make the same point. I have the three ads here. I ask that they be made a part of the record.

The CHAIRMAN. Without objection, we will make them part of the record.

[The advertisements referred to follow:]
IS CIGARETTE ADVERTISING A MAJOR REASON WHY KIDS SMOKE? NO.

Advertising is consistently ranked among the least important factors influencing college students to start smoking according to a study by a professor of psychology who heads a prominent university research center.

That notion is typical. Because the fact is cigarette advertising is not designed to influence people to start smoking, kids or anybody else. Its objective is to promote broad identification and brand loyalty among people who already smoke.

So why do kids start smoking? In a recent study of teenage smoking habits in which 1500 students were interviewed, the...
most asked questions about cigarettes.

A recent survey conducted for a Government agency showed continuing declines among teenagers through 1981. We think that's good. Because we think kids should not smoke. Smoking is an adult decision based on culture and informed judgment.

If you'd like to know more about this and other issues, write for our free information kit, "Answers to the most asked questions about cigarettes," Addressthe Tobacco Institute, Suite 931, 1875 Eye St. NW, Washington, DC 20006.

We offer this information in the belief that full and free discussion of this important public issue is in the public interest.

WEIGH BOTH SIDES
BEFORE YOU TAKE SIDES.

The relative decreases among teen age smokers were 25 percent during 1974-79. Source: 1979 "Teenage Smoking" study by U.S. Department of Health and Human Services.

1974
1979
QUESTION

3

DOES CIGARETTE ADVERTISING CAUSE KIDS TO START SMOKING?
most asked questions about cigarettes.

Advertising certainly is not the culprit. That's how a Director of the U.S. Office on Smoking & Health answered the question.

If advertising doesn't cause kids to smoke, what does? There are no pat answers. Research by experts indicates peer pressure is important, as well as many other complex psychological, cultural, and socioeconomic factors. There may be as many reasons why as there are kids who do.

While cigarette industry sales are increasing, fewer teenagers are smoking. For example, the American Cancer Society reports that among young women, smoking has decreased 17 percent since 1984.

We think that's good because we don't think kids should smoke. Smoking is an adult custom based on mature and informed judgment. That's right, adults, not children.

If you'd like more information, write for our booklet, "Answers to the most asked questions about cigarettes." Address: The Tobacco Institute, Suite 231, 1875 Eye Street Northwest, Washington, DC 20006.

We offer it in the belief that full and free discussion of these important public issues is in the public interest.

And that in matters of adult social customs, the vast majority of law-minded Americans honor individual freedom of choice.

WEIGH BOTH SIDES
BEFORE YOU TAKE SIDES.
DO CIGARETTE COMPANIES WANT KIDS TO SMOKE?
most asked questions about cigarettes.

No As a matter of fact No As a matter of practice No As a matter of fact.

The unfortunate fact is that some kids do smoke. But while cigarette sales continue to increase, fewer teenagers are smoking. For example, according to the American Cancer Society, smoking among young women has decreased 17 percent since 1974.

All of us need a time of "growing up" to develop the mature judgment to do so many things like driving, raising a family, and knowing enough to make an informed decision about all sorts of adult activities.

In our view, smoking is an adult custom and the decision to smoke should be based on mature and informed individual freedom of choice.

For more information, write for our booklet: Answers to the most asked questions about cigarettes. Address The Tobacco Institute, Suite 920, 1875 Eye Street Northwest, Washington, D.C. 20006. We offer it in the belief that full and free discussion of these important public issues is in the public interest.

WEIGH BOTH SIDES BEFORE YOU TAKE SIDES.
Mr. Judge. These ads have appeared in such major publications as Time, Newsweek, U.S. News & World Report, Sports Illustrated, People, and TV Guide, to name a few.

Senators, I will conclude my statement by emphasizing some major points which should be considered by the committee in its deliberations on this bill.

Exercising their right of free choice, 53 million Americans continue to smoke while 30 million Americans are said to have quit smoking.

Virtually everyone is fully aware of the assertions about the hazards of smoking.

The decision to smoke or not is an informed choice to be made by individuals as a matter of right.

Therefore, more governmental regulation of freedom of choice by legislation is unwarranted, improper, and unnecessary.

Even though the question of smoking and health is an open question, it is also a highly emotional subject. Public policy, however, must be based on more than the passionate advocacy and the heated claims of those who oppose smoking.

We ask only that you consider all the facts and all the arguments before you cast aside the present law which is functioning so well in the interest of all Americans.

Thank you, Mr. Chairman.

[The prepared statement of Mr. Judge follows:]

---

**Mr. Judge**

These ads have appeared in such major publications as Time, Newsweek, U.S. News & World Report, Sports Illustrated, People, and TV Guide, to name a few.

Senators, I will conclude my statement by emphasizing some major points which should be considered by the committee in its deliberations on this bill.

Exercising their right of free choice, 53 million Americans continue to smoke while 30 million Americans are said to have quit smoking.

Virtually everyone is fully aware of the assertions about the hazards of smoking.

The decision to smoke or not is an informed choice to be made by individuals as a matter of right.

Therefore, more governmental regulation of freedom of choice by legislation is unwarranted, improper, and unnecessary.

Even though the question of smoking and health is an open question, it is also a highly emotional subject. Public policy, however, must be based on more than the passionate advocacy and the heated claims of those who oppose smoking.

We ask only that you consider all the facts and all the arguments before you cast aside the present law which is functioning so well in the interest of all Americans.

Thank you, Mr. Chairman.

[The prepared statement of Mr. Judge follows:]

---
STATEMENT OF CURTIS H. JUDGE
ON S. 112
BEFORE THE COMMITTEE ON LABOR & HUMAN RESOURCES

May 12, 1983

I am Curtis H. Judge, President of Lorillard, a division of Loewe Corporation.

I am also the Chairman of the Executive Committee of The Tobacco Institute, an association of tobacco manufacturers with headquarters in Washington, D.C. I testify here today on behalf of our industry.

With me today on this panel are Professor Roger D. Blackwell, Dr. Theodore H. Blau, Dr. Sheldon C. Sommers and Dr. Edwin R. Fisher. I am also accompanied by Arthur A. Stevens, General Counsel of Lorillard.

While we appreciate the time you have provided for us to testify, Mr. Chairman, we want to point out with all due respect that additional time should also be provided to permit members of the Committee to consider and deliberate on the testimony that we have been permitted to present.
The issues raised by this legislation are serious ones and we believe — and others agree with us — that holding this hearing today, and a mark up tomorrow, suggests an inordinate rush to judgment.

Before the present national policy regarding where and the use of tobacco products is discarded, we would hope that this committee will not only hear a full presentation of the industry position, but also take a reasonable time to study the record and reflect on it.

We are here to express our strongest opposition, not only to S.772, but also to the other bills — S.1116 and H.R.1624 — that could be used in a coordinated program to regulate cigarette labeling and advertising. Despite the appearances of good intentions, this is bad legislation. It is, of course, bad for our business. More than that it sets a bad precedent for many other businesses. But most importantly, it is bad for our free society and its citizens.

We oppose this bill because of the fundamental issue it raises of whether this country will have a national policy of choice or of compulsion, of public information or of public conformity. The measure of effectiveness of S.772 will not be the degree to which the public is aware of official warnings, but rather the extent of public compliance with them. The goal of S.772 is not an increased level of awareness about tobacco and health, but a reduced level of smoking.
To ignore the present national policy, as this bill does, is unnecessary, unwise and unfair. S.772 is founded on the notion that people are informed if — and only if — they conform to the government line and stop smoking. In fact, S.772 is a not very subtle giant step down the road to prohibition.

The present policy of Congress and the purpose of the present law is to establish a Federal Program whereby the public may be adequately informed that cigarette smoking may be hazardous to health.

We submit that this has already been accomplished. The evidence shows that over 90 percent of the American public is aware of the claim that smoking is harmful — an unprecedented level of awareness that has been lauded by a Secretary of HHS and by the Surgeon General. There is also widespread awareness of specific claims. More than 90 percent of the public, for example, are aware of the claimed association between smoking and heart disease. Equally high percentages are reported for awareness of claims about smoking and lung cancer and smoking and pregnancy.

This high awareness is not recent nor limited to health professionals or adults. Nearly seven years ago, the Gallup Poll surveyed teenagers across the nation. It found that "a huge majority of teenagers, 96 percent, believe smoking to be bad for one's health, while 89 percent think that smoking is a cause of cancer, and 76 percent link smoking and heart disease."
On the basis of these findings, George Gallup concluded that "efforts to convince America's teenagers of the dangers of smoking apparently have been highly successful."

In spite of these facts, S.772 "finds" that present government and private efforts have been "insufficient" in conveying this information to the public.

Naturally, this legislation is aimed at the tobacco industry. But the precedent embodied in it could well be used against other industries -- food, beer, wine, alcohol, confectionery, cosmetics, are only a few of the obvious targets. The regulatory spirit that inspires this bill convinces me that the hit list will expand. Today, tobacco. -- tomorrow, who knows?

Let us detail the many ways that this bill inflicts its damage on our industry and our society.

One of the most insidious elements of the bill is found in the section on "findings." Resolving scientific questions by a majority vote of Congress is inappropriate and misguided, especially where complex scientific questions are involved. It freezes scientific research in its tracks. It stops the search for an answer -- because Congress has decreed the answer. It has the potential for diverting funds from the basic research needed to answer the questions of what causes many chronic diseases.
Mr. Chairman, I am not a scientist. But as I understand the nature of science, it is not establishing one's own answer and then structuring research designed to prove that predetermined answer. Science is an evolving, changing process of discovering answers to scientific questions.

For example, public health officials have urged people to reduce their smoking, their high blood pressure and their cholesterol levels in order to lower their risk of dying from heart disease. When this risk-factor-reduction hypothesis was put to the test by the Multiple Risk Factor Intervention Trial, or MR FIT as it's called, the results came as a surprise to the governmental scientific community. MR FIT had not proven out their carefully developed and often expounded hypothesis. Ten years, and $115 million tax dollars later, the question of coronary heart disease causation is still a question.

This giant project, sponsored by the National Institutes of Health and reported in the September 24, 1982 issue of The Journal of the American Medical Association, had proved nothing. There was no significantly different reduction in heart disease death rate in the group that had significantly greater reductions in cholesterol, blood pressure and smoking. It seems clear that this government research project casts grave doubt on the validity of one of the warning labels on cigarette packages concerning smoking cessation, and emphasizes the need for continued research and openmindedness.
As was mentioned at last week's hearing, The New York Times on May 3 reported on another study that also demonstrates the danger of legislating scientific findings. This new research, conducted by Columbia University, the National Institutes of Health and the Framingham Heart Study, surprised even its authors. According to the New York Times, it found no evidence that such commonly suspected risk factors as cholesterol, high blood pressure and cigarette smoking differed among those who did and did not have heart attacks. These researchers found that men who suffered heart attacks have more of a certain type of estrogen than men without heart disease.

"These findings," according to the Times, "suggest that heart disease could be primarily a hormonal disorder which, if true, would affect treatment and prevention." And I would add, Mr. Chairman, government health policy and legislation as well.

There will be scientific testimony later with respect to these studies and the proposed findings or S. 772. In addition, many other expert witnesses will have available for the Committee written statements on the scientific issues in the bill.

Turning to the section in the bill on "Smoking Research, Education and Information", there is nothing in this provision that the Secretary of HHS cannot do administratively. There is no authorization of funds in this legislation. But I am sure that the demand for dollars would swiftly follow its enactment.
Establishing a statutory "Interagency Committee on Smoking and Health" would establish an unnecessary and expensive anti-smoking bureaucracy and create another costly advisory committee — with the authority to coordinate — and thereby to control — research on smoking and health at the National Institutes of Health and throughout the entire federal government. In fact, as Dr. Brandt made clear last week, HHS neither wants nor needs a statutory interagency Committee on Smoking and Health.

One of the major innovations of the bill is to be found in Section 6. Rotating a series of new warning statements on packages is calculated to have a "shock effect," to deter smoking, and to force the industry to use its packages, and no doubt its advertising as well, for a Government mandated anti-smoking campaign.

I must note at this point that while S.772 contains four warnings, S.1116 proposes four times that number. Can we anticipate that cigarette packages and advertisements are to become adjuncts to medical journals? Will a bill be proposed for a new warning label every time an article is published alleging an adverse health effect due to smoking?

It is interesting to note that in 1980, after a study, the Department of Health and Human Services opposed warning statements on alcoholic beverages. Indeed, HHS rejected a specific warning label
for pregnant women because, it said, "singling out a specific health issue...may cause consumers to discount the importance of other significant alcohol related health hazards." The Department further pointed out that rotating warning labels "would confuse the public" and "pose potentially difficult administrative and enforcement problems." Finally, HHS expressed concern that "the public is becoming jaded over government warnings" and that the government should use such warnings with caution.

Senators, it is high time for these cogent concerns about health warnings to be applied evenhandedly. It is time to stop singling out tobacco -- and to stop using smoking as a scapegoat for all of the many factors associated with disease.

Testimony offered to both House and Senate Committees during the 97th Congress clearly demonstrated that this proposal for rotating warnings is an attempt to solve a problem that doesn't exist by a method that doesn't work.

The problem does not exist because -- as I pointed out earlier -- virtually everyone is aware of the government's claims about smoking and health. The contrary premise on which this bill and Senator Packwood's recent bill are based is supported by nothing more than the tortured arguments contained in the 1981 FTC Staff Report on Cigarette Advertising -- which, incidentally, the Commission has never
adopted. In that Report, the Staff erroneously concluded that anyone who does not believe the official line on smoking and health must be unaware of the alleged risks.

The Staff's faulty premise was described in a letter last year to this committee from Burns W. Roper, whose organization conducted one of the surveys principally relied upon by the Staff. In his letter, Mr. Roper criticized the Staff's misinterpretation of the survey data, saying:

The FTC staff concludes, based on our and other survey data, that the public is inadequately informed about the dangers of smoking. Using exactly the same data on which they base their conclusion, I would conclude almost exactly the opposite — that the public is highly aware of the reported dangers of smoking.

At the conclusion of my statement, Professor Blackwell, an expert in consumer behavior, will testify in more detail concerning the flaws in the premises underlying the FTC Staff Report and S.772. In addition, because the proponents of this bill have placed such emphasis on that Report, I would like to submit for Subcommittee review and for the record, The Tobacco Institute's Comments on the FTC Staff Report, which were submitted to the FTC in December 1981. Chapter one of those Comments rebuts both the reasoning and conclusions about awareness set forth in the Report.
The distinction between belief and awareness is as crucial as it is overlooked. The fact is that a poll of people's beliefs about smoking and health says nothing about their awareness of claims that smoking is harmful. And the fact that people do not believe these claims does not mean that they are unaware of them, but rather that they are not prepared to conform to government views on smoking and health. That result may frustrate those who want to prevent people from smoking, but it surely provides no support for the claim that people need to know more about the government's position on this issue.

There also is no reason to expect that the proposed rotating warning system will accomplish its sponsors' goal. There is, in fact, ample experience to demonstrate that such a system doesn't work.

Despite claims to the contrary, the fact is that in Sweden — the country that originated rotating labels — per capita cigarette consumption increased after the rotating warnings and a host of other anti-smoking measures went into effect.

Elsewhere in Europe, there are signs that the idea of multiple warning statements is one whose time has come and gone. England, after several years of tinkering with three rotating warnings, has cut back to one general warning which is much like the statement required in this country today and little different from the statement England
started out with in 1971. There may be a better lesson for this Committee in the experience of the British, a people whose traditions of government and individual freedom are more akin to ours, than in that of the Swedish, whose government mandates social engineering and behavioral modification.

Although rotating labeling has not worked where it has been tried, it will most certainly create a regulatory nightmare of confusion, conflict, and a host of logistical problems.

For example, the bill would require each of four warning statements — and in the case of §1116, as many as sixteen statements — to appear "a substantially similar number of times on each brand of cigarettes." Today there are more than 200 cigarette brand packings produced domestically. New brands are introduced and old ones withdrawn. There also are frequent changes in the amount of advertising devoted to each brand. In these circumstances, equalizing the appearance of four different warning statements on every brand would be a logistical impossibility.

All in all, the labeling provisions of Section 6, and the restrictive advertising provisions of §1116 that are sure to be added to the bill by amendment, would place such a severe burden on advertising as to discourage the use of this legal method of marketing a legal product. We suspect that this is an unstated purpose of the bill.
Although the bill in its present form limits the rotational warning requirements to cigarettes sold in this country, S.1116 would expand coverage to export the rotating warning system to all countries in which American manufactured cigarettes are sold. Aside from the presumptuousness of our government dictating to the rest of the world, the warning statements to be included on cigarette packages, the export provisions would place American manufacturers at an unfair competitive disadvantage. In some countries, for example, the sale of cigarettes bearing any warning statement other than that required by local law is prohibited — thus effectively barring U.S. cigarettes carrying the warning statements set forth in this bill or S.1116. In other countries that require specific warning statements, U.S. cigarettes would have to carry two or more such statements.

The problems inherent in a rotational warning requirement are severe enough without the additional burdens that would result from extending the requirement to exported cigarettes. In addition, the bill could also effectively bar the sale of cigarettes imported from countries which do not use the U.S. method of "tar", nicotine and carbon monoxide testing.

If this bill were amended to include the export provisions of S.1116, it would create confusion, chaos, and competitive disadvantage for American products in many overseas markets. It would also, we submit, conflict with existing federal export law and policy by
attempting to regulate an American export for a reason which has nothing to do with American security, foreign policy or domestic shortages.

The bill's requirement for disclosure of "tar", nicotine and carbon monoxide levels on cigarette packages also is unnecessary and unjustified. The public is continually made aware of "tar" and nicotine levels, which the cigarette manufacturers have included in their advertising since 1971.

Disclosure of carbon monoxide levels would serve no legitimate purpose. There is not sufficient scientific evidence for the conclusion that exposure to carbon monoxide from cigarette smoking causes disease. There is also no generally accepted method of measuring carbon monoxide in cigarette smoke.

Many of the attacks on this industry are based on the false premise that cigarette advertising encourages young people and nonsmokers to smoke. The facts are that cigarette advertising is brand advertising, intended to encourage smokers to switch from competitive brands. In the past decade, the percentage of the population who smoke has declined, while during the same period the number of cigarette brands and brand styles has substantially increased. Cigarette manufacturers must advertise to gain or maintain share of current smokers for each
of their competitive brands. Clearly, such advertising neither is designed for, nor has the effect of increasing the number who smoke.

In light of these facts, it is not surprising that in testimony before the House Committee on Commerce in 1969, the then Chairman of the FTC acknowledged that cigarette advertising is brand advertising. And in his 1979 Report on Smoking and Health, the Surgeon General said the following:

As the cigarette industry has asserted, the major action of cigarette advertising now seems to be to shift brand preferences, to alter market shares for a particular brand.

The provisions of Section 5 of the bill — requiring all companies to report "a complete list of each chemical additive used in the manufacture" of cigarettes to the Secretary of HHS — and the Secretary in turn to report that list to Congress — completely ignore the existing disclosure agreement worked out last year in good faith between the cigarette manufacturers and HHS — which agreement Secretary Brandt praised during his testimony in the House in March and which he called an "opportunity to learn a great deal" just last week.

Pursuant to that agreement, the manufacturers have made available to HHS, with suitable safeguards to protect this highly sensitive commercial information, a list of their commonly used ingredients — and, as Dr. Brandt pointed out to this Committee, those ingredients used in large quantities by even a single manufacturer.
By contrast to that agreement's careful balancing of the Secretary's interest in obtaining information about these ingredients and the manufacturers' interest in confidentiality, this bill creates a substantial risk of unauthorized disclosure to the public of vital trade secrets. Although the bill purports to grant trade secret protection to this information, it effectively nullifies that protection by requiring the Secretary to report annually to Congress a listing of all cigarette ingredients. The bill states that such reports shall not identify the ingredients used in any brand of cigarettes or by any specific manufacturer, but it would not be difficult for any manufacturer to determine from the list the ingredients that it does not use and that therefore must be used by one or more of its competitors, with that information the manufacturer could far more easily undertake to duplicate the tastes of the successful brands of its competitors. Beyond that serious problem, it is a sad fact that this industry in particular has been victimized numerous times by "leaks" of information submitted to the government in the strictest confidentiality. The present agreement with HHS adequately addresses that concern; the present bill does not.

§ 116 is even worse. It would require every package of cigarettes to "disclose each natural or synthetic ingredient,


including chemical additives, contained in such cigarettes. This provision would simply exclude the cigarette manufacturers from the trade secret protections that are available to every other consumer product industry. Yet, at the same time, it would not advance the stated intention of the Senate bill sponsors to "inform" consumers, since cigarette purchasers could hardly be expected to read — much less pay attention to — the clutter of detailed information that the Senate bills would require on cigarette packages: rotational warnings and the listing of "tar" and nicotine, carbon monoxide and all "natural or synthetic" ingredients. We can only conclude that the true purpose of these bills is not to inform the public but to harass the manufacturers of a lawful product.

This industry's contribution to our Nation's economy is enormous — more than $57.6 billion to the 1979 Gross National Product, according to a study by the Wharton Applied Research Center at the University of Pennsylvania. This amount was 2.4 percent of the GNP for that year. That contribution generated the equivalent of over two million jobs — and some $21 billion in taxes of all kinds to federal, state and local governments.

I don't need to remind you, Senators, that since the federal excise tax was doubled on January 1st of this year, tobacco's tax contribution is now even more.
In addition to being an economically strong segment of the private sector, this industry has also been a responsible member of our society. Since 1954, the industry has committed some $11 million dollars for unrestricted, independent research seeking answers to the continuing questions regarding smoking and health. In fact, during the last ten years, our contribution has been far greater than the combined expenditures for similar research by all the voluntary agencies who oppose smoking.

For almost 20 years, this industry has worked to avoid having its brand advertising reach young people. Most recently, we have conducted an advertising campaign which has already reached 122 million Americans. One of the ads asks, "Do cigarette companies want kids to smoke?" And it gives our answer: "No. As a matter of policy. No. As a matter of practice. No. As a matter of fact. No." Two other ads also make the same point. Here are the three ads. I ask that they be made part of the record. These ads have appeared in such major publications as Time, Newsweek, U.S. News and World Report, Sports Illustrated, People and TV Guide—to name a few.

Senators, I will conclude my statement by emphasizing some major points which should be considered by the Committee in its deliberations on this bill.
Exercising their right of free choice, fifty-three million Americans continue to smoke while thirty million Americans are said to have quit smoking.

Virtually everyone is fully aware of the assertions about the hazards of smoking.

The decision to smoke or not is an informed choice to be made by individuals as a matter of right.

Therefore, more governmental regulation of freedom of choice by legislation is unwarranted, improper and unnecessary.

Even though the question of smoking and health is an open question, it is also a highly emotional subject. Public policy, however, must be based on more than the passionate advocacy and the heated claims of those who oppose smoking.

We ask only that you consider all the facts and all the arguments before you cast aside the present law which is functioning so well in the interest of all Americans.
The CHAIRMAN. Thank you.

Mr. JUDGE. Dr. Blackwell will introduce himself and follow me.

The CHAIRMAN. Dr. Blackwell?

STATEMENT OF ROGER D. BLACKWELL, PH. D., PROFESSOR OF MARKETING, OHIO STATE UNIVERSITY, COLUMBUS, OHIO

Dr. BLACKWELL. Mr. Chairman, I am Roger D. Blackwell, professor of marketing at the Ohio State University. I am very pleased that you have asked for information which would analyze how consumers would respond to the bill.

Section 6 of S. 772 would replace the present Surgeon General’s warning statement on cigarette packages, and presumably in cigarette advertising as well, with a rotational system of four warning statements. I have reviewed that proposal, as well as the recommendation on which it apparently is based, set forth in a 1981 FTC staff report on cigarette advertising. I also have reviewed the testimony presented at the May 5 hearing on S. 772.

In my opinion, the change in the warning statement proposed by section 6 is flawed for two basic reasons.

First, the labeling provisions of section 6 would replace a highly successful program of informing consumers about the claimed health risk of smoking with a program of unknown and potentially counterproductive consequences. All of the studies conducted about consumer awareness of smoking and health issues lead to the conclusion that people are universally aware of the claims that smoking is hazardous to health, both in general and specific terms. Why abandon this program in favor of a course that is not only uncharted but, as I hope to demonstrate, likely to lead to results quite the opposite of those apparently intended by the bill’s sponsors?

The second fundamental flaw in section 6 is that the system of rotated warnings attributing specific health problems to smoking will probably lead to one of two unhappy effects: either consumers will erroneously believe that they will personally suffer the specific health problems identified in the warning statements, even though, I think, we all agree, such problems affect only a minority of the smoking and nonsmoking population—such that, if someone concludes that this will happen to them and it doesn’t, that’s a deceptive warning—or, more importantly, consumers will correctly perceive that only a small proportion of the population is at risk from the stated health problems and thus will conclude that the Surgeon General and other health authorities have now decided that smoking is not hazardous to all people. We are suggesting these warnings are something that would only apply to a few people rather than the present warning which would apply to all people.

Since it appears that the specific diseases identified in the proposed warnings apply primarily to older people and pregnant women, the probable effect of the law would be to reduce drastically the impact of the warning statement for young people.

As Dr. Mortimer Lipsett, Director of the National Institute of Child Health and Human Development, said in his testimony to this committee, young people think and live in the present. Accordingly, they tend to disregard warnings about diseases such as heart
disease and lung cancer, problems associated with middle-aged and older people.

The rotational warning system proposed in the present bill and the FTC Staff Report apparently is based on the assumption that the public is not adequately aware of the specific claimed health consequences of smoking. The same assumption underlies the testimony of the proponents last week of S. 772. For several reasons, this assumption is unfounded. This is discussed in my written statement at pages 4 to 9 in some detail. It is a fairly scientific approach, so I think I will just refer to that, if that is acceptable.

Even without considering the underestimating error that might occur because they were FTC studies, the studies consistently report that about 90 percent of the population is aware of the general claim of health hazards, but I think perhaps one of the things that has not come out clearly in previous hearings is the fact that 80 to 90 percent of the people are also aware of the specific problems mentioned in the warnings proposed in S. 772.

For example, the 1978 Gallup study found that 81 percent of Americans believe that smoking causes lung cancer. We are going to try to change that, but 81 percent already believe that. The 1980 Roper study found that 84 percent of the public believe that smokers are many more times likely to develop lung cancer than nonsmokers. The same study found that 92.5 percent believe that smoking causes at least some cases of lung cancer. And in the 1980 Chilton study, 90.3 percent of teenagers and 86.2 percent of adults agreed that smoking can produce lung damage in teenagers.

These percentages do not relate merely to a general statement about smoking and health. They concern the specifics of the warnings proposed by S. 772.

Emphysema is mentioned in the bill. In the 1980 Chilton study, 79.7 percent of teens and 90.7 percent of adults were aware of the claimed association between smoking and emphysema. In the 1980 Roper study, 85.2 percent of respondents believed that smoking causes at least some cases of emphysema.

Heart disease is another problem mentioned in the bill. In the 1980 Roper study, 80 percent of the public agreed that smoking causes at least some cases of heart disease. The 1980 Chilton study found that 90.2 percent of teens and 90.7 percent of adults believe that smoking is associated with heart disease.

In the area of miscarriage and pregnancy, the 1980 Chilton study found that 89.8 percent of teens and 89.7 percent of adults agree that smoking can have an effect on the smoker's baby.

In short, it is fair to conclude that public awareness of the various claims about smoking and health that are the focus of the bill is, as a practical matter, already universal.

Perhaps, Senators, you would say, well, if people are aware of this and these claims, why do they continue to smoke? The FTC Staff Report states several times that smokers are not as well informed as nonsmokers, implying that such informational differences are responsible for the decision whether or not to smoke, but that conclusion is flatly refuted by the 1980 Chilton study, the very study relied upon by the FTC staff to demonstrate the supposed difference in the levels of awareness between smokers and nonsmokers.
The conclusion reached by the Chilton study was as follows:

Factual knowledge about the health consequences of smoking was not found to be significantly related to current smoking behavior. No more differences between knowledge levels of smokers compared with nonsmokers were found to be significant at the 0.05 level than were to be expected by chance.

That is page 22 of the report.

This important finding that consumer knowledge has no relationship to smoking behavior refutes the notion that people who smoke do so because they are “uninformed” about the claimed dangers of smoking. The lack of a relationship between awareness and smoking also demonstrates that increasing consumers’ awareness about the health hazards attributed to smoking is unlikely to influence their smoking behavior.

Consequently, to the extent that the present bill is based on a desire to reduce smoking, and putting aside the question whether behavior modification is an appropriate goal for the Government of this country, the warning statements proposed by S. 772 are simply irrelevant.

Given these facts, a change of the sort contemplated by this bill should not be undertaken unless there is significant evidence that the proposed system of rotated warnings would better achieve the goal of informing the public. The little evidence that exists not only fails to support the proposition of this bill, but in fact contradicts it. That evidence is cited on page 12 of my written statement.

It shows that, even the preliminary research that has been done tends to support the conclusion that the proposed rotational warnings would be considered irrelevant by some consumers as compared to the present warning statement that announces to every consumer the Surgeon General’s conclusion that “smoking is dangerous to your health.”

These findings are particularly significant in view of one of the major premises of the FTC staff report, and presumably the present bill, that consumers should perceive information concerning smoking and health to be personally relevant. I believe that is one of the major theoretical positions embodied in the staff report and the bill.

Since it is basic to human nature to conclude that risks apply to “the other person,” specific warnings that might be more personally relevant to some consumers would by definition be personally irrelevant to most other consumers, who would be by far the majority, and would especially affect the young people.

For the individual who sees these diseases as unlikely to occur personally, then the proposed new warnings would be less relevant. Such individuals are particularly likely to be young consumers who may be making the decision of whether to smoke or not.

Then we have the other problem of one of the warnings would talk about cigarette smoking along with drugs like marijuana, which are euphoric, produce a rush, and are addictive. That is a little bit like saying to your 5-year-old, “I’m leaving the house, and there are some matches on top of the fireplace. Don’t play with them while I’m gone,” which is another specific problem.

While the decision to smoke is really not related to advertising, at least in the present situation parents or peers can say to people who are deciding to smoke that “the Surgeon General has deter-
mined that smoking is dangerous to your health." I see that as its primary value to parents, to be able to counsel with their children.

Under the proposed new warnings, the logical conclusion would be that the Surgeon General has changed the general warning and has decided that smoking is unhealthy only for certain segments of the population and in specific, limited situations.

Mr Chairman, last year I appeared before the Committee on Commerce to explain why, in my view, replacement of the current cigarette warning statement with a system of rotated warnings would be unnecessary and ill-advised. I regret to say that the rotating warning proposal set forth in S. 772 proceeds from the same faulty assumptions, which are well supported, I believe, by behavioral theory and research. No matter how laudable the intentions of this group, the proposed legislation really would not be desirable or beneficial to the American public.

Thank you.

[The prepared statement of Dr. Blackwell follows:]
I am Roger D. Blackwell, Professor of Marketing at the Ohio State University, specializing in the analysis of buyer behavior and development of marketing strategy. My Ph.D. degree was earned at Northwestern University, with a concentration in consumer behavior. I have authored or coauthored fourteen books and over fifty articles published in professional or business journals that report research that I and others have conducted concerning the communications process, consumer decision processes involved in buying and using goods and services, and variables involved in marketing strategy. My most recent book is the fourth edition of Consumer Behavior, published in 1982. The book describes psychological principles involved in buying and consumption and is the most widely adopted textbook in the field. A complete list of my publications is submitted with this statement.

This is my personal statement and should not be construed to reflect the views of the Ohio State University or any other institution with which I am or have been affiliated.

Section 6 of S.772 would replace the present Surgeon General’s warning statement on cigarette packages (and presumably in cigarette advertising as well) with a rotational system of four warning statements. I have
reviewed that proposal, as well as the recommendation on which it apparently is based set forth in a 1981 PTC Staff Report on cigarette advertising. I also have reviewed the testimony presented at the May 5 hearing on S.772.

In my opinion, the change in the warning statement proposed by Section 6 is flawed for two basic reasons. First, the labeling provisions of Section 6 would replace a highly successful program of informing consumers about the claimed health risks of smoking with a program of unknown and potentially counterproductive consequences. All of the studies conducted about consumer awareness of smoking and health issues lead to the conclusion that people are universally aware of the claims that smoking is hazardous to health, both in general and specific terms. Why abandon this program in favor of a course that is not only uncharted but, as I hope to demonstrate, likely to lead to results quite the opposite of those apparently intended by the bill's sponsors?

The second fundamental flaw in Section 6 is that the system of rotated warnings attributing specific health problems to smoking will probably lead to one of two unhappy effects: either consumers will erroneously believe that
they will personally suffer the specific health problems identified in the warning statements, even though such problems affect only a minority of the smoking and non-smoking population, in which case the warnings would be deceptive, or consumers will correctly perceive that only a small proportion of the population is at risk from the stated health problems, and will thus conclude that the Surgeon General and other health authorities have now decided that smoking is not hazardous to all people. Since it appears that the specific diseases identified in the proposed warnings apply primarily to older people and pregnant women, the probable effect of the law would be to reduce drastically the impact of the warning statement on young people. As Dr. Mortimer Lipsett, Director of the National Institute of Child Health and Human Development, said in his testimony to this Committee (at p. 9), young people think and live in the present. Accordingly, they tend to disregard warnings about diseases such as heart disease and lung cancer, problems associated with middle aged and older people.

The rotational warning system proposed in the present bill and the PTC Staff Report apparently is based on the assumption that the public is not adequately aware of
the specific claimed health consequences of smoking. The same assumption underlies the testimony of the proponents of 5,772. For several reasons, this assumption is unfounded.

In the first place, determination of what constitutes a "sufficient" level of awareness in such a complex area is both difficult and subjective, although it appears to me that, by any standard, the level of awareness about the claimed health hazards of smoking is astonishingly high. A basic question is the amount of information a consumer can reasonably be expected to be aware of in connection with a decision to use any particular product. Many of the questions posed in the surveys cited by the FTC Staff required a detailed scientific knowledge about questions of smoking and health, including a complete awareness of every health problem that has been attributed to smoking, the specific size of the increase claimed in the risk of incurring each problem if one smokes, the percentage of each particular health condition that is attributed to smoking, and the proportion or number of people who die from a given health condition. From the consumer viewpoint, what value is there in possessing such a complex array of information? When one considers the tremendous amount of information to which the consumer is exposed every day, and the fact that consumers do not possess unlimited processing capacities,
it clearly would seem more functional for the consumer to retain in memory the overall implication of these numerous bits of information about the claimed consequences of smoking, i.e., that smoking is dangerous. That is precisely the information conveyed by the present Surgeon General's warning statement.

Proper evaluation of the adequacy of consumer awareness also is hampered by the fact that there is no baseline for comparison. For example, how does consumer awareness about smoking and health compare to the information consumers possess about the health hazards attributed to other products such as automobiles, liquor, and hang-gliders? Without such comparison, judgments about the sufficiency of the level of consumer awareness are highly subjective and cannot serve validly as a basis for the far-reaching changes embodied in Section 6 of the bill.

Moreover, an examination of the studies on which the proponents of S.772 rely for the proposition that consumers are not sufficiently aware of the dangers associated with smoking reveals that the proponents have misconstrued the results of those studies.

Perhaps most significant is that many of the survey measures assessed beliefs rather than awareness. The
distinction between belief and awareness is a critical one given the existing controversy over the health threats presumed to be posed by smoking. Consider the likely situation of a survey participant who recognizes that smoking has been found to be associated with particular health problems but finds the evidence insufficient for demonstrating that smoking causes these health problems. As stated in the 1980 Burke Marketing Research Focus Group Study commissioned by the FTC:

"Further doubt about the direct relationship of smoking and cancer seems to be related to the fact that these persons had known smokers who had lived long lives without contracting cancer and non-smokers who had suffered from that disease." (Burke Study Analysis at p. 4.)

Many of the measures employed in the studies asked the respondents to indicate their agreement with or the correctness of statements such as "smoking causes X." Respondents who disagreed with these "supposedly true" statements are categorized as unaware. Alternatively, these respondents may be aware of the medical evidence but have concluded that while smoking is "related" to X, it does not "cause" X. Evidence supporting this alternative explanation is provided by the 1980 Chilton study conducted for the FTC Staff. At one point survey participants were
asked whether heart disease "had been found to be associated with cigarette smoking." (Question 42a). Over 90% of the adults and teenagers interviewed responded affirmatively, only 9.8% of the teenagers and 9.3% of the adults answered "No" or "Don't know." Later in the interview, these same persons were asked whether the statement "cigarette smoking is a major cause of heart disease" (Question 52) was true or false. 26.8% of the teenagers and 19.5% of the adults were presumed to be "unaware" of the claim embodied in this statement. Such response variations between questions involving the same disease, but which differ in positing smoking as either the cause of or simply associated with that disease, strongly suggest that many persons classified as "unaware" in fact are aware of smoking's asserted relationship to various health risks. These persons simply do not believe that smoking causes these health problems.

Question wording has long been recognized as a critical area in survey research. The FTC Staff Report acknowledges that "...conservative sounding statements have been found to be more likely to generate agreement." (Report at p. 3-3). By the same token, statements employing extreme wording or phrases are likely to inhibit agreement. Thus, the amount of agreement with the statement "smoking is by
far the greatest cause of lung cancer" used in the 1980
Roper Study was probably lower than had the statement been
phrased, "smoking is the greatest cause of lung cancer."
Wording ambiguity can also influence the response patterns
to a question. Phrases repeatedly appearing in the Roper
Study such as "by far," "greatly increases," and "signifi-
cantly increases," are very subjective. For example, some
people may perceive a 30% risk increase as a significant
increase, while others may not.

It is interesting to note that the FTC Report
cites evidence that people tend to ignore or discount
statistical information in making judgments. (Report at
pp. 4-14 and 4-15) Given this evidence, it seems in-
consistent to employ measures of "statistical knowledge"
for assessing the level of awareness concerning the claimed
effects of smoking. Measures of this type, however, were
frequently employed as indicators of consumers' awareness
about the asserted dangers of smoking (e.g., "What percent
of lung cancer cases are caused by cigarette smoking?" -
Chilton 1980; "Smokers are at least ten times as likely to
develop lung cancer than non-smokers" - Roper 1980).

For these reasons, it would appear that current
studies have underestimated consumers' awareness about the
health hazards associated with smoking. There is, unfortunately, no way of predicting how much underestimation error exists in these data. But even without considering this underestimation error, the studies consistently report awareness levels in the 80 to 90 percent range about the specific problems mentioned in the warnings proposed by S. 772.

For example, the 1978 Gallup Study found that 81 percent of Americans believe that smoking causes lung cancer. The 1980 Roper Study found that 84 percent of the public believe that smokers are many more times likely to develop lung cancer than nonsmokers. The same study found that 92.5 percent believe that smoking causes at least some cases of lung cancer. And in the 1980 Chilton Study, 90.3 percent of teenagers and 86.2 percent of adults agreed that smoking can produce lung damage in teenagers.

These percentages do not relate merely to a general statement about smoking and health. They concern the specifics of the warnings proposed by S. 772.

Emphysema is mentioned in the bill. In the 1980 Chilton Study, 79.7 percent of teens and 90.7 percent of adults were aware of the claimed association between smoking and emphysema. In the 1980 Roper Study, 85.2 percent of respondents believed that smoking causes at least some cases of emphysema.
Heart disease is another problem mentioned in the bill. In the 1980 Roper Study, 90 percent of the public agreed that smoking causes at least some cases of heart disease. The 1980 Chilton Study found that 90.2 percent of teens and 90.7 percent of adults believe that smoking is associated with heart disease.

In the area of miscarriage and pregnancy, the 1980 Chilton Study found that 89.8 percent of teens and 89.7 percent of adults agree that smoking can have an effect on the smoker’s baby.

In short, it is fair to conclude that public awareness of the various claims about smoking and health that are the focus of the bill is as a practical matter universal.

It would be wrong to conclude that consumers are not adequately informed about the claimed health consequences of smoking simply because many consumers continue to smoke. The FTC Staff report states several times that smokers are not as well informed as nonsmokers, implying that such informational differences are responsible for the decision whether or not to smoke. But that conclusion is clearly refuted by the 1980 Chilton Study, the very study relied upon by the FTC Staff to demonstrate the supposed difference in the levels of awareness between smokers and
nonsmokers. The conclusion reached by the Chilton Study was as follows:

"Factual knowledge about the health consequences of smoking was not found to be significantly related to current smoking behavior. No more differences between knowledge levels of smokers compared with non-smokers were found to be significant at the 0.05 level than were to be expected by chance." (Page 22)

This important finding that consumer knowledge has no relationship to smoking behavior refutes the notion that people who smoke do so because they are "uninformed" about the claimed dangers of smoking. The lack of a relationship between awareness and smoking also demonstrates that increasing consumers' awareness about the health hazards attributed to smoking is unlikely to influence their smoking behavior. Consequently, to the extent that the present bill is based on a desire to reduce smoking -- and putting aside the question whether behavior modification is an appropriate goal for government in this country -- the warning statements proposed by S.772 are simply irrelevant.

Given these facts, a change of the sort contemplated by this bill should not be undertaken unless there is significant evidence that the proposed system of rotated warnings would better achieve the goal of informing the public. The little evidence that exists not only fails to support that proposition, but in fact contradicts it.
An initial study for the FTC Staff was undertaken in 1980 by Walker Research to assist in the selection of specific warning statements, while another study conducted by Burke Marketing Research examined consumers' recall of various warnings. Importantly, neither study examined the impact of such warnings on consumer awareness. There are accordingly no data to indicate that a rotational system such as that proposed by the bill would meet the objective of providing consumers with "sufficient" awareness about the claimed consequences of smoking, particularly if sufficiency is gauged by the very detailed measures used in the surveys cited by the FTC Staff.

What the studies do demonstrate is that consumers are likely to discount warnings that link smoking to specific health problems. In its Summary of Key Findings, for example, the Burke Focus Group Study states:

"The messages related to birth control pills and heart attacks tended to confuse the participants, who did not thoroughly understand the synergistic effects which form the basis of the message. These two statements relating to oral contraceptives also had the least personal relevance and were rather easily dismissed as being intended "for someone else"." (Emphasis added.)
The Study thus concludes:

"It seems that the birth control message could have relevance to a highly select group of people who could be best reached through very specific media. The message might be lost to the population as a whole."

(Burke Focus Group Study Analysis at p. 6.)

That same conclusion appears to apply with equal validity to the specific disease warnings contemplated by S.772.

Thus, even the preliminary research that has been done tends to support the conclusion that the Proposed Rotational warnings would be considered irrelevant by some consumers, as compared to the present warning statement that announces to every consumer the Surgeon General's conclusion that "smoking is dangerous to your health."

These findings are particularly significant in view of one of the major Premises of the PTC Staff Report, and presumably of the present bill: that consumers should perceive information concerning smoking and health to be personally relevant. Since it is basic to human nature to conclude that risks apply to "the other person," specific warnings that might be more personally relevant to some consumers would by definition be personally irrelevant to most other consumers. For the individual who sees these diseases as unlikely to occur personally, then the proposed
new warnings would be less relevant. Such individuals are particularly likely to be young consumers who may be making the decision of whether to smoke or not. While the decision to smoke is not related to advertising, at least in the present situation parents or peers can say to people who are deciding to smoke that "the Surgeon General has determined that smoking is dangerous to your health." Under the proposed new warnings, the logical conclusion would be that the Surgeon General has changed the general warning and has decided that smoking is unhealthy only for certain segments of the population and in specific, limited situations.

Mr. Chairman, last year I appeared before the Committee on Commerce to explain why, in my view, replacement of the current cigarette warning statement with a system of rotated warnings would be unnecessary and ill-advised. I regret to say that the rotating warning proposal set forth in S.772 proceeds from the same faulty assumptions. No matter how laudable the intentions of the sponsors, the proposed legislation would not be desirable or beneficial to the American public.
The CHAIRMAN. Thank you.
Do you want each of these witnesses to testify?
Mr. JUDGE. Yes, I do.
The CHAIRMAN. I am going to give each of you 5 minutes. If you need more, just let me know. I would prefer you to summarize and then have questions from both Senator East and myself.
Shall we go to Dr. Blau next?
Mr. JUDGE. Yes, please.
The CHAIRMAN. Dr. Blau?

STATEMENT OF DR. THEODORE H. BLAU, INDEPENDENT PRACTICE OF CLINICAL PSYCHOLOGY, TAMPA, FLA.

Dr. Blau. Thank you, Mr. Chairman. I appreciate the opportunity of appearing before your committee.

My name is Theodore Blau. I hold a doctorate in psychology from the Pennsylvania State University, which I received in 1951. I have been in the independent practice of clinical psychology and consulting psychology in Tampa, Fla., since 1953. Formerly, I was professor of psychiatry at the medical school of the University of South Florida. I am a diplomate of the American Board of Professional Psychology as well as a diplomate of the American Board of Forensic Psychology.

I am a member and past president of the American Psychological Association. Last year I was president of the American Psychological Foundation. I am a member of the Evaluation Research Society and have conducted evaluation studies for private corporations as well as evaluation research for various branches of the U.S. military and the Federal Government. I am the author of one book, a number of chapters in other books, and over 50 scientific articles.

For several years I have been involved in the evaluation of the research and current state of knowledge about the psychological aspects of cigarette smoking. This evaluation research includes a thorough review of historical factors, current theories and research, and assessment of the quality of that research pertaining to dependence, addiction, and cultural aspects of smoking behavior. In addition to this literature review, I have continuing communication with active researchers in the area. I am the author of the forthcoming section on "Smoking Behavior" that will appear in the Wiley Encyclopedia of Psychology.

I have reviewed S. 772 and feel that the "public awareness statement" that "smoking is addictive" misrepresents current scientific understanding of smoking behavior. As a consequence, it is my belief that the provision of the proposed bill that mandates such a warning on cigarette packages is clearly unwarranted.

In this regard, I have reviewed the recent pamphlet published by the Department of Health and Human Services which is entitled, "Why People Smoke Cigarettes." This was discussed and submitted for the record at last week's hearing by Dr. Brandt. Frankly, I am disappointed in the lack of scientific objectivity of this pamphlet. One can only conclude that the people at the National Institute on Drug Abuse and the Public Health Service have decided that smoking is addictive and seek to prove it by emphasizing only the research findings that are consistent with that point of view.
I need only point out that the vast majority of the so-called supporting references in the new pamphlet are publications which are released or sponsored by these agencies. In all of these publications one finds statements by the scientist authors that research evidence does not support the conclusion that smoking cigarettes or that the active nicotine in cigarettes is addictive. I hasten to add that I am not criticizing anyone's motivations. Rightly or wrongly, cigarette smoking has become a very emotional issue in recent years and this can result in overenthusiasm even in the scientific arena.

I have noted that this DHHS pamphlet was developed from Dr. Pollin's testimony before this committee last year on a bill with provisions quite similar to those of S. 772. Based on the positions taken by Dr. Pollin in 1982 as well as those apparently taken last week, I am forced to conclude that the affiliated organizations, the National Institute on Drug Abuse and Office on Smoking and Health, are anything but unbiased, scientific sources. While these organizations have every right to maintain a view that smoking is an addiction, I must disagree that such a view is scientifically established.

To illustrate this point, recall that Dr. Pollin's testimony earlier this month began with reference to a claim that the American Psychiatric Association included cigarette smoking as a form of drug dependency. While the American Psychiatric Association's Diagnostic and Statistical Manual, known as DSM-III, does include a category labeled "tobacco dependence," does this necessarily mean that such a category is medically or scientifically justified? Certainly not. Many professionals within the psychiatric and psychological fields disagree very strongly with the diagnostic scheme outlined in DSM-III.

Furthermore, in my view, Dr. Pollin's testimony provides a good example of the pervasive definitional shallowness characteristic of discussions in this area. According to my training and my experience, the word "addiction" is a medical term referring specifically to the use of psychotropic drugs in increasingly escalating doses due to tolerance to their effects, and motivated strongly by the avoidance of medically consequential withdrawal symptoms which almost immediately follow abstinence. Yet, although Dr. Pollin initially describes his testimony as having to do with "the addictive properties of tobacco smoking," he quickly goes on to discuss the more vague and questionably relevant concepts of "dependence" and "abuse liability."

Also revealing is Dr. Pollin's own admission that "evidence is not yet conclusive as to whether or not there is physiologic dependence or what type of withdrawal symptom is associated with cigarette smoking." If such evidence is lacking, as I also believe it to be, it seems to follow that legislating a medical label of "addiction" as applicable to cigarette smoking is not scientifically or ethically justified.

In a sense, Dr. Pollin seems to have marshaled to his argument even the most speculative of suggestions, regardless of scientific support or lack of the same. For example, he testified that use of tobacco is an "integral and crucial step in the sequences of developing drug use patterns." The gratuitous nature of such a comment
becomes clear when one considers that even the National Institute on Drug Abuse has published evidence suggesting otherwise.

To say that cigarette smoking is addictive is to erode the concept of addiction, not only scientifically, but also in the public mind. And the suggestion that cigarette smoking leads to addiction in illicit drugs is a further challenge to the public's credence.

One must wonder what would be the anticipated effects of such arbitrary governmental pronouncements. Certainly, to warn a smoker that his habit or her habit is really an addiction cannot lead the smoker to readily stop the behavior. It could be perceived by many as an excuse for being unable or unwilling to stop. But the Government seems to have already given up on the current smoker and seems to be directing its attention to special groups of nonsmokers, particularly teenagers.

What is the teenager to make of such a warning that cigarette smoking is addictive? He or she sees smokers every day, and sees responsible, normal, and admired people smoking. How many Presidents of the United States were smokers? How many Congressmen and Senators? It is unreasonable to suggest that the teenager will contrast this habit with the impression he probably has received in regard to the dangers of addiction to illicit drugs such as heroin. Isn’t it possible that such a contrast could lead to further disrespect for governmental information? Even aside from the arbitrary nature of an addiction warning, as a clinician dealing with teenagers regularly in my practice, yesterday afternoon I met with eight teenagers in group therapy, as I do every Wednesday afternoon. I see them talk about smoking, talk about their models. It seems possible to me that such a warning could serve more as an enticement than as a deterrent.

For almost 400 years the smoking habit has been an issue never lacking for proponents as well as opponents. Some who smoke seem unable to give up the cigarette habit at least during certain stages of their lives. Others can quit with apparent ease. Few specific areas of applied psychological research have received greater attention than the cigarette smoking habit. Despite this intense scientific focus on the behavioral as well as the physiological aspects of cigarette smoking, no general agreement among scientists exists as to the question of whether cigarettes or their contents are addictive. The behavior and responses of cigarette smokers are quite different than those observed in individuals who are addicted to the opiates, the amphetamines, and alcohol. It is scientifically demonstrable that these substances result in responses quite different from responses to cigarette smoking.

Certainly the scientific literature has provided useful information to those of us working in behavioral sciences and who are concerned with the meaning and the effects of smoking behavior. For example, there’s apparently little difference in the severity of cessation responses between light smokers and heavy smokers. That is a consistent finding. No such finding exists when we talk about heroin and alcohol. Research has indicated that these effects are felt more severely by those who reduced their intake of cigarettes but did not stop completely than by those who abstained totally or who go “cold turkey.” Therefore, to be successful in stopping smok...
ing, one goes cold turkey. If one tries that with the addictive narcotics, most severe sometimes fatal results can accrue.

A third thing reported by various researchers is that the cessation of smoking results in relatively mild effects and in some cases there are no effects. They are of the nature which one might expect to follow the loss of any psychological rewards from any object which is loved or possessed to which a person has been long accustomed and which could be conceived as forming a part of the person’s view of themselves or everyday habits. Such findings and variations are simply not found in the study of addictive substances.

It has been reported that many smokers are able to refrain from smoking for relatively long periods of time for practical, safety, or religious reasons and do so without apparent discomfort and so on a regular basis. Some examples, for instance, are coal miners who may not smoke at the pitface, Orthodox Jews who give up smoking at sundown on Friday and cease smoking until sundown on Saturday, and so forth. Such behavior does not fit conventional views of addiction.

Whereas the effects of use and withdrawal are relatively consistent and predictable with the opiate substances, despite a wide range of uses, reported descriptions of tobacco effects are extremely varied and inconsistent. They simply do not look like addictive withdrawal responses. In this regard, tobacco use is much more like the use of caffeine rather than alcohol or opiates. It has been reported that the continuance of smoking appears more related to a wide range of psychosocial motives such as pleasure, stimulation, sensory motor manipulation, and reduction of negative feelings than to an addictive factor.

Smoking cessation data that have been reported by behavioral researchers and commercial clinics are highly variable and not very helpful in assessing the strength of the smoking habit. What I do find intriguing is a report issued by the U.S. Department of HEW in 1977 which indicates that 95 percent of those people who quit smoking do so on their own, without therapy, without substitutes, without clinics. This is not only a phenomenon in sharp contrast to experience with demonstrably addictive substances, but one which remains to be explored and to be understood.

Clearly, many aspects of smoking behavior are as yet not explored, including why some people smoke heavily and some people smoke lightly. As I discussed a moment ago, research examining the role of nicotine in the cigarette smoking habit is ongoing. Some researchers have even emphasized learning theory explains the reason for smoking and why some people have difficulty quitting, but definitive answers have not been found and emotional conclusions to the contrary serve no legitimate scientific purpose. So, gentlemen, at this time the scientific data do not support the statement that cigarette smoking is addictive.

I would like to very briefly discuss my clinical experience with smoking. During the past 33 years of practice, I have worked and continue to work with children who smoke cigarettes, teenagers, adults, and older people. In reviewing my experience with the thousands of people that I have seen professionally, many of whom are heavy smokers, some continued, some quit with some stress, and
some quit with little or no stress. These patients in no way acted, behaved, or responded like the patients that I have seen who struggle to be released from the addictions of opiates, amphetamines, or alcohol.

Cigarette smokers are very attached to their smoking behavior. They are often annoyed or distressed when they are not allowed to smoke. However, I have noted equally strong attachments in my adolescent and adult patients to such activities as jogging, for instance, and even to other people, usually of the opposite sex. Removal from these activities and persons can result in agitation, sleeplessness, irritation, depression, and other uncomfortable symptoms. I mention these analogies not to be facetious, but to emphasize that the very same behavioral responses one sees upon discontinuance of smoking are very similar in duration and intensity to those that occur after removal of a variety of things in everyday life.

If one thing in my clinical experience strikes me as certain, it is that the motivations for smoking are diverse and involve a panorama of psychosocial factors.

I, myself, smoked cigarettes for 24 years—two to three packages a day for the last 10 years of the habit. I stopped at the age of 35, one year before the Surgeon General’s report appeared. I have not smoked since. I was somewhat uncomfortable for a short period of time, but never highly uncomfortable. The craving never reached the level I have experienced or observed in, for instance, medical weight loss programs. In short, although cigarette smoking is a common and pervasive habit, I can find no convincing basis in the scientific literature or in my own professional clinical experience to justify labeling it or treating it as an addiction.

Thank you very much.

The CHAIRMAN. Thank you.

Dr. Blau took about 15 minutes. Dr. Sommers, can you summarize your statement in about 5 minutes?

Dr. SOMMERS. I will do my best, sir.

The CHAIRMAN. If you can, I would appreciate it.

STATEMENT OF SHELDON C. SOMMERS, M.D., CONSULTANT IN PATHOLOGY, LENOX HILL HOSPITAL, NEW YORK, N.Y.

Dr. SOMMERS. With permission of the chairman, I would like to submit these additional statements for the record.

The CHAIRMAN. Without objection, we will put those additional statements in the record at the appropriate point.

Dr. SOMMERS. I am Sheldon C. Sommers, M.D., a physician specializing in anatomic and clinical pathology, now consultant in pathology at Lenox Hill Hospital, New York, also clinical professor of pathology, Columbia University College of Physicians and Surgeons, New York, and University of Southern California School of Medicine, Los Angeles. I am president of the Arthur Purdy Stout Society of Surgical Pathologists, chairman of the New York State Mental Hygiene Medical Review Board, and past president of the New York Pathological Society and the New England Society of Pathologists.
I am coeditor of Pathology Annual and Diagnostic Gynecology and Obstetrics, and serve on the editorial boards of five other medical journals.

Since 1936, except for service from 1942 to 1945 in World War II, I have been involved in cancer research, with 310 publications. I have been particularly interested in cancers of the lung, pancreas, and esophagus, among other sites, for over 30 years.

Since September 1931, I have served as scientific director of the Council for Tobacco Research, U.S.A., Inc. This is an organization for support of biomedical research, funded by the major U.S. cigarette manufacturers, with a 1983 budget of $7.75 million for research grants. More than $85 million has been expended for research during the 30 years of its existence, about two-thirds for cancer research.

In 1983, approximately 200 grant applications will be considered, the largest number ever received. The donors of the money and the Council for Tobacco Research give complete scientific freedom to the grantees, who conduct their investigations, report and publish their findings freely, as they decide. Competition is now very intense for research funds and the Council's Scientific Advisory Board, aided by outside scientific consultants, exerts every effort to fund the best scientists at the best institutions.

With respect to U.S. Senate bill S.772, I am providing this statement voluntarily, as an individual and not as a representative of any organization. It is based on over 45 years of study, investigation, diagnostic experience, and personal contacts with other researchers in experimental pathology.

My purpose is to comment on some of the purported "findings" of S.772, the Smoking Prevention Health and Education Act of 1983, which, in my view, are inaccurate and do not reflect the current state of scientific and medical knowledge concerning the conditions referred to in the bill.

The bill characterizes cigarette smoking as "the largest preventable cause of illness and premature death in the United States." Based on my experience in New York, where I practice, I assure you that this is not the case. Various substances of abuse, some known long before tobacco was in use and some recently invented, together account for illnesses requiring hospitalization far beyond all other diseases put together.

The term "premature death" has no medical meaning, except for some infants born too immature to survive. The alleged 300,000 deaths were questioned at a recent House of Representatives hearing on a similar bill. This number has no factual basis.

The original Surgeon General's report stated that cigarette smokers as a group did not live as long as nonsmokers. The same report also stated that pipe smokers lived longer than nonsmokers. It is now understood that the increasing population of cigarette smokers from 1910 to 1960, and the concomitant decrease in pipe smokers, explains the phenomenon. These are called secular changes and have no health relevance.

Some studies have reported a statistical association between smoking and death from all causes. These studies, however, do not establish that this association is causal. Other studies, including Scandinavian studies of twins, indicate that a genetic or constitution
tional factor may be a better explanation for this association than smoking. For example, in the Scandinavian studies, the researchers found that in identical twins, only one of whom smoked, there was no difference in number of deaths from all causes.

Epidemiology is one approach to investigating human disease causation. Epidemiological studies involve an experimental group and a control group. For a valid comparison, the groups must be alike as nearly as possible in all respects except for the factor being investigated. In studies of cigarette smoking, smokers and nonsmokers were matched by sex and age. The assumption that the two groups were comparable, however, is not true. There were significant differences in body build, introversion or extroversion, marital history, alcohol use, use of nonprescription medications, police records, military records, and other aspects.

The fallacy of one-to-one comparisons of smokers and nonsmokers with respect to mortality was demonstrated by Rose and Bell in 1971. They studied predictors of longevity in war veterans in Boston, who were reexamined at intervals. One-on-one comparisons placed cigarette smoking No. 1 as predictor of early death, as have other studies. Multifactorial statistical analysis, however, dropped smoking to below No. 30 as a predictor, and dissatisfaction with job became No. 1. The lesson is, beware of facile and sweeping conclusions in the relatively undeveloped practice of epidemiology.

Two other points about statistical epidemiology must be made. Every textbook states them, every active scientist knows them. First, by the nature of the mathematics so far developed, epidemiologic studies deal mainly with random populations. Smokers, however, are self-selected, as are nonsmokers. Mathematical methods developed for random populations cannot be properly applied to selected populations.

Second, epidemiology does not prove cause and effect. All it can demonstrate is a relationship, the nature of the relationship, causal or otherwise, has to be worked out by other methods, usually experimental.

Any experienced researcher knows that scientifically "cause" means something both necessary and sufficient to produce a condition. People who have never smoked develop lung cancer, and very few lifetime cigarette smokers ever develop this disease.

In chronic conditions like cancer no individual factor, such as cigarette smoking, can be singled out. Many factors have been associated with cancer, including genetic influences, oncogenes, male or female sex differences, diet, occupation, geographic location, infectious agents, aging, degenerative tissue changes, and declining immune responses.

There is some evidence that dietary vitamin A and related compounds reduce the incidence of the most common type of carcinoma of the lung, oral cavity, larynx, and esophagus. Widespread clinical trials are now underway to test the validity of this hypothesis.

W. C. Hueper, a famous investigator of carcinogens and a government employee, wrote that the clearly evident multifactorial nature of human lung cancer causation militates against the scientifically unsupportable claim that it is caused by cigarette smoking.

Moreover, despite a great effort, no one has been able to produce human-type lung cancer in experimental animals exposed to fresh
whole cigarette smoke. The Council for Tobacco Research in 1970 undertook a large-scale research program to investigate whether cigarette smoking causes lung cancer in animals. Almost $14 million went into the project in 12 years. To take account of heredity, inbred mice were used and tested for and vaccinated against respiratory viruses. To show these animals could develop the major types of human lung cancers, pure chemical carcinogens were introduced down their tracheas. They were known to metabolize the carcinogens into biologically active forms. About 20 percent in younger mice and over 95 percent in older mice developed human-type lung cancers as a result of exposures to these pure chemical carcinogens.

Thereafter, thousands of mice were exposed daily to fresh whole cigarette smoke up to maximum tolerance during their whole lives—up to 40 months in some cases. The smoke was generated from cigarettes with either low nicotine and high tar or high nicotine and high tar levels. There was no question the cigarette smoke had penetrated into the mice's lungs, because it was worked out quantitatively. At one point there were 11,000 animal manipulations per day, including sham smoking and control mice. After all these years of cigarette smoking, practically zero lung cancers developed and not one case of the human-type cancer most often blamed on smoking.

In other experiments in this research program, mice were primed with intratracheal pure carcinogenic chemicals, then exposed to cigarette smoke during their whole lives. No increase in lung cancer occurred over the incidence found after using pure chemicals alone and, in one experiment smoking was associated with a reduced lung cancer rate.

I understand that at the hearings last week it was stated that on autopsy it is possible to tell, by gross examination of lungs, which were from smokers and which were from non-smokers. I do not agree that this is possible, either grossly or microscopically. Nor can one see deposits of so-called tar in the smoker's lung.

A serious problem in the field of lung cancer is the decline in autopsy rates, now falling below 20 percent in this country. Death certificate causes of death are estimated to be erroneous in as many as 50 percent of the cases when autopsies are performed. Cancer that spreads to the lung from large bowel, ovary, et cetera, is often mistaken for primary lung cancer.

Women who smoke cigarettes have from one-third to one-sixth the incidence of primary lung cancer found in men. My own studies suggest that hormonal factors protect women from lung cancer. Moreover, at least half the lung cancers in women are adenocarcinomas, including low grade peripherally located tumors, cancers arising in scars, and other special tumors not considered to have any relationship to smoking. Some people ignore the basic hormonal, cytologic, and other studies on lung cancer in women and simply blame all such tumors on smoking without regard to the other contradictory and inconsistent evidence.

Chronic bronchitis and emphysema are overlapping conditions, presently difficult to identify, diagnose, and distinguish. Pathologically, chronic bronchitis and emphysema lack any generally accept-
ed diagnostic criteria by which different institutions could compare their material and then search for causative agents.

Considerable new data have accumulated implicating occupation, biochemical reactions, genetic influences, hormones, viruses, and other agents in various chronic lung diseases, including emphysema and bronchitis. There are still many unanswered questions about these diseases. For example, black men who smoke almost never develop emphysema. Women also have far less emphysema than men, without regard to their smoking.

Some studies report miscarriages, stillbirths, and premature births to be associated with smoking during pregnancy. Other studies do not find such an association. Most studies in this area fail to take adequate account of the lower socioeconomic status, alcohol use, dietary deficiencies, increased infections, and other salient differences between the smoking and control groups. Modern multifactoral statistical comparison of smokers and nonsmokers have generally concluded that drug and nontherapeutic agent use, malnutrition and alcohol are more likely to account for any differences in rates of these conditions.

Mothers who smoke may have smaller babies that weigh less, but these babies are not deficient in any way and they grow and develop normally. Moreover, years ago, Yerushalmy discovered that such women had smaller babies before they started smoking cigarettes. This field is controversial, still developing, and the current evidence cannot be regarded as conclusive for U.S. women.

In the field of science, knowledge is gained through experimentation and interpretation. In using the scientific method, a theory is proposed. Thereafter, experiments confirm or refute it. If the latter, a new theory is developed. It is a continuous evolutionary process and needs a critical and open mind. One must be constantly alert for surprises, as Lewis Thomas has written:

In summary, I have reviewed and carefully evaluated the purported findings of Senate bill S. 702. In my opinion, on the basis of current biomedical scientific knowledge, they are unsubstantiated and misleading. Cigarette smoking has not been scientifically established to be a cause of chronic diseases, such as cancer, cardiovascular disease, or emphysema. Nor has it been shown to affect pregnancy outcome adversely. Rapidly accumulating new basic scientific discoveries and reports in the medical literature render the simplistic statements in the proposed bill invalid.

[The prepared statement of Dr. Sommers and statements referred to follow:]
COMMENT BY SHELDON C. SOMMERS, M.D. 
ON STAFF ANALYSIS

The article in the May 1983 issue of the American Journal of Medicine identified a subpopulation of men with coronary heart disease from the Framingham cohort who had elevated blood estradiol not found in the controls. There were no differences between the patients and controls with regard to cigarette smoking, blood cholesterol or other so-called risk factors.

All patients with coronary artery disease can be divided into subgroups of patients with other characteristics such as diabetes mellitus, hypertension, obesity and familial hypercholesterolemia. It is well established that such subgroups have a higher incidence of coronary artery disease than the population as a whole. Now according to the study reported in the American Journal of Medicine men with hyperestrogenemia can be added to the list of subgroups.

This finding is quite interesting because it runs counter to other studies that suggest that elevated estrogen levels protect women against the development of coronary heart disease. The group is relatively small and was studied retrospectively; while the study is subject to some criticisms, its results should not be ignored. Instead, it should form the basis for further research on constitutional or host factors which might contribute to coronary arteriosclerosis. Likewise, the observation that cigarette smoking was not statistically associated with coronary artery disease in these individuals should not be ignored or discounted.
The proposed bill, S. 772, states that "Congress finds that smoking is the primary cause of emphysema" (Section 2(2)). Furthermore, the proposed bill is requiring many changes in cigarette labeling such as the disclosure of carbon monoxide level (Sec. 4(b)(1)), yet there is uncertainty as to its role in the pathogenesis of smoking-associated diseases.

The above statement on pulmonary emphysema paraphrases one sentence in the introduction of the Chapter on neoplastic bronchopulmonary diseases of the 1979 Report of the Surgeon General on Smoking and Health, which reads "cigarette smoking is the most important cause" (1). However, a careful examination of that statement because of the complexity of the causation of pulmonary emphysema, which, together with chronic bronchitis, is collectively known as chronic obstructive lung disease.
Although there are a multiplicity of suspected pathogenic mechanisms and of etiologic factors, the Surgeon General's Report refers only to those relating to cigarette smoking with an incidental discussion of outdoor pollution, indoor pollution, infections, and familial or genetic predisposition. The author of this submission intends to discuss his personal research investigations and those of others questioning the conclusion that smoking is the primary cause of pulmonary emphysema, and the suggestion that carbon monoxide in cigarette smoke has a significant role in the causation of smoking associated diseases. Briefly, his reasons are as follows:

(a) Animal experiments have failed to reproduce pulmonary emphysema from long-term exposure to cigarette smoke, although this has been successfully done for sulfur oxides, nitrogen oxides and other inhalants.

(b) Functional and histopathological studies in humans show uncertain and inconsistent effects of cigarette smoking that do not support the causal relationship between cigarette smoking and pulmonary emphysema.
Epidemiologic studies suggest the association of pulmonary emphysema with several risk factors, such as levels of outdoor and indoor pollution, alcohol consumption, occurrence of previous infections, familial predisposition and genetic susceptibility.

That the content of carbon monoxide in cigarette smoke contributes to the pathogenesis of pulmonary and coronary disease has not been proven. To the contrary, the results of an epidemiologic study reported in March 1983 show that alterations in carbon monoxide yield of cigarettes do not influence pulmonary lung function and coronary heart disease mortality.

A. EXPERIMENTAL ANIMAL STUDIES ON PULMONARY EMPHYSEMA

From 1948 to 1977, the author and his colleagues conducted smoking-related research at the University of Pennsylvania School of Medicine where he was a Professor of Pharmacology. The nature of this investigation was guided in part by comments in the 1964 Surgeon General's Report on Smoking and Health which suggested the need for definit
data on the relationship between cigarette smoking and pulmonary emphysema (2). Although eight of the author's publications were mentioned in the 1967, 1968, 1969 and 1971 Surgeon General's Reports, the coverage was brief and out of context (3-6). Over thirty of his articles on cigarette smoking and over a hundred articles published in the 1970's on related subjects were not mentioned. The government writers cannot be criticized entirely however, because they have been examining only "Smoking and Health" rather than conducting a global review of all suspected risk factors for pulmonary emphysema. Yet this isolated emphasis on one factor (cigarette smoking) has limited the usefulness of Surgeon General Reports as scientific documents.

a. Experimental Pulmonary Emphysema in the Dog. The 1981 report of the Surgeon General (7) states that "there are no published studies that acceptably show in an animal model that the development of emphysema is induced by cigarette smoking. ... One study in which dogs received smoke directly through chronic tracheotomies reported the development of emphysema (Auerbach, Hammond, Kirman, Garfinkel and Stout (1967)). The lesions were not conclusive and the results have not been confirmed by others."
The initial series of studies conducted by the author and his colleagues dealt with the effects of cigarette smoke in dogs (8-17). These studies failed to show that inhalation of cigarette smoke caused pulmonary emphysema. It is therefore not surprising that there has been no confirmation of the above mentioned single study reporting the development of experimental emphysema. It should be noted that the single positive study, although questioned in the 1981 Surgeon General's Report, was highlighted in earlier Reports (including 1979) and may have been responsible for the impression that cigarette smoking is an established cause of pulmonary emphysema.

b. Experimental Pulmonary Emphysema in the Rat. In 1967, the author and his collaborators developed an experimental model for producing pulmonary emphysema in rats (18). Unlike other models reported hitherto, this model permitted pulmonary functional measurements in addition to histopathologic observations signifying pulmonary emphysema. The exposure of animals to cigarette smoke did not cause pulmonary emphysema (19). Other investigators have also used the same model and have confirmed essentially its acceptability for the study of pulmonary emphysema.

c. Experimental Pulmonary Emphysema in the Mouse. In 1974, the author and his collaborators developed a technique
for measuring lung function in the mouse that could not be performed previously (20-22). Chronic exposure to cigarette smoke simulating the dosage of smokers did not cause pulmonary changes signifying emphysema. It is a disappointment to the author that the only study comparing high and low nicotine cigarettes on pulmonary function of mice was overlooked in the 1981 Report of the Surgeon General entitled The Changing Cigarette (7). It is possible that the study was ignored because of the unexpected observations that the lungs seem to adapt to repeated exposure of cigarette smoke.

Comparative Studies with Air Pollutants. Dogs, rats and mice are useful experimental models for the study of toxicity of air pollutants. Animals exposed to low levels of products of fuel combustion show impaired lung function and pathological lesions characteristic of pulmonary emphysema. The studies are described in the new series of Criteria Documents that have been prepared by the Environmental Protection Agency on ozone and photochemical oxidants (23), nitrogen oxides (24) and sulfur oxides with particulates (25). This author questions the logic of finding cigarette smoke "a major cause" of pulmonary emphysema when primary air pollutants have been shown to cause pulmonary emphysema in experimental
animals, and using the same animal models, cigarette smoking has not.

B. HUMAN STUDIES ON PULMONARY FUNCTION AND HISTOPATHOLOGIC CHANGES IN CIGARETTE SMOKERS

The Chapter on chronic obstructive lung diseases in the 1979 Surgeon General's Report (pages 6-7 to 6-52) starts by defining the terms chronic bronchitis and pulmonary emphysema. For each definition, there is a positive statement on structural or pathological alterations such as hypertrophy of mucous secreting apparatus and epithelial metaplasia as well as more classic histopathological evidence of inflammation for bronchitis and abnormal enlargement of the air spaces distal to the terminal nonrespiratory bronchiole accompanied by destructive changes of the alveolar walls for emphysema.

These definitions are essentially ignored in the remainder of the chapter because functional tests, macrophage changes and enzymatic contents are regarded as pathological or adverse signs of pulmonary emphysema. The definition of adverse and nonadverse effects has been vigorously debated in recent years by the National Academy of Sciences (26) and the World Health Organization (27, 28). The National Academy of Sciences defines adverse effects as responses that are
The reversible effects are regarded as adaptation or responses of the defense mechanism of the lung to the inhalant. The predominant opinion is that irreversibility is a prerequisite to the definition of an adverse effect. In terms of tobacco use, most of the functional effects described in the 1979 Surgeon General's Report are reversible, not adverse according to the prevailing definition, and are indicative of physiological and biochemical adaptation of the lungs.

b. Respiratory symptoms and pulmonary functional changes. The section on the natural history of chronic obstructive lung disease (Pages 6-10 to 6-11) ends with the qualifying statement that pathological data are the most specific and sensitive parameters relating to pulmonary emphysema. It is further stated that "the relationship of early respiratory symptoms to subsequent development of lung disease is unclear" and that "longitudinal studies demonstrating that individuals with abnormal tests of small airways function are at greater risk for lung disease are unavailable". Respiratory symptoms and small airway functional tests therefore are not necessarily indicators of adverse effects or pathological processes.
That smokers show abnormal lung function has not been uniformly observed. In addition to a single study mentioned in the Surgeon General's Report, there are five other publications during the late 1970's that were not cited (29-33).

b. Clearance Mechanisms. The discussion of mucociliary transport (page 6-32) is another example of selective citations from the literature showing negative aspects of cigarette smoking, and omission of conflicting results. Articles that show smoking accelerates bronchial clearance in humans have been omitted. The studies of Albert et al. on accelerated clearance in donkeys have been cited, but their article on human clearance stimulation in smokers (34) has been omitted, thus giving readers the impression that the phenomenon has only been seen in donkeys.

Camner, who pioneered in clearance studies in man and whose technique has been adapted by many clinical pulmonary physiologists, has been overlooked. The basic concept that has evolved from Camner's work is that adrenergic stimulation increases mucociliary transport (35), an effect to be expected from cigarette smoking. Patients with predominant pulmonary emphysema are reported to have no impairment of mucociliary transport (36). Therefore, it is unlikely that smoking, by
accelerating airway clearance, would contribute to the pathogenesis of emphysema.

a. Proteolytic lung damage. The hypothesis that cigarette smoke causes a protease-antiprotease imbalance and in turn potentially leads to pulmonary emphysema is based on a group of selected observations. There is no discussion as to why the hypothesis has not been generally accepted. Turino (37), the first researcher to show that proteolytic enzymes influence the pulmonary mechanics in vivo, has subsequently noted flaws in the hypothesis that emphysema is caused by the imbalance of protease and antiprotease:

"An inconsistency for the primary role of elastin alteration as a mechanism for inducing pulmonary emphysema has been a failure to demonstrate alterations in elastin content or composition in human emphysema. Morphologically, elastin fibers have appeared disrupted and diminished, yet most data from human lungs (Johnson and Andres 1970; Pierce and Bocott 1960; and Pierce et al. 1961) with the exception of that of Briscoe and Lorinc (1958), have shown no reduction of parenchymal elastin content by gravimetric techniques."
The above comments can be reinforced by the 1976 observations of Ruhn et al. (38) that hamsters treated with elastase intratracheally and developing emphysema also show normal elastin content. Even lung fluid samples collected from smokers and nonsmokers showed no difference in proteolytic enzyme content (38a).

C. EPIDEMIOLOGIC STUDIES

Increased mortality from pulmonary emphysema among smokers compared to nonsmokers is suggested by seven prospective studies summarized in the 1979 Report (page 6-10). However, these studies are far from being consistent and tend to suggest that many risk factors including cigarette smoking need to be considered when studying the etiology of pulmonary emphysema.

a. Air Pollution. The role of air pollution as a risk factor in pulmonary emphysema has been the subject of several publications not cited in the 1979 Report. Studies in Tucson, Arizona (39) and in Busselton, Australia (40) noted that an urban environment may contribute to the normal increase in closing volume with age, and to the incidence of respiratory symptoms. The higher levels of pollution in England than in
the United States have been cited as a cause of the lower ventilatory functional measurements among British males (41).

h. Socioeconomic Status. The illustrative table presented in the 1979 Report (page 6-39) is based on a survey by Higgins of residents of Tecumseh, Michigan. The overall prevalence of chronic obstructive lung disease for various occupational groups ranged from 12.3 for professionals and managers to 30.0 for laborers. An uncited 1977 survey by Lebowitz (42) is more accurate because the diagnosis of pulmonary emphysema and chronic bronchitis was confirmed by medical examination. His most important conclusion is that smokers do not have a higher incidence of chronic obstructive lung diseases than nonsmokers or ex-smokers when the adults were grouped according to income and educational backgrounds. Education and income were inversely related to prevalence of chronic obstructive lung diseases even after controlling for sex, age and smoking.

c. Alpha-1-antitrypsin deficiency. The summary statement in the 1979 Report that individuals with severe deficiency have the onset of symptomatic pulmonary emphysema probably "abbreviated by smoking" was based on selected citations. Two publications in Chapter 5 were not accurately quoted. Chan-Yuang, et al and Morse et al did not only examine
subjects with mild deficiency but also individuals with severe antitrypsin deficiency who did not deteriorate with smoking. Kidokoro et al. (43) concluded in their study of subjects with severe deficiency that "variables other than age and cigarette smoking influence the severity of emphysema."

4. Other genetic and familial factors. The limited discussion in the 1979 Report needs expansion due to increasing evidence of familial aggregation of pulmonary emphysema. The studies of familial prevalence of lung disease conducted by Cohen et al. clearly indicate that there is a familial factor unrelated to cigarette smoking and antitrypsin deficiency that is involved in pulmonary emphysema (44).

Familial aggregation of chronic obstructive lung disease independent of cigarette smoking, sex, respiratory illness history, residence in common household, geographical distribution and antitrypsin variants has also been reported by Tager et al. (45) in 1978. There has also been revived interest in the concept of a congenital or developmental etiology of pulmonary emphysema (46). Additional research directed at the basic cause of pulmonary emphysema may lead to the identification of additional genetic factors that result in developmental abnormalities leading to adult form of this disease.
(e) Infections. A recent report on the Tucson study reveals that acute infection may play an independent role in the pathogenesis of chronic obstructive lung disease (47). For serologic infection rates of three viruses and H. influenzae in males, the incidence of bronchitis is higher for non-smokers (23%) than in smokers (9.8%). Lebowitz and Burrows (48) arrived at a similar conclusion, i.e. acute respiratory illness is a major risk factor in the etiology of chronic obstructive lung disease.

(f) Alcohol consumption. Since 1977, there have been several reports that consumption of alcohol is associated with higher incidence of chronic obstructive lung disease (49-51). In the Tucson study, Lebowitz (52) has concluded that alcoholism is an independent risk factor for chronic obstructive lung disease. There are also earlier observations that alcohol feeding in rats causes pulmonary cellular changes, depression of immunologic reaction, and increased susceptibility to infection (53-55).

6. Carbon monoxide content of cigarette smoke

The proposed labeling of cigarette packages which discloses carbon monoxide yield is not based on definitive evidence that the concentration of this chemical in cigarette
smoke influences the incidence of cigarette-associated diseases. In the contrary, there is recent information that disproves the assumption that the amount of carbon monoxide is pathogenic. The collected data of an epidemiologic study on smoking habits and health of 18,403 males initiated in the 1960s, were recently analyzed to determine any influence of carbon monoxide yield of cigarettes. Borland et al. (56) reported that forced expiratory volume (FEV-1) was not influenced by the yield of carbon monoxide. The mean levels of FEV-1, age adjusted, were as follows:

2.66 liters for subjects who smoked cigarettes with less than 18 mg. carbon monoxide yield/cigarette.
3.04 liters for subjects who smoked cigarettes with 18 to 20 mg carbon monoxide yield/cigarette.
3.09 liters for subjects who smoked cigarettes with greater than 20 mg. carbon monoxide yield/cigarette.

As the carbon monoxide yield of cigarettes increased, there was no corresponding significant change in measurements of airflow resistance. Borland et al. (56) concluded that carbon monoxide content in cigarettes has no influence on measurements of airflow obstruction that signify pulmonary emphysema. Their results suggest that carbon monoxide content of cigarettes does
not influence mortality from coronary heart disease. A review by Weir and Páháno (37) is consistent with these recent findings of Borland et al (38) as they relate to cardiovascular disease.

D. CONCLUDING REMARKS

It is the opinion of the author of this submission that cigarette smoking is not the primary cause of pulmonary emphysema. This opinion is based on the author's own research studies and his interpretation of literature on the subject. It is clear that additional research is needed to determine which of the different risk factors for pulmonary emphysema are important in a causal sense.

Domingo M. Ariado, M.D.
April 20, 1983
P. BIBLIOGRAPHY


34. Albert AW, Peterson WT Jr., Bohnig DE, Lipmann MW: Short-term effects of cigarette smoking on bronchial...


STATEMENT OF RICHARD J. BING, M.D.

Concerning Senate Bill S.772

I, Richard J. Bing, M.D., am currently Professor of Medicine Emeritus at the University of Southern California, and Director of Experimental Cardiology at the Huntington Medical Research Institutes in Pasadena and Visiting Associate at the California Institute of Technology in Pasadena.

My scientific career commenced at the Rockefeller Institute in New York where I worked with Charles Lindbergh and Alexis Carrel on the first artificial heart to keep organs alive outside the body. Subsequently, I worked at Columbia and New York Universities, at the Johns Hopkins Hospital, the University of Alabama, Washington University and as Chairman of the Department of Medicine at Wayne State University in Detroit. I have been a member of the Scientific Advisory Board of the Council for Tobacco Research since the early 1950's.
I am the recipient of the American Heart Association Research Achievement Award for 1974, and of the Mitchell Foundation Award, Washington, D.C., 1970. I am Honorary Life President of the International Society for Heart Research.

It has become obvious during the last 40 years that many dread diseases are multifaceted disturbances, for which a common denominator does not exist. Rather a whole variety of factors appears to be responsible, such as genetic and environmental. In contrast to most infectious diseases, in which a common denominator, usually a known bacterial strain, is responsible, most dread diseases originate and develop from multiple causes. A typical example is coronary artery disease, which is in the field of this writer's expertise. To name just a few known factors, which may play a role in its etiology, there are the so called risk factors, such as diabetes, hypertension, disturbances in blood lipids, and genetic factors. Equally important may be disturbed function of blood
Platelets, genetic susceptibility to atherosclerosis, and alterations in properties of endothelial cells of blood vessels. Recently spasm of the coronary arteries has been added as a factor. The cause of arterial spasm is not known. These are just a few of the mechanisms which may constitute the basis for development of coronary artery disease. From a scientific point of view it would therefore be unwise to ascribe a simple cause to this multifaceted disorder.

We have learned over the years that each of these risk factors has a multiplicity of underlying causes, many of them not yet fully understood. Diabetes is an example; we know that this condition results in the development of coronary artery disease. Yet we do not even comprehend the etiology of diabetes. It is a mosaic of genetic, immunological and multiple endocrine factors. Equally we still do not fully understand why diabetes causes coronary artery disease.
A similar situation also applies to hypertension. This condition also represents a spectrum of many different causes: genetic, endocrine, renal. Consequently there exist many and different types of hypertension. We do not, as yet, comprehend the link between high blood pressure and coronary artery disease. Is it increased uptake of lipids by the arterial wall, or damage to the arterial wall which is responsible, just to mention a few examples.

With regard to cigarettes, in normal individuals smoking causes an increase in coronary flow, heart rate, and blood pressure. In patients with coronary heart disease the effect on blood pressure and the heart rate is more pronounced but the rise in coronary blood flow is less. There is no experimental proof that smoking in patients with healthy coronary arteries adversely affects the heart.

Likewise, there is no experimental proof that smoking or nicotine causes hardening of the coronary arteries. For
example, carbon monoxide (CO), an important constituent of tobacco smoke, when given to monkeys for 12 months, does not result in coronary artery disease. (We gave 200 to 400 ppa's of carbon monoxide (CO) in the inspired air for 10 alternate half hours daily for 12 months.) In short, in my opinion, there is no experimental link between the production of hardening of the coronary arteries (coronary atherosclerosis) and smoking.

I have restricted my remarks primarily to experimental and clinical observations. The purported "findings" set forth in S. 772 with regard to cardiovascular disease stem primarily from some but not all epidemiological studies. Yet, there is no experimental proof that smoking causes atherosclerosis, coronary artery disease or congestive heart failure. It is essential to keep in mind the difference between the epidemiological approach and the experimental and clinical approach. Epidemiological studies rely on
questionnaire or death certificate data, which in many cases are inaccurate. Patients who fill out questionnaires are not always aware of the nature of the disease and fail to give accurate information. Data from death certificates may also be unreliable. Furthermore, epidemiological studies involve people who have already decided for themselves whether or not to smoke, i.e., they are a self-selected population. In contrast, the experimental approach is scientific, accurate, and based on controlled observations. It is through this latter approach that an understanding of the causation and pathogenesis of cardiovascular disease will be achieved.

If anything has been learned from both basic and applied research carried out over the last 30 years, it is that we must beware of oversimplification. It is simpler and more convenient to deny the multiplicity of factors in the causation of many of the dread diseases. But this attitude is not
scientific. Oversimplification has never led to the recognition of scientific truth.

The thought I wish to convey is that as a scientist and physician I have learned to beware of oversimplification. Only an in depth investigation of a disease such as coronary artery disease can bring us closer to the truth. As a physician and scientist I feel that the Scientific Advisory Board of the Council for Tobacco Research U.S.A., Inc. has recognized the varied spectrum of biological origins of disease. The Board has justly focused on the mosaic of biological factors underlying various diseases. The members of the Board have been motivated by strict scientific considerations. Thus, efforts have been made over the years to study the underlying mechanisms of coronary artery disease and to investigate them by a variety of strictly scientific approaches. One fundamental fact has emerged from these studies: there is a wide spectrum of underlying disturbances of the cardiovascular system and only a multiplicity of approaches will bring us closer to the scientific truth. There exist no convenient solutions in fundamental and applied biology.
My name is Walter M. Booker. I am President of Walter M. Booker and Associates, Inc., an incorporated biomedical group in Washington, D. C. I am also Professor Emeritus of Pharmacology at Howard University, where I was Chairman of the Department of Pharmacology for 20 years.

My postgraduate professional training consists of a Master's Degree in Cellular Physiology and Biochemistry from the University of Iowa and a Ph.D. in Physiology and Pharmacology from the University of Chicago. I also spent a postgraduate year as a Senior Fulbright Scholar in Belgium and Sweden, studying with two Nobel Laureates.

I hold membership in numerous scientific societies including the American Society of Pharmacology and Experimental Therapeutics, the American Physiology Society, the American College of Clinical Pharmacology, and the American College of Cardiology of which I am a Fellow.
The views I am expressing in this statement are based upon my training and experience as a scientific researcher in the fields of pharmacology and physiology. In these areas my research endeavors included studies on the effects and mechanisms of action of nicotine on the heart and the autonomic (sympathetic) nervous system.

My main concern with Bill S.772 is that it proposes congressional action based on findings that have not been clearly established. It is important to note that the Bill makes such summary statements as: "Smoking is the primary cause of lung cancer and emphysema", and "one-third of heart disease deaths are associated with smoking". While such opinions are undoubtedly based on some published information, there is by no means unanimity in the scientific community that cigarette smoking can be incontrovertibly labeled as causal.

Despite what those in the legislative arena might believe, the cause or causes of cancer of the lung (and other organs) remain unknown. Both smokers and nonsmokers contract cancer and other diseases often associated with cigarette smoking, and we still don’t know why.
The most accurate and appropriate statement that can be made is that cigarette smoking has been identified as one of the many risks or associated factors that may cause or aggravate certain commonly occurring diseases. We are still not able to say whether all relevant risk factors and associations have been identified and consequently, it is practically impossible to control for their effects in experimental studies. While attempts have been made to evaluate certain factors individually, research has not reached the point of being able to say a particular factor is causative.

The scientific fact of the matter is that no one knows why someone like an industrial worker develops cancer of the lung. Is it because he or she is a smoker or because he or she is an industrial worker? Should cigarette smoking be used as a 'scapegoat' or should meaningful efforts be made to afford protection to the industrial worker?

Having devoted a large part of my research career to functional underlying processes that might contribute to certain diseases of the heart, I am particularly concerned with that part of the Bill that seems to ask Congress to decide what specifically causes coronary artery disease. In all sincerity, I raise the question: If smoking has been scientifically shown to be a cause of heart disease, what are the established mechanisms?
As a research scientist, I believe that such mechanistic data are necessary before conclusions on causation are possible.

In addition, the results of the MRFIT study, the Multiple Risk Factor Intervention Trial, sponsored by the National Heart, Lung and Blood Institute, lend support to my contention that much more research is required if we are to understand the causes of heart disease. This study has cast considerable doubt on the validity of the assumption that reducing levels of classical risk factors including smoking lowers the death rate from heart disease. It may be time to take a fresh look at questions of heart disease causation and pursue other avenues of research.

Diet, whether deficiencies or excesses, lack of exercise, emotional stress, genetic factors, auto-immune factors (particularly where cancer is concerned) must each bear its responsibility as a possible cause of disease; but not one of these can be given sufficient responsibility to justify congressional action of the type contained in the Bill being discussed here today.

In summary, I strongly disagree with the proposed Bill because it misrepresents the present state of scientific knowledge. The Bill is asking Congress to give the Nation a scientific verdict on the causes of complex diseases when much of the evidence is either conflicting or has yet to be presented.
My name is Victor Buhler and I am a pathologist living in Kansas City, Missouri. I am certified by the American Board of Pathology in both pathologic anatomy and clinical pathology. I have held faculty appointments at the University of Kansas School of Medicine and the University of Missouri-Kansas City School of Medicine. I have served as the President of the College of American Pathologists, the Missouri State Medical Association, the Missouri Society of Pathologists and the Kansas City Society of Pathologists. I am a Fellow of the American Society of Clinical Pathologists, the American College of Physicians, the American College of Pathologists, and a member of the International Academy of Pathology and the American Association for the Advancement of Science. I participated upon invitation by President Johnson in the White House Conference on Health in 1965.

At the present time, I am associated with a group of pathologists serving one major hospital and several community hospitals in the Kansas City area, with primary responsibility for the Liberty Hospital, Liberty, Missouri.

I have become familiar over the years with many of the articles cited in the various Surgeon General's reports on smoking
and health and with the general medical literature pertaining to this subject. I testified before the Committee on Interstate and Foreign Commerce in 1969 in regard to proposed legislation concerning cigarette labeling and advertising. At that time, I told the Committee that my knowledge of the medical literature, my own investigations and my laboratory experience established "my firm conviction that neither cigarette smoking nor any other etiological agent has been shown to be the cause of cancer of the lung." I have seen no research reports in the intervening 14 years which would change that view. I am pleased to have the opportunity to give you my views on the present legislative proposals embodied in S. 772.

I am most disturbed by the legislative finding included in this proposal that "smoking is the primary cause of lung cancer." I am also concerned by what is called a "Public Awareness Statement" that "Cigarette smoking causes lung cancer." Such statements are, in my view, not supportable by the pathological and clinical observations I have made during my practice, nor by my review of the medical literature. I am disturbed to see such findings and statements in proposed federal legislation because I believe they are deceptive and misleading to the public and the medical community. The pressure to find causes and cures of cancer and other chronic diseases is considerable, as it should be. However, this pressure,
It is always important to realize that objectivity is vital to scientific progress. The cause or causes of lung cancer are unknown, and a Congressional finding to the contrary does not alter that situation. Our only hope for progress in the fight against this dread disease is in innovative medical research, not legislative pronouncements.

My view that cigarette smoking has not been proven to be a cause of lung cancer is based on my personal knowledge of the sources for the epidemiological evidence frequently used to support this theory. Epidemiological studies, for the most part, are based on mortality data derived from statements of physicians or others who sign death certificates. In approximately 80 percent of deaths, no post-mortem examinations are done to verify the information on the death certificate regarding the primary or underlying causes of death. Even when autopsies are performed, the results often are not available until after the death certificates have been completed. The recorded causes of death, then, are primarily based on clinical diagnoses, and it has been my experience as a practicing pathologist that these are frequently incorrect. In view of these factors, it must be understood that, at the present time, 80 percent or more of all death certificates cannot be considered as having been scientifically validated. Consequently, I have grave doubts about the statistical associations which have been derived from data which are subject to serious flaws.
In addition, the data in statistical studies of lung cancer do not distinguish between primary and secondary lung cancers. Primary lung cancer is a malignant tumor originating in the lung. Secondary lung cancer, often called metastatic, is cancer that has spread to the lung from another body site. I have all too frequently found in my own work that tumors clinically considered as primary in the lung are often metastatic and result from cancerous growths in other parts of the body.

One of the reasons why primary lung cancer may be frequently diagnosed when it is not present is that physicians depend largely on chest x-rays for diagnostic purposes. In this regard, it should be noted that chest x-rays present fewer problems in interpretation than x-rays of any other organ or part of the body except the skeletal system. This is because the air in the lungs provides an excellent contrast medium so that disease states are more readily detected. We find, therefore, that it is easier to observe on x-ray an abnormality in the chest than, for example, in the liver. Even then, however, one cannot always tell from an x-ray whether an area of density in the lung is a nonmalignant or malignant growth, and, if malignant, whether or not it originated in the lung.

There are, of course, techniques other than x-rays for diagnosing lung cancer. Biopsy and sophisticated scanning techniques
(CAT scan), two other routinely used methods, do not always allow for distinguishing between secondary and primary tumors. It should be stressed that primary lung cancer is simulated by secondary tumors arising from other organs of the body with sufficient frequency to challenge the diagnostic accuracy of many deaths certified as primary lung cancer without autopsy exclusion of other primary sites.

In my judgment, the failure of epidemiological studies to distinguish between primary and secondary lung cancer in their statistical analyses raises serious questions about causal interpretations of statistical associations.

Some advocates of the smoking causation hypothesis have said that lung tissue from smokers exhibits certain abnormalities that they describe as "pre-cancerous." They conclude that cigarette smoking causes these changes which eventually will lead to lung cancer. Let me say, first of all, based on my examination of thousands of lungs in microscopic detail, that no one can determine whether or not a lesion described as "pre-cancerous" will progress into cancer.

Metaplasia (changes of one type of tissue to another type), hyperplasia (increases in cell numbers, and dysplasia
(atypical cytologic changes in cells) can be seen in lungs of both smokers and non-smokers. These changes are rather common and should not be considered as pre-cancerous. In fact, the weight of scientific evidence is that these lesions will not progress to cancer. Metaplasia, common in older individuals, occurs frequently in the trachea or windpipe, and yet tracheal cancer is extremely rare. Any study of autopsy cases that suggests that metaplasia, hyperplasia, or dysplasia provides evidence that smoking leads to lung cancer oversteps scientific bounds. Based on my own experience, I cannot confirm that significant cell or tissue changes in the lung occur more frequently in smokers than in non-smokers. Certainly, there are smokers without changes, and non-smokers with changes.

As a pathologist, I find arguments that rely on "pre-cancerous" lesions to indict smoking as a cause of lung cancer to be unconvincing and indeed inconsistent with my own clinical observations.

I might add that, contrary to the reports that one often hears, it is impossible for the pathologist to determine from both gross and microscopic examination of lung tissue whether a person does or does not smoke.

I am equally unconvinced by the evidence from animal experiments which some consider to be confirmation of the causal
In March of this year, for example, several government witnesses testifying before a U.S. House Subcommittee referred to a "beagle dog study" as demonstrating the production of human-type squamous cell lung cancer in test animals by tobacco smoke inhalation. It was asserted that this study substantiated the claim of a causal connection between smoking and lung cancer.

I am very familiar with this study because I analyzed it extensively after its publication over ten years ago by Dr. Oscar Auerbach, et al., in The Archives of Environmental Medicine. The results of my critique of its many scientific shortcomings are contained in a letter to the Subcommittee which I prepared at the request of one of the members. As I stated in the letter, I view references to the study, such as those made by the government witnesses, to be scientifically unjustified and, consequently, misleading. The study has been severely criticized in the scientific literature and, to my knowledge, is no longer relied on or even cited by serious researchers.

Again, I must emphasize that the Auerbach, et al., study should not be relied on as support for the claim that squamous cell lung cancer has been produced in test animals by inhalation of tobacco smoke.

Based on my years of experience as a pathologist and on my reviews of the smoking and health literature, I must disagree strongly with the proposed Congressional finding that cigarette smoking is the primary cause of lung cancer and with the government determination that cigarette smoking "causes" lung cancer.

Victor B. Euhler, M.D.
Statement of Professor Hans J. Eysenck Regarding S. 772

I am Hans J. Eysenck, professor of psychology at the Institute of Psychiatry, University of London, and psychologist to the Maudsley and Bethlem Royal hospitals in London.

I received my Ph.D. in 1940 and my D.Sc. in 1964, both from the University of London. I was Senior Research Psychologist at Mill Hill Emergency Hospital from 1942 through 1946. In 1949 and 1950, I was a visiting professor at the University of Pennsylvania in Philadelphia. Between 1950 and 1954, I was a Reader in Psychology at the University of London's Institute of Psychiatry. In 1954, I was a visiting professor at the University of California at Berkeley.

I am a Fellow of both the British Psychological Society and of the American Psychological Association.

I have founded and edited three psychological journals, and I am on the editorial boards of some 15 other international psychological journals. I have written or edited
for publication approximately 35 technical books and over 600 articles dealing with various aspects of the psychological field, particularly with respect to personality, intelligence, behaviour therapy and behavioural genetics. I have conducted research in the area of smoking for over 20 years and have authored two books, the most recent of which is entitled *The Causes and Effects of Smoking*, as well as numerous articles on this subject. In my book I have been concerned with an examination of the two major theories of the relationship between cigarette smoking and disease, in particular lung cancer and coronary heart disease. The *causal* theory asserts that cigarette smoking is in part responsible for these diseases, thus going beyond reports of a statistical relationship between smoking and disease. An *alternative* theory suggests that the observed correlations can be explained in terms of common genetic factors, underlying both the propensity to smoke and the probability of developing certain
diseases. My own work has been concerned mainly with the issue of the origins and the maintenance of the smoking habit, because these questions are vital to an understanding of the evidence concerning these two theories.

Working together with Professor Lindon Eaves, a geneticist formerly of Oxford University, and now at Richmond, Virginia, I administered special smoking and personality questionnaires to various samples of the population, including over 1,000 identical and fraternal twins, 340 fostered children and 230 pairs of foster parents; as well as a variety of familial relationships (parents and children, grandparents and grandchildren, uncles and aunts, nephews and nieces, first cousins, etc.), making a total of 2,469 individuals. Appropriate statistical analyses were made of the data and in many cases new statistical methods had to be developed for this purpose. The major principle used was to set up specific theories or models, and test these against the empirical data.
increasing the complexity and sophistication of the model until an acceptable fit was reached.

As regards the onset of smoking, genetic factors seemed to play little, if any part. The data support the hypothesis that taking up smoking was largely due to environmental factors, particularly the influence of peers; this factor emerged as far more important than other factors traditionally cited, such as the smoking habits of parents, which we found to exert little influence on the origins of the smoking habit. Equally, the results are not readily compatible with a theory which would assign advertising any marked influence on the taking up of smoking. These results have been verified by direct studies undertaken by Professor C. Spielberger of the University of South Florida; he found that social pressures, particularly peer influence, exerted a very strong effect on young people taking up smoking, whereas the influence of advertising was negligible.
As regards the maintenance of the smoking habit, our data indicate the powerful influence of genetic factors. People who give up smoking are intermediate between those who have never smoked and those who persist in smoking; this finding is important as it demonstrates that giving up smoking is in part determined by genetic factors, so that people who give up, and those who continue, are different types of people genetically. As regards the amount of tobacco consumed, it seems that the genetic factors determining this are rather different from those which influence a person's becoming, or not becoming, a smoker; non-smokers are differentiated genetically from smokers along quite a distinct dimension from that which discriminates between the different degrees of cigarette consumption among smokers. On the whole, we thus find little evidence of genetic determination for the taking up of the smoking habit, which seems to be much more closely related to peer influence; we find strong evidence of genetic
determination for the continuation of smoking, and for giving up smoking, and we find strong evidence of genetic determination for how much a person will smoke.

These data are relevant to the proper evaluation of the claims of the causal and the genetic theories concerning the relation between smoking and disease. A widely accepted theory asserts that cigarette smoking causes lung cancer, coronary heart disease, and other diseases with which it is statistically linked. It is not always realized that (a) such a theory is far from proven, and is beset by many anomalies and doubts, and that (b) there is an alternative theory which is based on undeniable facts which are not explained by the causal theory. The present position seems to be that either theory may explain the incidence of lung cancer and coronary heart disease (to which this brief account will be restricted) or that both may be needed to complement each other.
Statements in S. 772 such as that cigarette smoking is associated with the unnecessary deaths of over 300,000 Americans annually, or that one third of the deaths attributed to cardiovascular disease are associated with smoking, or that cigarette smoking causes lung cancer, and is a major cause of heart disease, as stated in the proposed warning, are not scientifically valid, and represent arbitrary extrapolations from a very doubtful data base. The Multiple Risk Factor Intervention Trial, the results of which were published in September 1982, illustrates how difficult it is to make such extrapolations, and may also serve to demonstrate the dangers of doing so. In this "randomised primary prevention trial" to test the effect of a multifactor intervention programme on mortality from coronary heart disease, men were randomly assigned either to a special intervention programme consisting of step-care treatment for hypertension, counselling for cigarette smoking, and dietary advice for lowering blood
cholesterol levels, or to the usual sources of health care in the community. Risk factor levels declined to a greater extent in the special intervention group, but there was no significant difference in the number of deaths due to heart disease per 1,000 in the two groups, and indeed the total mortality rate was higher in the intervention group than in the control group. Thus a decrease in smoking, a decrease in blood cholesterol, and a lowering of hypertension failed to have the confidently expected outcome, showing that extrapolation of this kind cannot as yet be regarded as scientifically permissible.

There is agreement that smoking is neither a necessary nor a sufficient cause of lung cancer. Of 100 heavy smokers, less than 10 will develop lung cancer; hence smoking is not a sufficient cause. And of 100 people who develop lung cancer, approximately 10 will be non-smokers; hence smoking is not a necessary cause. This simple fact (the precise numbers differ
of course from country to country, but indicate the correct
order of magnitude) suggests that the scientific proof for any
particular theory will be difficult to arrive at, and that any
such theory will almost certainly be complex and multi-faceted.

Much of the evidence cited in favour of the causal
theory is statistical, but many statisticians have severely
criticized the evidence on statistical grounds. Such suggested
proofs as the correlation between smoking and lung cancer
within a given country are evidence of correlation, not of
causation: one of the first lessons the budding statistician
learns is that correlation does not imply causation. (There is
a very high correlation between countries linking meat eating
and cancer of the large intestine, yet we do not conclude that
eating meat causes cancer of the large intestine!) Hence this
method of investigation, while suggestive, is far from
compelling. This would be so even if the figures usually
quoted could be taken as accurate; however, there are good
reasons for doubting their accuracy.

The figures quoted are based on clinical diagnosis of lung cancer, but these are very unreliable and imprecise. If we take as our criterion autopsy data, and compare these routine diagnoses, we find that prior to World War I, out of 100 people found on autopsy to have died of lung cancer, only 3 were so diagnosed. This is typical of the very obvious under-diagnosis of lung cancer then prevalent. In recent years, exactly the opposite has been found, namely an over-diagnosis of lung cancer of up to 200% and more! Whether these changes in diagnostic preference are completely responsible for the alleged tremendous increase in lung cancer over the years or not, and whether they may in part account for the observed correlation between lung cancer and smoking, it is impossible to say; all we can say is that with the basic data so completely unreliable, the statistics based on them are suspect.
Another very important point concerns the isolation of smoking from other, correlated habits, such as drinking, living it up, staying out late, wenching, etc., i.e. a certain style of life the totality of which may increase the "rate of living", so that smokers are biologically older than non-smokers at a given age, for reasons only partly involved with smoking. Non-smokers are different types of persons from smokers, are generally more self-protective, and the personality traits and habits thus linked with non-smoking may be more relevant to the longevity of non-smokers than their refusal to smoke.

It is often suggested that sex differences, with males showing more lung cancer, are the product of the tendency of males in the past 50 years or so to smoke more. However, as several authorities whom I quote in my book have pointed out, sex differences of the same kind as those observed now were found before cigarette smoking became popular. Again, it is
found that changes in the rate of increase of lung cancer diagnosis occurred simultaneously for men and women, although the women, who took up smoking much later than men, should have shown these changes at a much later date than men.

If the causal theory is true, then we would expect a definite dose-response relationship; in other words, the heavy smoker should be stricken with cancer earlier than the light smoker. Yet the amount smoked makes no appreciable difference to the mean age at which the person is reported first to the clinic. Again, inhalation should make lung cancer much more likely than smoking without inhaling; yet the figures show if anything an opposite trend. Indeed, in the most recent study (published in 1982 in the Journal of Epidemiology and Community Health) lung cancer rates were higher overall for non-inhaling, particularly in groups of heavy smokers. These two observations are difficult to reconcile with the causal theory of smoking.
What is often claimed to be the most impressive evidence for the causal theory has been the report that physicians who gave up smoking showed less lung cancer than members of the general public who continued to smoke. Thus, it might appear that giving up smoking has saved the lives of those who did so. But this proof is only acceptable if those who continue to smoke, and those who later on give up smoking, are essentially identical with respect to their health before some of them gave up smoking. Clearly, if those who later on give up smoking are already much healthier than those who later on continue to smoke, then the final differences in health may be due to the already existing differences before anyone gave up smoking, rather than to the cessation of this habit. But there is good evidence to show that smokers and ex-smokers already differed with respect to their health record before the ex-smokers gave up smoking. Similarly, there is evidence that from the point of view of personality and genetics ex-smokers
are different from continuing smokers. Thus this alleged proof is based on an erroneous assumption.

These objections to the causal theory, and others made in my book, do not prove the theory to be wrong; they simply argue that it is still only a theory, not a scientific law. More convincing proof is required before the theory can be accorded a more advanced status. But further than that, there are numerous facts suggesting an alternative theory, and these facts cannot easily be integrated with the causal theory. Yet a proper theory demands that attention be paid to all relevant facts, and thus again the causal theory is found wanting.

The alternative theory, first suggested by the eminent geneticist and statistician Sir Ronald Fisher, suggests that genetic factors are important in causing lung cancer; that genetic factors are active in causing people to maintain the smoking habit, and that possibly the same genetic factors may be involved in both these trends, thus producing the observed...
correlation between smoking and cancer (insofar as such a correlation is real). There is evidence that genetic factors play a part in the causation of lung cancer; this is not in doubt. As already mentioned, I have brought forward evidence (in addition to already very convincing evidence produced by many other people) to show that genetic factors are relevant to the maintenance of the smoking habit. Thus there is evidence for both the assumptions on which Fisher's argument was based.

My own contribution has been to suggest that the mediating factor between cancer and smoking may be the personality of the people involved. Thus it is assumed that people of a certain personality are more likely than others to die of lung cancer irrespective of smoking. It is also assumed that people of a certain personality are more likely to smoke than others. There is evidence for both these propositions. My original work with Dr. Kissen, an eminent British oncologist, showed very marked personality differences between
lung cancer patients and patients suffering from non-malignant tumours, with the personality assessment made before diagnosis. Since then, a large-scale study in East Germany has replicated our findings (themselves replicated in another study by Kissen), and has found similar personality traits to those characteristic of lung cancer patients in women with cancer of the breast. Other studies, also indicating a relation between lung cancer and personality, are cited in my book.

In a similar way, my early work with Tarrant and Wodif established a correlation between personality and smoking, and many studies in different countries have since confirmed our findings, and added new ones. We may thus say that the fundamental assumptions of Fisher's genetic theory have found empirical support, and we may add that there is also some modest support for my own attempt to integrate these two major fields. Unfortunately there has been too little work along these unusual and somewhat unorthodox lines to say that the
results are anything more than suggestive, and the theory linking them is still in a very elementary stage; nevertheless, as far as the findings go they support the genetic rather than the causal theory, although they do not necessarily contradict the latter. It seems unfortunate that the premature crystallization of spurious orthodoxies has prevented the genetic theory from attracting sufficient research grants to work it out in sufficient detail, and to carry out the research necessary to put it on a more acceptable footing.

Recently some progress has been made on the theoretical development of the genetic hypothesis by linking it with research on stress, in particular the differential effects of chronic and acute stress, and the 'inoculation' theory of stress. However, in the absence of large-scale research into the refinements of this theory, and more widespread familiarity with and criticisms of its details, not too much should be claimed for it other than that it presents a viable alternative
In relation to the causal theories of coronary heart disease (CHD), similar criticisms apply as do in the case of lung cancer. There are considerable unreliabilities in diagnosis; there are large numbers of factors other than smoking which have been associated and which are not usually controlled for in studies of the effects of smoking; inhalers do not on the whole differ from non-inhalers in disease proneness; the statistical relation between cigarette smoking and CHD disappears in many countries, e.g., Finland, Holland, Yugoslavia, Italy, Greece and Japan; there is an absence of dose-response relationship, i.e., there is little or no relation between duration of heavy cigarette smoking and risk of myocardial infarction; and the correlation between number of cigarettes smoked and CHD is not linear; ex-smokers in some studies appear to be safer than non-smokers; some types of CHD, such as angina pectoris (which comprises some 20% of CHD),
men) fail to show even a statistical correlation with cigarette smoking; some types of smoking (cigar, pipe) fail to show even a statistical correlation with CHD; etc.

In the most recent study from the Mayo Clinic (published in the May/June 1982 issue of Atherosclerosis), over 15,000 patients with coronary artery disease, proven by arteriography, were studied. No positive correlation (indeed, in some subgroups a negative correlation) occurred between the arteriographic measures of disease and the cigarette smoking history (ever or never, number of pack-years of smoking, duration of cigarette smoking, and peak daily cigarette consumption). These are anomalies or failures of the causal theory which demand an explanation before the causal theory can be accepted. Some of these facts are much more readily explained in terms of a genetic-personality theory; thus the differential effects of cigarette vs. pipe/cigar smoking may find an explanation in terms of the known differences in
personality type associated with these different smoking patterns.

The general conclusion would seem to be that in the case of CHD, as in the case of lung cancer, proof for the causal influence of smoking is still lacking. Such evidence as exists is by no means as clear-cut and decisive as is often alleged. There is evidence in the case of CHD for genetic factors, and there are published correlations with personality; here too there appears an important element of stress determining the appearance of CHD, and stress is intimately linked with personality. No formal theory of genetic determination of CHD has yet been put forward, but it seems likely that such a theory is needed as an alternative (or perhaps as complementary) to the causal theory for an explanation of the many gaps and anomalies in the latter.

It is interesting to note that if one were to use the same statistical type of argument of correlation as favouring a
causal relation, smoking would seem to protect against certain illnesses. Thus colorectal cancer in women is found significantly more frequently in non-smokers; so are primary central nervous system neoplasms, Parkinson's Disease, trigeminal neuralgia, diabetes and ulcerative colitis. There are other curious relationships between diseases which the smoking causes cancer hypothesis does not begin to explain; one of them is the almost complete absence of lung cancer among schizophrenics, in spite of the high degree of cigarette smoking prevalent in that group.

One important function of the genetic theory has been that of explaining the reasons why people smoke, and to link these reasons with their differential personality patterns. Another important function of the genetic theory has been to suggest better designs for research in this complex field. A good example is the use of the discordant twin method by Cederlof, Lundman and others, i.e. the investigation of the...
illness patterns of identical twins of whom one smokes, the other not. If this type of research had been carried out on the large and international scale required, instead of investing in the redundant and scientifically not very valuable replication of correlational studies, we would know far more about the relation between smoking and disease than we do now. Such studies allow us to look at environmental factors, including those of smoking, while controlling for genetic factors; this is essential if any convincing results are to be achieved.

In summary, I would like to state that the causal theory of smoking as being responsible for lung cancer and coronary heart disease, while it has found many supporters, is far from being established, and has many gaps, anomalies and contrary findings to contend with; these are too frequently glossed over and dismissed as unimportant, when in reality they may be found to discredit the causal theory in whole or in
part. An alternative theory, based on genetics and implicating personality factors, is much less well developed, more complex, and at present not too well known to oncologists; nevertheless there are many well-established facts which suggest that in part if not in whole it can account for the major findings. At the very least, this alternative theory suggests novel research methodologies which would serve to overcome the difficulties of the older methods and remedy their lack of proper controls. The possibility has also been raised that the two theories may be complementary, rather than opposed to each other; this possibility too should be looked into from the experimental point of view. What is certain is that at the moment no final decision can be made about whether, or the degree to which, cigarette smoking may cause lung cancer or coronary heart disease, how it interacts with other factors (stress, personality), or how we can best protect the health of our citizens in relation to these diseases. "In ignorance, abstain!" warned the famous French scientist, Claude Bernard; hasty action on the basis of partial knowledge is unlikely to be in the best interests of those most concerned, namely the prospective victims of lung cancer and coronary heart disease.

H. J. Eysenck
May 6, 1983
Statement of Richard J. Rickey, M.D.

I am Richard J. Rickey, a senior research investigator in the Department of Statistics of The Wharton School, University of Pennsylvania, Philadelphia. I received a Ph.D. degree in biophysical chemistry and microbiology at Iowa State University, Ames, Iowa, in 1941. Thereafter, I performed research and development work on methods for production of antibiotics, vitamins, and other biochemicals. In the mid-1960's, I became involved in research on the air pollution/public health problem at the University of Pennsylvania. Since 1973, I have been investigating ecological relationships of environmental chemicals and other variables to chronic disease risk at the University's Wharton School. Many of the journal articles and book chapters published, of which I was author or co-author, pertain to environmental pollution and its effects on human health.

On March 2, 1982, I submitted a statement regarding the present subject. It involved comments on H.R. 4957 and S. 1923, which were under consideration during 1982. That statement was published in the Appendix to Hearings regarding the Comprehensive Smoking Prevention Education Act, before the Subcommittee on Health and the Environment, of the Committee on Energy and Commerce, House of Representatives, 97th Congress, 2nd Session, on H.R. 5653 and H.R. 4957, March 5, 11, and 12, 1982, Serial No. 97-107. The statement appears on pages 618-625 of the Appendix.

The following comments concern both H.R. 1824 and S. 772, being considered in 1983. A basic criticism of both proposed acts is the implicit assumption that "findings" (1), (2), (3), and (4), which are used to justify both the labeling statements and the proposed actions contained in S. 772 are known and proven. However, there are very serious scientific questions concerning the foundations for the claims that cigarette smoking causes the various conditions associated statistically with cigarette smoking, such as lung cancer, heart disease, and low birth weight.

Documentary biological and biochemical evidence establishing proof of causality is not given in the proposed bills. It is clear, however, from the wording of the "findings" that they rely heavily upon the alleged causality evidence, and
Jahns, presented in some publications and reviewed in recent U.S. Surgeon Generals' reports on smoking and health. Unfortunately, these reports are not balanced, even-handed reviews of the available evidence. There are several reasons for rejecting the conclusions of these Surgeon Generals' reports, including:

(A) **Selective Reporting or Selective Exclusion.** Selective reporting is present in the Surgeon Generals' reports. Reports that conform to the predetermined smoking-causality position of the Surgeon Generals' reports are cited quite extensively, while studies that challenge or discredit this position are often selectively omitted. Published warnings regarding misuse of statistics have also been excluded. The Surgeon Generals' reports are characteristic of briefs for the prosecution only; the defense has been largely neglected or omitted even though these reports, using public funds, should certainly have been unbiased. The presence of bias raises serious questions regarding ethics.

(B) **Uses and Misuses of Statistics.** The Surgeon General's report of 1964 states (p. 127): "Statistical methods cannot establish proof of a causal relationship in an association. The causal significance of an association is a matter of judgment which goes beyond any statement of statistical probability." These two statements lie at the root of the problem. The first sentence is a correct statement. Causality cannot be established by statistical associations. This is a standard caveat that is taught routinely in elementary statistics courses. The second statement is false. Subjective judgment or personal opinion cannot be used to interpret causal implications of a statistical association, no matter how strong and no matter what level of statistical significance. Judgment and opinion do not constitute substantive evidence, nor are they objective assessments of the facts. They are subject to personal bias and biopolitical persuasions.

It should be recognized that statistical associations are mathematical quantities. When properly obtained, they may suggest possible causal relationships, but they can never prove such relationships. It is essential to present several alternative hypotheses to explain a confirmed statistical association. Scientific tests are necessary to evaluate the validity of each hypothesis. Some will be rejected. The House and Senate bills ignore this basic scientific requirement. Human conditions such as cancer, heart disease, spontaneous abortion, stillbirths and birth weight deficiencies are all basically biological and biochemical problems, not mathematical or statistical problems. When ill, one visits his physician, not his statistician.

Properly used, statistical methods are capable of rejecting an hypothesis to
the extent that it is incompatible with observed data, but statistics alone can never establish that an hypothesis is certainly true. This fundamental fact is widely misunderstood or ignored. However, recent Surgeon Generals' reports have rather routinely inferred causality from statistical association, e.g., that cigarette smoking causes lung cancer since the two variables are positively associated in the statistical sense. Moreover, a statistical association cannot distinguish cause from symptom. It should be recognized that statistical malpractice is a form of scientific malpractice.

Statistical associations may be due to chance or to dual influences of some third factor. For example, it was reported in 1952 that "there is ... a significant correlation in the United States between the population to physician and the population to bed ratio and the number of reported deaths from cancer of the lung." [R.M. Rigdon and R. Kirchoff, "A Consideration of Some of the Theories Relative to the Etiology and Incidence of Lung Cancer," Texas Dept. Biol. Med., 4, 76-91 (1952) (p. 88)]. It seems unlikely that anyone would conclude from this correlation that physicians "cause" lung cancer.

It is known that cigarette smokers tend to be coffee drinkers; a positive correlation exists between measures of cigarette and coffee consumption in some populations. Is it to be concluded, therefore, that smoking "causes" coffee drinking, or that coffee drinking "causes" smoking? Or does an underlying third variable, such as perhaps individual genetic or constitutional characteristics, influence both coffee drinking and smoking behaviors?

Another common fallacy is the presumption and belief, by some, that non-random samples are unbiased. This belief is especially hazardous when working with genetically heterogeneous populations such as humans. For example, studies that employ self-selected samples such as those of heavy smokers, light smokers, non-smokers, and ex-smokers are very likely biased. There is no assurance that they are not. This point was addressed by T.D. Sterling ["The Statistician vis-a-vis Issues of Public Health," American Statistician 27, 212-216 (1973)(pp. 212-213)]: "The manner of selecting populations to study the effects of smoking has been criticized by some of the world's most respected statisticians but to little avail (Berkan, 1958; 1960; Fisher, 1957; 1958; Mainland, 1956; just to mention a few). Of concern to statisticians has been the major reliance on 'volunteers' in most of these studies and the consequent multiple sources of serious bias. Ordinarily, results from a study such as Bell's in which over 30 per cent of the queried population did not participate would be considered, if at all, with greatest suspicion, especially if, as is freely admitted, the respondents were quite different from the reference pop-
ulation (Bell, 1964). Similarly, data collected by volunteers of the American Cancer Society and used to link smoking to a variety of diseases stem from a study population (also of volunteer families) that dramatically differs in some significant respect compared to measures from the U.S. population (Stetler, 1971a, 1972a).

Further, regarding sample bias, consider one group of persons who are regular consumers of insulin and compare the health of this group with controls who do not consume insulin. It may be observed that the prevalence of circulatory and certain other diseases is considerably greater among insulin consumers than among non-consumers. Does this mean that insulin "causes" the increased incidence of these afflictions? Or might it be that an underlying metabolic anomaly, diabetes mellitus, is the basis for both insulin consumption and the elevated risk of certain chronic diseases known to occur among diabetics?

(c) The Genetic/Constitutional Alternative. In the late 1950's, Sir Ronald A. Fisher, a former President of the Royal Statistical Society, London, suggested the following alternative to the smoking-causality hypothesis: "Cigarette smoking and lung cancer, though not mutually causative, are both influenced by a common cause, in this case the individual genotype." The problem of resolving the conflict between the smoking-causality hypothesis and the genetic/constitutional hypothesis was reviewed recently in depth by P.R.J. Burch ("Smoking and Lung Cancer: The Problem of Inferring Cause with Discussion," Journal of the Royal Statistical Society, Series A (General), Vol. 141, pp. 427-477 (1978)). Burch concluded (p. 476): "The discussion has allowed me to undermine one causal model and it has revealed divergent opinions that might surprise those nurtured on the reports of the Royal College of Physicians and the Surgeon General of the United States. I am also happy to record that the constitutional theory, proposed by a former President of this Society, the late R.A. Fisher, is alive and well."

To this date the genetic/constitutional hypothesis has not been discredited. It has, however, been denigrated subjectively and widely ignored. Despite the fact that Burch's paper appeared in a major statistical journal, it had not been cited in several recent Surgeon General's reports. Further, the constitutional hypothesis has been "criticized" by some who seem to object to the subjective or rhetorical criticism with valid rejection. Some criticism has been of the "you don't like" variety.

Unless the genetic/constitutional alternative hypothesis is rejected on valid scientific grounds, it would be dangerous to base public policy upon the smoking-causality hypothesis. Numerous scientific reports, such as that of Burch, support the genetic/constitutional hypothesis.
It is established to the contrary that such habitual smoking is symptomatic of an underlying genetic trait that both influences smoking behavior and contributes to increased risks of developing those diseases associated statistically with smoking. If anti-smoking legislation is passed and it eventually is established that such habitual smoking is symptomatic of an underlying genetic trait that both influences smoking behavior and contributes to increased risks of developing those diseases associated statistically with smoking, then the U.S. government would be placed in a totally untenable position. The prospect that R.A. Fisher, considered by some to be the father of modern statistics, and one of the world's great scientists, has been correct all along is very real and cannot be ignored. The Congress must not act on such an issue.

Air Pollution and Chronic Disease. In addition to the omission of publications such as that of Borch, the Surgeon General's reports have also extensively excluded from consideration published epidemiological reports on the complex relationships between a number of common air pollutant chemicals and mortality rates for several categories of cancer, heart disease, and certain other causes of death in the United States such as in U.S. cities.

Several studies have shown that concentrations of several common air pollutants such as sulfur dioxide, nitrogen dioxide, and certain trace metals in the air of 18 U.S. cities were significantly associated statistically with lung cancer mortality rates for those cities. In fact, over 70% of the variance in lung cancer mortality rates was explained statistically by air pollutant characteristics of these cities. Furthermore, over 50% of the variance in mortality rates for several other cancer classifications was also explained statistically by air pollution concentration data. Furthermore, also over 50% of the variance in mortality rates for certain heart disease classifications was explained statistically by air pollutant concentration data for these 18 cities. Statistically significant relationships were also found between air pollutant concentration data and mortality rate data for infants under one year of age, and for mortality rates for congenital malformations. For example, R.J. Rickett et al., "Ecological Statistical Studies Concerning Environmental Pollution and Chronic Disease," IEEE Transactions on Geoscience Electronics, Vol. GE-14, 186-202 (1976); R.J. Rickett, "Air Pollution," pp. 189-212 in: Environment, Resources, Pollution & Society (U.S. Murdoch, ed.), Ballinger Associates, Stamford, CT, 1977; R. Mendelsohn and G. Orcutt, "An Empirical Analysis of Air Pollution Dose-Response Curves," Journal of Environmental Economics and Management, Vol. 5, 85-106 (1979).

Comments on Four "Findings" of S. 772. The following comments refer to "Findings" (1), (2), (3), and (4) that appear on page 2 of S. 772. The most detail is offered regarding finding (4) which pertains to pregnancy, low birth weight, and...
related problems of infant health.

Finding (1). S.772 states that "cigarette smoking is the larger preventable cause of illness and premature death in the United States and is associated with the unnecessary deaths of over three hundred thousand Americans annually." This statement pertains in large part to deaths from cancer and from cardiovascular disease. However, it says nothing about the constitutional hypothesis nor about epidemiological and other evidence regarding the relation of air pollutant chemicals to chronic diseases.

Finding (2). S.772 states that "smoking is the primary cause of lung cancer and emphysema in the United States and is associated with other cancers." This claim has been made in various ways in the U.S. Surgeon General's reports. However, as stated previously, this claim is defective because it involves in part selective reporting, misuse of statistics, and it ignores evidence regarding the genetic/constitutional influences, and fails to consider published evidence regarding strong statistical relationships that have been found between concentrations of air pollutant chemicals and cancer mortality rates. It is also an important fact that despite many attempts, lung cancer has never been induced in experimental animals in well-designed studies by fresh tobacco smoke when experimental conditions resembled those of natural exposure.

As mentioned above, deficiencies in the theory that smoking causes lung and other cancers were examined at length by Burch in 1970. Earlier, in 1965, K.A. Brownlee ("A Review of 'Smoking and Health'", Journal of the American Statistical Association, Vol. 60, 722-729 [1965]) examined the 1964 Surgeon General's report, Smoking and Health, and found it defective in both biochemistry and statistics. It is noteworthy that reports claiming that the smoking-mortality associations are causal appear largely in medical and biomedical journals rather than in statistical journals. Statisticians are not necessarily included among the authors. Statistical journals do not accept reports that imply inference of causality from statistical association to be "valid". It is also noteworthy that Brownlee's review was not mentioned in several subsequent Surgeon General's reports.

Finding (3). S.772 states that "heart disease accounts for nearly one-half of the deaths in the United States and one-third of the deaths attributable to heart disease are associated with smoking." Death rates from heart disease increase rapidly with increasing age, especially beyond the age of 40 years. Thus, heart disease appears to be in large part diseases of aging, a fact not evident in this finding in S.772.
The statement that one-third of these deaths are "attributable" to smoking implies cause. "Associated with" does not. There is no question about the association in some populations. But anyone who then claims or argues for a causal relationship makes a huge leap based on faith alone. Any causal implication is defective because it involves in part those technical deficiencies discussed previously. In addition, quite strong statistical relationships have been reported to exist between documentations of a number of common air pollutant chemicals and mortality rates for several classifications of heart disease. The air pollution-heart disease relationship has often been overlooked, ignored, or minimized in Surgeon General's reports.

Paying (6). S.272 states that "the risks of miscarriage, stillbirths, pre-mature births, and child weight deficiencies for pregnant women who smoke, are higher than for pregnant women who do not smoke." The term "risks", is strictly statistical and is not biological or biochemical. The implication is that because of statistical associations, the smoking-causality hypothesis is true. This is the same misuse and abuse of statistics and of science, noted in preceding discussions. The implication that, except for smoking or non-smoking, all other genetic, physiological characteristics of the women are the same is erroneous; to be known as the fallacy of typology. Populations are genetically heterogeneous.

Causality cannot be inferred from statistical association. The causal implications of claims, as stated in Surgeon General's reports, are also defective because of selective reporting. Among the reports that address this problem is: R.J. Hickey, R.C. Clelland, and R.J. Gowers, "Maternal Smoking, Birth Weight: Infant Death, and the Self-Selection Problem." American Journal of Obstetrics and Gynecology, Vol. 133, 805-811 (1978). Misuse of statistics is pointed out along with other errors and fallacies, including the overlooking of or subjective designation of the publications of the late Professor J. Yamashita, of the School of Public Health, University of California, Berkeley.

That low birth weight has been associated statistically with maternal smoking during pregnancy in some populations is not in question. However, the multiple implications or claims in Surgeon General's reports that association means smoking-causality constitute misuse of statistics and thereby misuse and abuse of science. Factors other than smoking also correlate with low birth weight, such as certain socio-economic variables, maternal height and weight, geographic differences, and altitude variations. The 2,500 grams birth weight criterion, implying that
less than this weight is unhealthy. ignores the large variations in these factors. For example, there is evidence that birth weights of children of large, tall women tend to be heavier than birth weights of children of small, shorter women. [See J. H. Tanner, L. LaJeunesse, and G. Turner, "Within-Family Standards for Birth-Weight," Lancet 2, 193-197 (1972)]. Surely genetic and constitutional factors influence birth size and weight in many populations just as genetic characteristics specify hair and eye color, and sex.

An informative but controversial kind of evidence that addresses the conflict between the smoking-causality hypothesis versus the constitutional hypothesis pertains in part to whether women who later take up smoking ("future smokers") tend to have low birth weight children even before they started smoking. This question was examined by Professor J. Yarushek in "Infants with Low Birth Weights Born Before Their Mothers Started Smoking Cigarettes," American Journal of Obstetrics and Gynecology, Vol. 112, 277-284 (1972). He found that, indeed, birth weights of children of future smokers tended to be lower than birth weights of children of never smokers. Such a finding is in accord with the genetic/constitutional hypothesis rather than with the smoking-causality hypothesis. Yarushek's findings are consistent with those reported by D. Silverman in "Maternal Smoking and Birth Weight," Thesis, Johns Hopkins University, Baltimore, Md., 1972. These findings have not been particularly popular, they have often been ignored or selectively excluded from consideration in smoking and health reports.

It is noteworthy that Yarushek also found that the smaller babies of smoking mothers were healthier than the smaller babies of non-smoking women.

To summarise, in view of selective reporting in Surgeon General's reports, of evidence supporting the genetical/constitutional hypothesis, of missed statistics, and the sometimes biased samples, it seems desirable to table any impliant actions until the Congress has received a more complete and unbiased analysis of the evidence. At the present time, statements about deleterious effects of smoking are often presented based on support from evidence and claims that are scientifically unconvincing, biased, subjective rather than objective, and very possibly invalid.

Richard J. Hickey
May 2, 1983.
STATEMENT
OF
ROBERT CASAD HOCKETT
Regarding S. 772

I am Research Director of The Council for Tobacco Research - U.S.A., Inc. I have been employed by The Council and its predecessor, the Tobacco Industry Research Committee, since 1954, first as Associate Scientific Director, then Acting Scientific Director, and since 1973 in my present capacity. My curriculum vitae is attached.

I was strongly attracted to this organization at its inception, by the leadership of the late Dr. Clarence Cook Little whose intense interest was in the pathogenesis and etiology of the non-infectious "constitutional" diseases - originally cancer - but eventually extended to disorders of the heart, arteries and lungs. These are all conditions that become more prevalent with aging. This affiliation also brought me into contact with
the late Dr. Milton B. Rosenblatt, an outstanding medical historian who was just then completing an exhaustive historical study of carcinoma of the lung.

The summary of his findings, submitted in July, 1957 to the subcommittee on Legal and Monetary Affairs, Committee on Government Operations, House of Representatives, is still so highly relevant to the bill presented by Mr. Hatch that I am including it here in full. The passage of time since 1957 has had little impact on the facts he states.

"1. Cancer of the lung has been recognized for more than 150 years antedating the popularity of cigarette smoking by more than a century."

"2. In hospitals interested in the disease, and/or with routine autopsies, there has been no real increase in lung cancer with respect to total cancers. This holds for the 19th century as well as for the 20th century. For example in a survey of the hospitals conducted by the London
County Council, the percentage of lung cancer to total cancers was 25 in 1936 and 27.1 in 1947."

"3. The attempt to implicate tobacco was first made in this country by Adler in 1912. Inasmuch as cigarette smoking had not yet achieved popularity, the onus was placed on cigars. With the greater consumption of cigarettes in later years, the blame was gradually shifted to cigarettes finally culminating in the recent barrage of statistical epidemiological studies conducted on the grand and loud scale." 

"4. A statistical association does not imply a cause and effect relationship, particularly when the validity of the conclusions is seriously questioned by such responsible statisticians as Joseph Berkson of the Mayo Clinic or Edward
Lew of the Metropolitan Life Insurance Company."

"5. Bronchogenic carcinoma has never been produced by tobacco or its products in any experimental animal despite the multiplicity of attempts. Dr. Greene, of Yale, who has devoted a lifetime to experimental cancer research found no causal relationship between smoking and lung cancer. Actually, he found tobacco tar to be among the lowest of the carcinogenic agents used experimentally. The production of skin cancer by tobacco products is of little significance, particularly, when reputable investigators could not reproduce the experiment."

"8. History has repeatedly demonstrated that whenever an inaccessible cancer becomes
accessible, the [reported] incidence automatically increases. Prior to 1930, the clinical facilities for the diagnosis of lung cancer were few and far between even at thoracic disease hospitals. The perfection of diagnostic tools (exfoliative cytology, bronchoscopy, radiology, and exploratory thoractomy) occurred within the past 25 years. This is obvious to all who have worked through this era. Many hospitals now perform more bronchoscopies per day than they did per year a quarter of a century ago. The records of a world famous cancer institute show that its staff recognized only about half a dozen lung cancers annually before 1930."

"Lung cancer is a disease of older age groups. There are more older people among us and more
potential candidates for lung cancer. The total number of cases is increasing but the rate of increase is slowing down and will eventually be stabilized like laryngeal or other upper respiratory cancers in which diagnostic methods have been long established."

"5. The nasopharynx and the larynx are greatly exposed to tobacco products and if tobacco is a carcinogen for the lung, it should also act on the upper respiratory passage. However, no statistical association has been demonstrated between increased consumption of cigarettes and increased incidence of upper respiratory cancer. In 1940, the United States death rate from laryngeal cancer was 11 per 100,000 and in 1950, it was 1.2 per 100,000."
It is clear that lung carcinoma is not a new disease and that it was well known in nineteenth-century hospitals where post-mortem examinations were made routinely, especially in England, France and Germany. The general prevalence of the disease in the last century is not known since the disease was not discovered by the available clinical diagnostic procedures of the time.

I would modify Rosenblatt's comments about "tobacco tar" by pointing out that it is simply a condensate of smoke particles caught in a cold trap or other device. It lacks the gas and vapor phases of whole smoke, and since fresh whole smoke is an unstable aerosol of particles containing thousands of individual substances undergoing rapid changes, it is very different in properties from condensate that has been trapped and preserved.

S. 772 speaks of the level of "(A) tar; (B) nicotine; and (C) carbon monoxide; contained in such cigarettes." No cigarette contains any tar or carbon monoxide. These are products of the burning process: carbon monoxide is a single identifiable gaseous compound, but "tar" is an
extremely complex mixture of organic compounds, that varies with the composition of the cigarette, the conditions of its burning and the techniques of condensation and storage.

The purported "finding" that cigarette smoking is the "primary cause" of emphysema is scientifically unfounded. Emphysema is a gradual loss of elasticity in the walls of the tiny air vesicles of the lung that occurs universally with aging. When the changes are mild, they are hardly thought of as disease. In some persons, however, the loss is rapid and severe and produces a serious handicap. The severity of the emphysema is often measured by determining the maximum volume of air that the patient can push out of his lungs in a given time. (Forced Expiratory Volume or FEV).

Dr. Charles Fletcher of the Royal Postgraduate Medical School in London has carried out extensive studies on cigarette smoking and rates of decline in the FEV with aging. He finds that a great majority of smokers show very little loss of FEV, while the disease is well-known to occur in some non-smokers.
While cardiovascular disease is still one of the leading causes of death in the United States, the incidence of heart attacks has been diminishing steadily for more than twenty-five years, for reasons that are unknown or conjectural. The MRFIT study, recently completed, did not produce the expected results nor provide an obvious explanation.

Biochemical studies are however throwing light on the possible role of lipoproteins in the development of atherosclerosis and in its inhibition. The indications are that there are many genetic variations in the metabolic processing of these substances. While disorders of genetic origin often baffled our scientific predecessors as difficult to treat, it now appears that biochemical techniques may offer new hopes of overcoming the effects of congenital predispositions.

It is obvious that the historical findings of Dr. Rosenblatt constitute a strong caution against dogmatic pronouncements on labels or in other contexts. Dissillusionment can have disastrous effects upon the public confidence. I cannot endorse the wording of the present labels, let alone the new ones.
The path of real progress, I am convinced, lies in studies of the etiology and pathogenesis of cancer, heart and artery disorders, and emphysema, with full exploitation of the new techniques of molecular biology.

A number of eminent cancer researchers, notably Dr. Lewis Thomas, have pointed out that "the overall incidence of cancer of all types, in all societies, whatever the difference in environmental hazards, is estimated to be fixed at around 25%, suggesting that three quarters of us may be in possession of mechanisms for successfully resisting cancer throughout our lives." Dr. Thomas says:

"It may be that there is an immune reaction to the appearance of the first cancer cells, which is mobilized as soon as the alien nature of these cells is recognized. Such a mechanism, if it exists in human cancer, might be the one that protects the 75 percent of us who will never develop the disease. Perhaps all of us are experiencing, from one carcinogenic..."
environmental influence or another, the emergence of single cancer cells and a few of their progeny from time to time, in one tissue or another, and eliminating them promptly when they are perceived as foreign by our lymphocytes. If the recognition comes too late, or not at all, cancer develops — and that accounts for the susceptible 25 percent of us. I proposed this notion 25 years ago, and it was elaborated later under the term "immunosurveillance." It remains an unproven theory, but I retain high hopes for it as well as a certain affection, since it was one of my few excursions into theoretical biology."

There is a new note of optimism among cancer researchers who see in the rapid new developments in immunology research a promise of unprecedented advances in prevention of cancer.

In conclusion, the proposed "findings" set forth in S.772 regarding cancer, cardiovascular disease, atherosclerosis and emphysema are not supported by the scientific evidence; it would truly be unfortunate for science to be frozen in its tracks by such unfounded pronouncements.
CURRICULUM VITAE OF DR. ROBERT C. HOCKETT

Dr. Robert C. Hockett, Research Director of The Council for Tobacco Research - U.S.A., Inc., 110 East 59th Street, New York, New York 10021, was born in Fayette, Missouri on July 7, 1906.

A graduate of the Ohio State University, Dr. Hockett pursued graduate study in the same institution and received the Ph.D. degree in chemistry there in 1929. As a National Research Council Fellow in Chemistry, he was a guest scientist at the National Institute of Health, U.S. Public Health Service from 1929 to 1931 and then Associate Chemist on the Institute staff from 1931 to 1935.

In 1935 he joined the faculty of the Massachusetts Institute of Technology as Assistant Professor of Chemistry, becoming Associate Professor in 1939.

In 1943 he was granted a leave of absence from M.I.T. to serve as Scientific Director of the Sugar Research Foundation, Inc., which position he occupied until 1952. He has also been Visiting Professor at the Universities of Illinois and North Carolina.

From 1952 until 1954 he worked as a consultant to industrial firms on problems relating to foods, nutrition, pharmaceuticals, fermentation, and sponsored research.

In 1954 he joined the present Council for Tobacco Research's predecessor organization - the Tobacco Industry Research Committee - as an Associate Scientific Director.

Dr. Hockett is a Fellow of the American Academy of Arts and Sciences, the New York Academy of Sciences, the American Public Health Association, the Royal Society of Arts, and the American Institute of Chemists. He holds membership in the American Chemical Society, in which he has served as an Alternate Councillor, member of the Nominating Committee, Vice-Chairman of the Division of Carbohydrate Chemistry in 1944, Chairman in 1945 and 1946, and Secretary-Treasurer from 1930 to 1946. He also holds membership in the American Society of Biological Chemists, American Association for the advancement of Science, Friends of the World Health Organization, Royal Society of Edinburgh, and the Phi Beta Kappa Association.

He has served as a Lecturer in Scotland for the American-Swedish Foundation for Scientific Exchange, member of the Food Industries Advisory Committee of the I.SEVER Foundation, Inc., Colleague to the United States Department of Agriculture, member of the Advisory Committee for Advancement in Carbohydrate Chemistry, as Associate to the Senior Researcher on Carbohydrates for Tri-Continental Scientific Publication and member of the U.S. Advisory Committee to the Administrator of the Food and Agriculture, U.S. Department of Agriculture.

He is author of numerous research papers on the chemistry of carbohydrates, of articles and lectures on nutrition and public health, of essays for newspapers and magazines on tobacco and health research and formulation of New Sugar Economies.
My name is Duncan Hutchison. I am Professor of Pharmacology and Medicine at the University of Medicine and Dentistry of New Jersey, where I have also been an attending physician at the University Hospital and New Jersey Medical School. I received my M.D. and B.Sc. (med) degrees from the University of Toronto, and in 1950 I obtained my D.Phil. from Oxford University as a National Research Council (Canada) Post-doctoral Fellow in the Department of Pharmacology.

My research has been primarily in clinical and cardiovascular pharmacology, and I have approximately 70 research publications. I have also contributed to several textbooks, including Drills’ Pharmacology and Medicine, Treatment of Heart Disease in the Adult by Hultet et al., and Cardiovascular Therapy by Ruskell. Since 1977, I have served as Editor of the Journal of Clinical Pharmacology.

I hold memberships in a number of medical and scientific societies including the American Society for Pharmacology and Experimental Therapeutics and the Society for Experimental Biology and Medicine. I am a fellow of the American College of Physicians and American College of Clinical Pharmacology of which I am a past-president.

In 1980, I helped establish the Princeton Institute of Environmental Medicine (PIEM), an organization of medical scientists concerned about the health effects of chemical substances in the environment. Our research focuses on the relationships between various environmental risk factors and chronic diseases such as heart disease and cancer. Environmental factors we are investigating include tobacco smoke...
compounds, industrial chemicals, pesticides, and emissions from jet, diesel, and gasoline engines.

Based on my research experiences, I believe that it is reasonable to make strong, unequivocal statements about the contribution of any single factor to the occurrence of cancer and heart disease. For this reason, many of the statements in S.3.772 regarding the health consequences of smoking are unacceptable scientifically and appear to represent an attempt to offer easy solutions to what are actually complex multifaceted problems.

Furthermore, it is my belief that Congress has the responsibility of encouraging thorough and unbiased investigations into the spectrum of risk factors associated with these diseases rather than limiting its consideration to only one aspect. Why, for example, doesn't the proposed legislation mention environmental hazards? After all, in terms of both quantity and possible carcinogenicity, teratogenicity, and carcinotocicity, environmental chemicals such as polyyclic hydrocarbons (PAH's), halogenated hydrocarbon pesticides, and industrial solvents pose serious public health problems.

These views are based on our field work that involves correlating mortality data provided by the National Institutes of Health with our own clinical and laboratory findings. By this method, we have examined the following environmental pollutants: benzo(a)pyrene (BaP) and other PAH's, carbon monoxide (CO), and halogenated hydrocarbon pesticides as well as certain household and industrial solvents, such as trichloroethylene and tetrachloroethylene.

Our studies show that there is considerable misinformation regarding the possible biological effects of the various chemicals to which we are regularly exposed. Moreover, there is little appreciation of the way in which such substances are handled by the body (i.e., how and when they are eliminated), and the relationship between the amount of chemicals present in the environment and the amounts necessary to initiate carcinogenic and teratogenic activity.
For example, we have measured serum (half) levels by radioimmunoassay procedure in urban and suburban populations groups in the New York metropolitan area and found significantly higher levels in the group living in the areas with the highest atmospheric pollution. 

Half was chosen as a marker of urban air pollution because it is a suspected carcinogen that is produced mainly by burning fossil fuels. It is also considered to be a useful indicator of potentially carcinogenic PAH's in the atmosphere. Our observations indicate that geographic location may be an important risk factor for respiratory cancer. While these findings are preliminary, they suggest that environmental studies should be conducted on a much larger scale. Such studies could assist in identifying populations at environmental risk and pinpointing geographic areas where environmental control efforts should be directed.

During the past three years, my laboratory has also focused on the effects of environmental chemicals on cardiac performance. This has involved investigating the association between geographic location and the prevalence of heart disease and sudden cardiac death. Our findings suggest that certain chemicals associated with adverse cardiac effects are present both in ambient air and in the drinking water supply of various communities. The chemical agents that may be related to cardiac mortality rates in areas of greatest risk are the halogenated hydrocarbons.

Our research group has also conducted experimental studies on the possible effects of carbon monoxide exposure. Although we originally assumed that CO might play some role in heart disease causation, our findings have suggested that (1) CO at levels in the environment does not appear to predispose the heart to arrhythmias or electrically-induced arrhythmias in experimental animals, and (2) although the CO levels are higher in the ambient air of certain urban-industrial areas, the concentrations do not appear high enough to cause significant health consequences.

This view is supported by the epidemiological and other reports issued by federal and state governments which show no significant correlation between the prevalence of heart disease and concentrations of carbon monoxide in the ambient air.
Our laboratory studies have also led us to an appreciation of the importance of the absorption/elimination characteristics of environmental chemicals. It is our view that the toxic properties of foreign chemicals ultimately depend on how well the body biotransforms and eliminates them and on how the body's immunological and other defense systems operate. It is clearly evident that more research is needed on the health effects of long-term, low-level exposure to chemicals.

In summary, our work at PIER indicates that a balanced scientific approach is necessary in the study of the potential health consequences of environmental risk factors. Any approach that limits its consideration to only one factor, like tobacco smoke, will hinder the pursuit of epidemiological, pharmacokinetic, and toxicologic research needed to tackle our nation's health problems. On the basis of our laboratory research and the epidemiological information available to us, I therefore cannot agree with the statement contained in S.B. 772 that suggest that the scientific community already knows what causes this country's most serious chronic diseases.
My name is Eleanor Macdonald, Professor Emeritus of Epidemiology at the University of Texas System Cancer Center, M.D. Anderson Hospital and Tumor Institute, Houston. I have worked in the field of epidemiology for over 40 years in three state programs in Massachusetts, Connecticut and Texas; in fact, I established the first state cancer registry in the U.S. From 1948 through 1974, I chaired the Department of Epidemiology at the University of Texas System Cancer Center. I have authored or coauthored about 150 publications, most of which deal with different phases of cancer epidemiology, and I remain actively involved in a number of ongoing research studies in epidemiology. I am also the editor of the Epidemiology, Statistics and Cancer Control section of the Yearbook of Cancer.

I share the concerns of this Committee regarding cancer morbidity and mortality, and I encourage legitimate efforts to control this disease. However, as a scientist who has dedicated her professional career to a careful study of cancer epidemiology, I am appalled at the belief implicit in S. 772 that Congress can legislate scientific fact. I urge this august
body of well-intentioned legislators to avoid putting itself in such an untenable position.

This bill is a misdirection of governmental energy and purpose because of its narrow focus on smoking as the primary cause of lung cancer. We do not, in fact, know the cause or causes of lung cancer and it is not in the best interest of either science or government to take a simplistic approach to the task of identifying them. There are obviously many factors in cancer causation that we have not yet begun to understand. For example, the depth of our uncertainty is still far too great regarding the effects of nutrition, work history, lifestyle, ancestry and environment.

One of the main reasons that I disagree with this bill's finding regarding smoking and cancer stems from my knowledge of the primary type of evidence on which these judgments are based, i.e., epidemiological. As an epidemiologist who has conducted large-scale population studies, and who has reviewed the data of vast numbers of research reports, I am perhaps more aware than many scientists -- and certainly more aware than most laymen -- of the limitations of this type of data. Congress must understand that epidemiology is not an exact science, and no amount of wishing can make it so. We cannot turn humans into animals directed at will toward various exposures.
We cannot send all death certificates to one infallible pathologist for correction and confirmation. We cannot force all individuals supplying the raw data to epidemiologists, or even all epidemiologists themselves, to demand the type of precision necessary in data gathering and review to insure that the quality of the data meets high scientific standards. Consequently, judgments about chronic disease causation based on epidemiological findings must be highly tentative; they do not enjoy the absolute certainty implied in the findings of this bill.

Mortality data are frequently used by epidemiologists to study disease trends, often without full awareness of inherent weaknesses in these data. Mortality rates are obtained from information on death certificates furnished by physicians or local health officials. Although I am certain that every effort is generally made to fill these out conscientiously, errors do occur—in diagnosis as well as in recording of the data.

In addition, other exogenous influences can cause profound fluctuations in mortality data over the years that can easily be misinterpreted as changing disease trends. For example, the introductions of each of the seven revisions of the International Lists of Diseases and Causes of Death (ICD) have had effects, generally improvements, on the classification of
diseases. With each revision, however, the continuity of the disease trends has been broken and in some cases seriously distorted. Sadly enough, even though it has taken us centuries to attain the classification system that we now have, it is still imperfect.

The possibility of shifting classifications bringing about a rise or fall in certain causes of death makes some investigators question the value of any effort to compile statistics on causes of death. For example, anyone attempting to trace the patterns of lung cancer during the twentieth century must remember that the 1948 ICD revision, first applied in 1949, introduced a classification separating primary cancer of the lung from metastatic cancer of the lung (coded thereafter as "unspecified as to primary or secondary"). For two years, there was a sharp reduction in the reported primary lung cancer rate, and then a rise in both the primary and unspecified rates. How long did it take for the new classification to catch on? How accurately did physicians distinguish between primary and secondary? When epidemiologists study twentieth century lung cancer trends, do they include or exclude the unspecified and secondary cancers? We cannot be sure about the answers to these questions.
The 1965 ICD revision, adopted in 1968, seriously complicated the situation by deleting the unspecified category. This revision effectively combined primary and unspecified lung cancer, thereby removing a needed safeguard for accuracy. Primary and secondary lung cancer are separate disease entities, with quite possibly distinctly different causes. Since ten percent of all cancers spread to the lung, and since, for nearly twenty years, more deaths were coded in the unspecified than in the primary category, the combination of these two categories seriously confused the lung cancer data. The damage done to epidemiological investigations of lung cancer by the 1965 classification change cannot be underestimated. In the ninth revision (1980), greater confusion was added by placing the bronchus and lung unspecified as the ninth subdivision of primary lung cancer (162.9). Death certificates seldom list more than a three-digit number, such as 162. We simply cannot separate out from the mortality data the specific information we need to study lung cancer scientifically.

Another aspect of the problem of changing disease classifications can be found by comparing trends in stomach and lung cancer. In the first half of this century, the phrase "question of gastric cancer" was used frequently on death certificates when a cachectic patient died within days or hours of first summoning a physician. It is fascinating to see that in
47 states, within two years of the introduction of the 1948 revision, the reported gastric cancer mortality rates dropped as suddenly as the unspecified lung cancer ones rose. Thus, the two changes may in part represent a shift of questionable diagnosis from the difficult to diagnose stomach cancer to the equally difficult to diagnose lung cancer classification.

Besides studying anomalies that have developed in overall lung cancer trends, I have also examined trends in the reported lung cancer mortality rates for women in this century. For years, those individuals who believed that smoking caused lung cancer focused almost solely on male lung cancer, and generally tended to ignore female lung cancer. In recent years, however, more attention has been given to female lung cancer because of reports of an increase in female lung cancer and the claim that increased cigarette smoking was responsible. I do not accept this interpretation because I have serious scientific reservations about both the accuracy of the underlying data and the conclusions drawn from it.

First of all, we simply do not have reliable data on smoking prevalence in women — or in men, for that matter. The best information we have is from the National Health Survey reports, yet the 1978 report, for example, has a standard error
of 30 percent. That percentage of error is too high to provide a basis for drawing definitive conclusions.

Secondly, I have doubts about whether lung cancer in women has actually increased dramatically. As I indicated earlier, national mortality data may not be reliable. If we assume for the sake of argument, however, that they are reliable, we can still question the argument that female lung cancer has increased sharply in recent years. Although the age-adjusted U.S. death rates from respiratory cancer in white females show a consistent increase from 1953 through 1975, the rate of this increase has varied over this period. A smoothed year-to-year variation in the rate of increase -- the slope -- indicates that a sharp rise began around 1960, but leveled off around 1970. Although the rate of increase has begun to rise again, it is not as steep an ascent as in the 1960's.

This is important because the slope gives a predictive picture of the overall mortality trend. And the slope that I see for female lung cancer suggests a stabilization. It may even predict a decline in lung cancer mortality in the coming years.

Other factors must also be considered in an analysis of the apparent increase in female lung cancer. One such factor, inaccurate reporting, appears to have played a predominant role.
My analysis of both reporting techniques and the underlying data suggests that lung cancer in women was underreported in the past and is being overreported now.

Underreporting may have occurred primarily because of three clinical factors: 1) less clinical interest in female lung cancer, 2) inadequate diagnostic tools, and 3) clinical misdiagnosis of lung cancer as tuberculosis or other respiratory diseases. First of all, for many years, clinicians tended to believe that women were less likely than men to develop lung cancer, and that belief could have affected their diagnoses. Second, physicians were less capable of detecting lung cancer, because they simply did not have adequate means of discovering the presence of a cancer. Third, mainly as a consequence of these limitations, physicians may have confused lung cancer with other respiratory diseases, particularly tuberculosis. In other words, they may have diagnosed tuberculosis when in fact the patient had lung cancer.

The introduction of antibiotics and other drugs led to a sharp decline in deaths from tuberculosis and other infectious diseases. With isoniazide and these other drugs at their disposal, physicians could suddenly separate the lung cancer patients from the tuberculosis patients, because the TB patients generally improved after drug therapy whereas the cancer patients...
did not. The abrupt drop in tuberculosis mortality rates after the introduction of isoniazide in 1952, and the nearly simultaneous increase in reported lung cancer rates, provide substantial support for my belief that these diseases were often confused clinically. Therefore, as these drugs became increasingly available, underreporting of female lung cancer became less common.

Unfortunately, there is evidence which suggests that today lung cancer in women is overreported. This may occur in one of two ways: (1) clinicians are not always able to distinguish between primary and secondary lung cancer. As discussed earlier, the distinction is of obvious importance for an actual count of primary lung cancer cases. The only truly effective way to determine whether lung cancer is primary or secondary is through autopsy, and few cases of cancer deaths in this country are autopsied. In general, the autopsy rate is about 12 percent. When one considers that this percentage includes all accidental and violent deaths, one understands just how low the autopsy rate for lung cancer may be. This issue becomes even more crucial because primary lung cancer is one of the more difficult diagnoses to establish clinically. (2) The current ICD classification combines primary and unspecified lung cancer into one category. Even if the clinician could correctly distinguish primary from unspecified lung cancer,
the effort would be negated by classification methods. Thus, both primary and unspecified lung cancers are combined in the reports that have described increases in lung cancer in women.

Therefore, changes in diagnostic techniques and changes in disease nomenclature are very important because, speaking from the epidemiologists' point of view, we cannot be sure that the reported trends in lung cancer among women accurately reflect the true incidence of the disease.

Further support for my skepticism regarding an identification of cigarette smoking as the primary cause of lung cancer stems from my own research investigations of this disease. In one study of lung cancer in Spanish surnamed and Anglo women in El Paso, we found a high incidence of the disease in the Spanish surnamed, 64 percent of whom were smokers. Although the same percentage of the Anglo women also smoked, their rate of lung cancer was half that of the Spanish surnamed. We concluded that we had to rule out smoking as the significant factor. Instead, the most important factor in the Spanish surnamed women's lung cancer was their residence from birth to adulthood in adobe houses. These are solidly built, nearly airtight and poorly ventilated structures heated mostly by wood fires, which exposed the women to known carcinogens.
In a much larger study, my colleagues and I examined the relationship of environmental and ethnic factors to cancer mortality by site for every state and region of the U.S. for a twenty year period. We began with a very large number of variables or factors, from which we selected 12, among them cigarette consumption, intake of different kinds of alcoholic beverages, ethnic background, pollution, income, temperature, etc. We used these to derive sophisticated statistical models of the geographic patterns of cancer mortality in the U.S. These models were an attempt to describe by statistical equations the relationships between certain factors and cancer mortality.

We found that all the factors used in the study accounted for only 36 percent of the geographic variation in white female death rates from cancer of the bronchus and lung specified as primary. The best model -- which eliminated the unimportant factors and brought into focus the important ones -- accounted for 29 percent. (In contrast, the models accounted for 89.5 percent of the variation in cancer of the breast.) This indicates that unknown but critical factors were not included in the variable pool from which the model for primary lung cancer was derived. Cigarette consumption, which was included, turned out not to be a factor.
We also discovered a warping in the basic data dividing primary lung cancer from the unspecified category of lung cancer — for both white females and white males. This is important because it shows we can’t be sure lung cancer rates, as reported, present a factual picture.

There are other important considerations required in a balanced discussion of lung cancer in women. For instance, women in increasing numbers have, over the last 35 years, left the jobs they have traditionally performed and have entered work areas formerly reserved for men. Women are now exposed to the same community and work environments as men and there are many unknown carcinogenic potentials in these environments.

The tremendous variety of toxic and carcinogenic airborne chemicals produced by industry and released into indoor and outdoor environments, in our homes and our workplaces, suggest a set of factors which may be important in lung cancer etiology. In 1976, I published, a 30 year study which examined the possible influence of the petro-chemical industry exposures in Houston upon lung cancer mortality, heart diseases, and other respiratory diseases. In certain areas, clustered around industrial facilities, mortality rates from respiratory cancer and other diseases were found to be very high — so high, in fact, that deaths from respiratory diseases nearly doubled over the
last 15 year period. Outside the industrial "pathway," mortality rates had remained stable for twenty years. A similar finding was made by Clemmesen in an industrial city in Denmark. These epidemiological findings are strongly suggestive of the fact that industrial exposure factors underlie the etiology of lung cancer. I might add that I saw no evidence that cigarette smoking could explain the peculiar disease patterns I found in Houston.

Our increasing ability to identify industrial carcinogens has opened many avenues of study and emphasized the importance of industrial environments. Control of these carcinogens at their source offers the most positive potential for prevention of lung cancer.

To date, epidemiological studies of lung cancer have not generally included adequate information about occupational exposures. Frequently, an epidemiologist may get little more than a brief indication of occupation -- such as, "construction worker." This tells us nothing about that person's actual exposure. At the M. D. Anderson, one of the major centers in the U. S. for the study and treatment of cancer, records are even less informative: the vast majority of cancer patients' records simply list "retired" as the occupation. How helpful, then, can we expect the records of private practitioners and community hospitals to be? Even when attempts are made to elicit full
occupational exposure history, the patient’s knowledge and/or memory of previous exposure will undoubtedly be faulty and incomplete. I seriously doubt if the typical industrial worker has an exact record of the hazardous substances, much less the amounts, to which he may have been exposed over a 30-40 year time period. And his wife, who may well describe her occupation as housewife, would undoubtedly know even less about the substances she cleaned from his work clothes all those years.

Still other important areas of research, besides the above mentioned, are emerging in our study of the puzzle of cancer causation. Scientists are beginning to recognize the possible roles played by nutritional imbalances in the etiology of cancer. Although this discipline is still in its infancy and conclusions do not appear to be immediately forthcoming, this new area of research does point to the fact that there exist many scientific unknowns in the study of cancer causation.

From my own studies and from a considerable amount of other research reported in the literature, it is therefore apparent that calling cigarette smoking the primary causative factor in female lung cancer is an oversimplification of a very complex epidemiological problem.
There is a concept widely accepted by public health schools that if many studies, in themselves not based on definable populations, all arrive at the same conclusion, then that conclusion must be valid. This concept disregards the fact that the same intrinsic error in method might produce the same result.

In the first and subsequent Surgeon General's reports, one of the main sets of supportive data was based on the survey of the American Cancer Society. The inadequacy of this survey was recognized and critically analyzed by many of the foremost statisticians and epidemiologists at the time, including Sir Ronald Fisher, the father of statistical methods as we now practice them, Dr. Joe Berkson of the Mayo Clinic, who devised the method of reporting survivals we all use, Professor K. A. Brownlee, for the American Statistical Association, Dr. Donald Mainland, physician and teacher of medical statistics, and numbers of others.

In the Cancer Society study, thousands of women volunteers asked of their acquaintances many questions about their personal habits. No one questions the sincerity of the women, but no business would consider basing its sales policy on
the results of such a study. The proportion of smokers interviewed bore no resemblance to the proportion of smokers in the population, which demonstrated that the women surveyors went out to find smokers. Twenty-five states were not in the study. It was largely an urban New York, New Jersey population of upper middle class people.

An enormous literature has grown up on the subject of smoking. But the bulk of the evidence is statistical. In no single instance has cancer of the lung been produced in any meaningful animal experiment using tobacco smoke inhalation, even though millions of dollars have been spent in the effort.

In all my years as a cancer epidemiologist, I have seen many theories studied and debated. But only in the instance of the smoking theory has the theory itself become sacrosanct. Any of the many valid scientific observations which refute the theory are discounted as bizarre. Many a doctorate on human psychology will be earned in the future, when the actual cause of lung cancer becomes known, which will attempt to explain the intensity of the promotion of this unproved theory.

Science, by explaining one fragment after another of genetic, biochemical, somatic, behavioral, environmental, and nutritional information, is building gradually the structure, which, upon completion, will help us understand the genesis of cancer. It is becoming increasingly clear that there is no single, simplistic answer to the question of what causes respiratory cancer. It is hardly in the best interest of either science or government to create the illusion that an attack on a single lifestyle factor will provide the solution for such a complex problem. The proposed legislation creates this illusion and should not be passed into law.
I am a social psychologist, and since 1962 have been Professor in the Department of Mass Communications at the University of Denver and Director of the University's Center for Mass Communications Research and Policy. From 1970 through 1978 I also was Chairman of the Department of Mass Communications. For over 35 years I have conducted research and published in the fields of social relations, attitudes and public opinion, communications, public health and the sociology of politics. I have appeared for the Federal Trade Commission in several cases as an expert witness on the effects of product advertising. I have authored or co-authored four books and numerous monographs, commissioned policy papers, and book reviews. Attached to this statement are my biography and a list of my publications.

I have examined in detail various documents that appear to provide the grounding for the "cigarette labeling" proposal contained in Section 6 of S. 772. I have given particular critical analytic attention to the May 1981 Federal Trade Commission "Staff Report on the Cigarette Advertising Investigation." Additionally, I have reviewed, from the perspective of an expert on communications effects and public opinion survey research, the principal public opinion surveys.
and effects studies on which the key conclusions of the FTC Staff Report apparently are based.

I shall focus my testimony on the scientific bases for rejecting the rotational warning proposal set forth in S. 772 and the FTC Staff Report. Put succinctly, that proposal is unnecessary because people already have the information sought to be provided and misguided because it assumes that telling people more about the claimed health hazards of smoking will affect their smoking behavior. That assumption, which is inherent in both the FTC Staff Report and the present bill, is a prime example of wishful thinking without basis in fact.

The premise that the American public lacks sufficient information about smoking and health claims is utterly unsupported by the FTC Staff Report. That Report relied on a handful of disparate, isolated and unintegrated "studies" and public opinion polls that bear no intellectual, methodological or scientific relationship to each other. They do not relate to any recognizable theoretical body or tradition; they do not emerge from any scientific model; nor do they reflect any system of integrated hypotheses or hypotheses-testing that are grounded in scientific empiricism. Public policy should never be based on such singular, isolated, and unintegrated ad hoc "studies."
Moreover, the Report presents as an authoritative "data base" a handful of disparate public opinion polls (erroneously misinterpreted as tests of public information levels) plus a so-called focused interview study (again erroneously misinterpreted as a carefully controlled experiment) based on highly selected, biased "intercept" samples, rather than on representative area probability samples, the only scientifically acceptable sampling procedure for public opinion surveys. Hence the data base on which the FTC Staff based its recommendations is so seriously flawed that it cannot pass even the most minimal scientific muster. The Report's "findings" are without value as a grounding for public policy formation.

These fundamental defects aside, the FTC Staff's claim that significant sectors of the population are uninformed about the dangers of cigarette smoking has no basis in fact. Data from the very studies the FTC Staff selected to cite in their Report indicate just the contrary, as does the 1979 Surgeon General's Report:

"The public health campaign against cigarettes has produced notable changes in public awareness of the health consequences of cigarette smoking. It appears that the dramatic changes noted in adult smoking, especially among middle-aged males and certain professional groups can be attributed largely to the effectiveness of information and
educational campaigns since 1964. Moreover, Warner has estimated that the effect of specific "events," such as the 1964 Surgeon General's Report, on cigarette consumption (mean number of cigarettes consumed per day) may appear small and transitory, but that the cumulative effect of persistent publicity appears to have reduced consumption by 20 to 30 percent below its predicted 1975 level." 1979 Report at 19-9. (Emphasis added.)

As a promotional effort, the various government and private anti-smoking campaigns in this country, together and cumulatively have been remarkably successful in informing the largest possible number of Americans of the claimed dangers of smoking cigarettes.

Still, many Americans choose to smoke cigarettes. They do so not because they are unaware of the health hazards that are claimed to accompany such behavior. Smokers choose to smoke cigarettes despite their awareness of claims that risks to their health may be involved. They do so for myriad of reasons other than lack of "information."

The motivations to smoke or not are manifold and complex. "Information" about smoking and health claims alone must be viewed as just one possible relatively weak factor among such powerful causal factors as personality; socialization experience; learning experiences; beliefs and values; peer pressures; physiological, metabolic, and chemical balances.
and imbalances, rel... background, loci of personal control and such. The scientific literature suggests that above all conformity to group norms is the most powerful motivational factor in influencing smoking behavior -- not exposures to advertising or other communications.

This very point is confirmed by the testimony submitted to this Committee by Dr. Mortimer B. Lipsett, Director of the National Institute of Child Health and Human Development. Dr. Lipsett there stated "that the most important factors influencing experimentation with and acquisition of smoking are social and familial relationships." (P. 49).

Thus, even if every man, woman and child in the country could score 100% on any test of information regarding the possible hazards that may flow from that behavior, it is unlikely that the current rates of cigarette smoking would be affected significantly.

Perhaps the major reason for this dichotomy between awareness and behavior is that the latter is far more a function of what we are willing to believe than of what we appear to know. And since beliefs serve emotional as well as intellectual functions, often simultaneously, they may not always be "logical" or "consistent." Thus, without discomfort of any sort we often hold, and hold on to, beliefs that appear to be simultaneously contradictory and irrational (e.g., the belief in science and the belief in astrology). Changing any single belief, other than our most central and cherished
beliefs, does not necessarily produce appropriate changes in all others that may apply to a given phenomenon. If they are to serve as guides to behavior, beliefs must above all first be personalized and internalized.

Beliefs concerning health usually have a probabilistic aspect to them. That is to say, a good portion of our beliefs about health is concerned with "likelihood":

1. The likelihood of coming down with a serious incapacitation or fatal condition or disease; and
2. The likelihood that certain actions to be taken by the believer will actually prevent, reduce, or eliminate that threat.

As a consequence, in order to persuade individuals to accept a particular health warning, the warning information must be processed through the psychological filters of message recipients' subjective beliefs that their susceptibility to severe health threats will indeed be substantially lowered or eliminated with compliance. Additionally, message recipients must be given a guarantee of a specific benefit to be experienced as a reward for compliance. The positive attributes of the promised benefit must fit in with message targets' beliefs about which is more gratifying -- their current behaviors or the alternatives proposed from an anonymous outside source such as the government.

The "Russian roulette" labeling proposals of S. 772 completely fail to take these personalizing problems of risk
perception, motivation and gratification dynamics, and modification of health beliefs into account. Indeed, they are counterproductive in several significant respects.

1. By reflecting what appears to be yet another Federal government manipulative public relations gimmick, the credibility of warning labeling overall may be seriously eroded.

2. Turning away from the one consistent current message to four separate warnings (not all equally relevant to everyone) simply serves to dilute the potential effect the current message may have through repetition by a factor of four. Generally speaking, public attentiveness to and awareness of a particular claimed health danger or threat depends to a considerable degree on how frequently the warning claim is repeated.

3. None of the four statements contains any accurate exposition of the actual claimed risk. Consequently, no effect on consumers' risk perceptions can be expected. The statement, for example, that "cigarettes now greatly reduce the risks to your health" is totally meaningless without explicit metrics for the phrase "greatly reduces." The assertion as it stands is gross and imprecise and therefore is potentially more confusing than enlightening.

4. The proposed statements can be expected to produce a "boomerang" effect -- a result precisely the
opposite of what was intended—by negative reinforce-
ment. For example, the proposed statement claiming that
“cigarette smoking by pregnant women may result in
miscarriage, premature births and low birth weight
babies” implies that smoking is potentially hazardous
only to pregnant females rather than to all women. By
inference, this label actually sanctions cigarette
smoking for all women so long as they are not pregnant.

Further, we all have witnessed women who smoke and
who give birth to perfectly normal infants as well as the
reverse. Nonsmokers who unfortunately produce offspring
with a variety of defects. How will the proposed labeling
cope with these realities? Women who know other women
who smoke and yet give birth to healthy children would be
likely to disregard this warning.

5. The proposed warnings are based primarily on
the appeal to fear. Label message recipients would be
told by inference that they can avert the dangers of
cancer, heart disease, birth defects and so on only if
they do not smoke cigarettes. Often, when consumers
encounter such strong or exaggerated fear appeals, they
become immobilized to the point where they resort to
“defensive avoidance” rather than to taking a recommended
action.

6. The most useful and productive function of
health warning labels is to reinforce and to serve as
reminder for what consumers already know. For this reason alone, tampering with the current well-known Surgeon General's warning statement would appear to be imprudent.

Several years ago I testified as an expert witness concerning a proposal to place rotating health warning labels on alcoholic beverages and in advertisements for such products. I explained that such labels would be ineffective for much the same reasons that I have outlined here. The Department of Health and Human Services referred to my testimony in a report critical of the proposed rotating warnings for alcoholic beverages, and it is my understanding that Congress ultimately rejected the proposal. Analytically there is no significant difference between that proposal and the present one regarding rotating cigarette warning statements. I believe the same result is warranted.

It is apparent that current public information programs in the cancer and allied health fields are in embarrassing disarray. Insipid slogans frequently are offered in place of facts and precise instructions for acting appropriately. Giants like the "Great American Smoke-Out" substitute for integrated and sustained sober communications and meritorious programs of demonstrated merit. And truly essential information regarding, for example, the identity of the best cancer specialists in town is quite consciously withheld from concerned publics. Added to this state of affairs
are the inconsistent positions that the health establishment itself takes from time to time with regard to such consequential matters as the efficacy of annual screenings for cancer of the cervix/uterus, the dangers of early or frequent mammography, the carcinogenic character of food additives, the positive/negative attributes of cholesterol, and conflicting claims as to whether most cancers are environmentally rather than genetically or virally induced.

The purpose of information should be to enlighten by virtue of its ability to reduce uncertainty. Contemporary public health efforts appear to be doing just the opposite -- adding to the public's uncertainty -- by virtue of their imprecision, obfuscation, gimmickery, clutter, inconsistency and avoidance of embarrassing and difficult truths. Under such circumstances the public's beliefs in the efficacy of actually preventing cancer and heart disease is undergoing severe testing. Small wonder that the laetrile and coffee enema dispensers flourish in today's gimmick-laden public communications atmosphere. For the Federal government to contribute to this sorry situation with its own groundless gimmickery, such as the rotational warnings proposed by S. 772, will make more sober and promising health education efforts in the future all the more difficult to carry out with success.
STATEMENT BY DR. L.G.S. RAO REGARDING S. 772

My name is Dr. L.G.S. Rao; I am Senior Biochemist at Bellshill Maternity Hospital in Glasgow, Scotland. I obtained my Ph.D. in Biochemistry from the University of Newcastle in 1966. I am the author of numerous scientific publications and have made presentations at scientific meetings in Europe and the United States.

My experience in clinical biochemistry over the past 20 years has been varied and has resulted in the development of interests in several methodological and clinical problems including perinatal medicine. Over the past ten years, I have become deeply interested in the investigation of problems relating to pregnancy and early childhood, and in particular the causes of the high incidence of low birthweight and perinatal mortality which is found among the poorer patients of Bellshill Maternity Hospital. I have found, as have other
researchers in this field, that the biological characteristic of the poorer social groups that is of relevance to fetal growth retardation is their poor nutritional status and not, as has been claimed, their smoking habits. This finding could be of considerable practical importance because nutritional deficiencies can be corrected by dietary advice or supplementation. A program of such dietary intervention could lead to a striking reduction in the incidence of low birthweight and perinatal mortality and morbidity.

S. 772 states, at pages 1-2, that "Congress finds that the risks of miscarriage, stillbirths, premature births, and child weight deficiencies for pregnant women who smoke are higher than for pregnant women who do not smoke." Based on the medical research I have engaged in during the past 10 years and my review of the relevant scientific literature, it is my opinion that a causal connection has not been scientifically established between maternal smoking and
the various problems related to pregnancy referred to in S. 772. As discussed below, and in the statement which I submitted to the Committee on Labor and Human Resources in 1982 regarding S. 1929, the claim that maternal smoking is causally related to miscarriage, stillbirths, premature births and birthweight deficiencies, is, based on unreliable evidence. Furthermore, there is both direct and indirect evidence against such a causal theory.

The main defect in the studies which are claimed to support the causal theory is that they have not corrected for the factors already known to be of causal significance with regard to problems of pregnancy and early childhood. There also appears to be a substantial amount of bias in the interpretation of the data reported in these studies. What is of most concern to me, however, is that acceptance of unproven claims (such as the causal theory) as 'scientific fact' will divert the attention of the public and the scientific community from efforts aimed at ascertaining the actual causes of
miscarriage, stillbirths, premature births and birthweight deficiencies.

As far as birthweight deficiencies in smokers are concerned, I have direct evidence that smoking is not the cause of low birthweight. My initial observations on smoking in pregnancy and birthweight showed that in the upper socio-economic groups there was no difference in the percentage of low birthweight babies born to mothers who smoke compared to those who did not smoke. A difference in the incidence of low birthweight between babies born to mothers who smoke compared to those who did not smoke was found only in the lower socio-economic groups. This observation indicates that some factor other than smoking, which is peculiar to the lower socio-economic groups, was the cause of the excess of low birthweight babies in these groups of mothers.

The most likely difference between the upper and lower socio-economic groups that could be of biological significance
is maternal nutrition which is already known to be deficient in
the lower socio-economic groups. This theory was verified by
measuring the protein intake in pregnancy and relating it to
birthweight in smokers and non-smokers. It was found that
there is no higher incidence of low birthweight babies between
mothers who smoke compared to those who do not smoke, provided
that they are not deficient in nutrition during their
pregnancy. In mothers who had a normal protein intake, the
percent of low birthweight babies born to smokers and
non-smokers was 3.81 and 3.78 respectively, an insignificant
difference. Further details regarding this study are set forth
in my 1982 statement.

The reported excess in the rate of stillbirths in
mothers who smoke in pregnancy is associated only with the
poorer socio-economic groups. Excess rates of stillbirths are
not reported for smoking mothers from the upper socio-economic
groups. Further evidence of the lack of any significant excess
in the rates of stillbirths and neo-natal (first two to three weeks of life after birth) deaths in mothers from the upper socio-economic group who smoke in pregnancy are given in the attached paper from *The Lancet* (May 5, 1974, p.976) and further details are set forth in my 1982 statement.

My own studies on stillbirths and neo-natal deaths (perinatal mortality) in relation to smoking and pregnancy are in agreement with the above referenced findings. I have found that perinatal mortality in smokers from the upper social groups was no greater than those of nonsmokers (in fact smokers had a lower perinatal mortality, 2.14% compared to nonsmokers, 3.22%). In the poorer social groups babies of smokers did appear to have a somewhat higher perinatal mortality than those of nonsmokers, 3.79% and 2.78%, respectively. If smoking during pregnancy is the cause of higher rates of perinatal mortality, why does it occur only in certain groups of mothers and not in others? It can scarcely be argued that the
so-called "effects" of smoking respect the upper social classes and affect only the poor and underprivileged mothers. The evidence so far suggests that the reported excess rates of perinatal mortality found in certain groups of mothers is attributable, not to smoking, but to deficient maternal nutrition in pregnancy that is frequently found in the poorer social groups.

The same arguments also apply to the alleged higher rate of prematurity found in some groups of smokers, because prematurity is also associated with poor socio-economic status. Mothers from poorer social groups have more than twice the prematurity rate as mothers from the upper social groups. See Table No. 1 and related text from my 1982 statement which discusses the problem in greater detail.

As far as spontaneous abortion is concerned, the evidence in favour of the causal theory is scanty and controversial. The most serious deficiency in the evidence
cited in support of the causal theory is that maternal nutrition, which has a crucial effect on problems in pregnancy, has not been taken into account and corrected for. There is a great deal of direct and indirect evidence that deficient nutrition and not smoking is the cause of reproductive problems found in some groups of mothers.

A review of the literature on the causes of birth defects, spontaneous abortions and prematurity shows that these disorders are consistently associated with a history of reproductive failure, involving infertility and a higher incidence of low birthweight infants. Much of the epidemiological data available implicates nutrition as the main cause of these disorders of reproduction.

It is well-known that severe nutritional deficiencies cause complete infertility in women, as seen in Holland, Germany, and Russia during World War II. Examples of such infertility in peacetime are afforded by women who are
suffering from anorexia nervosa, a disease in which the patient refuses food and undergoes a severe weight loss. These patients do not menstruate at all, but successful treatment which results in weight gain restores menstruation and fertility. There are numerous studies in animals which have highlighted the various nutritional deficiencies, including those of essential trace elements, that cause infertility. Between the extremes of total infertility caused by starvation and normal reproduction associated with good nutrition, there is a grey zone (penumbra) in which nutrition is not so deficient as to cause infertility but is not good enough to produce healthy babies. (See Fig. 1.) Examples of groups of women in this grey zone can be found both in wartime and in peacetime. Severe food shortage is normally accompanied by epidemic of spontaneous abortions and birth defects, as was seen in Germany during the war years (Fig. 2). The highest spontaneous abortion rates occurred when the women were
recovering from complete infertility, as the food supplies improved and they had to cross the grey zone to reach normal reproductive health.
A study in Sweden on the causes of amenorrhoea found that 49 out of the 51 women who were not menstruating were below the Norrøn standard weight for height and 25 of these were dieting for cosmetic reasons. (Holmberg N.G. and Nylander, I. (1971) Acta Obstet. Gynaec. Scand. 50, 241). It has been known for a long time that women with a history of reproductive failure involving infertility also have a high incidence of recurrent abortions, birth defects, prematurity and low birthweight babies. The poorer socio-economic groups also have an excess of birth defects and recurrent abortions in addition to having a higher incidence of low birthweight babies (See Fig. 3).
Fig. 5. All types of malformations by social class 1973 per 1,000 live and stillbirths.

Source: Registrar General for Scotland.
The social gradient in reproductive performance could have the same nutritional basis as that found in clearly malnourished women because the poorer social groups are known to have some nutritional deficiencies. Since maternal smoking is reported to be statistically associated with a higher incidence of low birthweight babies in only the poorer women with nutritional deficiencies, it is more likely that the reported higher incidence of birth defects, spontaneous abortions and prematurity in some mothers who smoke in pregnancy could also have a nutritional cause.

CONCLUSION

In conclusion, it has not been scientifically established that smoking is a cause of reproductive problems in mothers who smoke during pregnancy. Furthermore, treating such unproven claims as scientific fact may do harm to the mothers concerned, because it diverts the attention of the public and the scientific community from the real causes of these reproductive problems which can only be solved by better prenatal care of the mother with special attention to her nutritional status during pregnancy.
Statement of Henry Rothschild, M.D., Ph.D.
in response to
S. 772, the "Smoking Prevention
Health and Education Act of 1983"

I am Henry Rothschild, a medical doctor and doctor of
philosophy specializing in genetics, molecular biology, and
oncology, currently professor of Medicine and Anatomy at the
School of Medicine, Louisiana State University, New Orleans,
Louisiana.

Since 1962 I have been engaged in research involving
the genetic basis of disease and am director of the Louisiana
Ethnogenetic Disease Association.

I currently serve as consultant to the Louisiana
Department of Health and Human Resources and am a member of the
Research Committee of the American Heart Association of
Louisiana. I have published over 40 scientific articles. My
Curriculum Vitae and list of publications are attached.

Studies conducted in Louisiana have demonstrated an
unusual distribution of lung cancer mortality. The lung cancer
mortality in white males in Louisiana is the highest in the
nation. From 1954 through 1976, the 27 southern nonurban
parishes ranked in the top 3% nationally for this disease.
Since 1976, the incidence of lung cancer in these parishes has
risen. Studies that I have been conducting since 1975 on this
population indicate that genetic factors may play a significant
role in this excess mortality from lung cancer.
In our initial investigation we interviewed the next-of-kin of 284 of the 815 persons who died of lung cancer from 1971 through 1977 in the 10 southern, nonurban parishes. We found that 188 (66%) of the decedents had been employed for at least six months as sugarcane-farm workers. The relative risk of lung cancer mortality for sugarcane-farm workers was more than twice that of the controls. Neither employment in other industries nor tobacco consumption could account for the elevated risk of lung cancer mortality associated with sugarcane farming. (Rothschild and Mulvey, 1982)

We then began to explore the thesis expressed by many investigators, including a group from the National Cancer Institute (Coffman et al., 1982), that there may be "an inherited predisposition to lung cancer." Studies have shown that there are genetic factors associated with lung cancer pathogenesis. It has been found that first-degree relatives of individuals diagnosed as having lung cancer have higher rates of lung cancer that cannot be accounted for by age, sex, birth cohort, or cigarette smoking. It was shown, for example, that a non-smoking first-degree relative of an individual with lung cancer has about a fourfold risk of dying of lung cancer when compared to controls. (Tokuhata and Lillienfield, 1963; Tokuhata, 1964) A similar study reported an increased risk of lung cancer mortality in siblings of individuals with lung cancer. (Fraumeni et al., 1976) It has also been shown that first-degree relatives of lung cancer patients have higher
rates of pulmonary dysfunction that cannot be accounted for by age, sex, race or smoking habits. The authors concluded that the etiology of lung cancer included a familial component. (Cohen et al., 1977)

The existence of a genetic factor has been recently confirmed by a study of the lung cancer incidence among relatives of lung cancer patients. (Lynch et al., 1982) A genetic factor has also been shown to exist with respect to other cancers as well. (Esophageal cancer: Pour et al., 1974; pancreatic cancer: Friedman and Fialkow, 1976; kidney cancer: Cohen et al., 1979; and bladder cancer: Manboubi et al., 1981.)

The argument for the genetic regulation of tumor incidence (Bodeau et al., 1974) is supported by results of studies indicating that many mouse strains differ in their incidences for specific tumor types (Murphy, 1966). Human populations also vary in their predispositions for specific tumors. These differences may be caused by genetically based differential susceptibilities. (Reif, 1981)

Studies of cancer rates among different ethnic groups further support this proposition. For example, the rate of increase of lung cancer has been greater for blacks than whites while in Americans of Mexican and Chinese extraction the incidence rates of lung cancer have been greater than for whites. American Indians, on the other hand, have a lower incidence of lung cancer than whites. (Buell et al., 1968)
Fraumeni and Mason, 1974; Craigan and Fraumeni, 1972) Chinese in Hong Kong have an unusually high prevalence of adenocarcinoma of the lung, a relative infrequent cell type in other areas. (Belamaric, 1969)

In analyzing our data we obtained results that indicate a strong genetic component for lung cancer in the Louisiana population. (Cook et al., 1982; Rothschield et al., 1983, unpublished data). Of the 446 people who died of lung cancer from 1976 to 1979, we interviewed the next-of-kin of 33%. Of the 336 case families, 86 (25.5%) contained first-degree relatives of the index case who had had lung cancer, whereas among the control (spouse of the case) families, only 30 (9.60%) did. Among the 2708 first-degree relatives of index cases, there were 104 (3.84%) lung cancers; among 2206 control relatives there were 35 (1.59%). This aggregation of lung cancer could not be explained by differences in age at death, family size, or other factors.

Sixteen (4.6%) of the case families had two or more first-degree relatives who had had lung cancer, compared with only 5 (1.63%) control families (p < 0.05) who did. First-degree relatives of persons with lung cancer had, therefore, a lung cancer risk three times that of the controls.

When case and control relatives were compared by stepwise logistic regression, we found that a person's relationship to the index case, and the square of a person's age were the strongest predictors of lung cancer outcome, even
after controlling for the effects of age, sex, the cumulative exposures to risk occupations/industries, and the number of pack-years a person smoked cigarettes.

We are currently conducting a study of this population to determine whether a gene (or genes) may be linked to the pathogenesis of lung cancer.

We are identifying index cases with more than one first-degree relative affected by lung cancer, and testing for protein polymorphisms and the presence of viral and cellular oncogenes in these high-risk families. By these means we can determine whether a linkage exists between the lung cancer and a known chromosomal marker.

To date, 56 index cases have been recruited. Thus far, two index cases have each had two first-degree relatives with lung cancer. One of these, a 75-year-old man diagnosed as having well-differentiated squamous-cell carcinoma, had a brother who died of squamous-cell cancer, a half-sister who died of adenocarcinoma, a brother who died of stomach cancer, and a niece who had uterine cancer. A third index case has three first-degree relatives with mesothelioma of the pleura and two first-degree relatives with cancers in other sites.

The DNA of living first-degree and second-degree relatives of the index cases is being analyzed for the presence of genetically transmitted tumor-virus sequences and altered oncogenes. These studies may demonstrate an association between the tumor and a polymorphic marker allele, which may be viral in composition, an oncogene, or some other form of genetic marker.

If we can isolate such genetic markers, it will be a major step toward unravelling another aspect in the mystery of lung cancer causation.

Henry Rothschild, M.D., Ph.D.
STATEMENT OF
JACK MATTHEWS FARRIS, M.D.

My name is Jack Matthews Farris. I am Associate Dean of the School of Medicine and Emeritus Professor of Surgery at the University of California at San Diego. I practiced surgery in the Los Angeles area for more than 25 years as Staff Surgeon at the Good Samaritan Hospital, Los Angeles; Chief of Surgical Service at the Harbor General Hospital, Torrance, California; and Chief of Surgical Services at the California Hospital Medical Center, Los Angeles. I also served as Senior Consultant to Surgery for the Veterans Administration, for whom I directed the surgical activities at several of their institutions in California.

I am on the Board of Directors and Board of Trustees of the Scripps Clinic and Research Foundation, and was on the Board of Directors for the Medical Research Association of California. I am a member of numerous medical societies where I have served on advisory committees. I have published approximately 74 papers in scientific journals and have contributed several chapters for surgical textbooks. My full Curriculum Vitae and list of publications are submitted with this statement.

Throughout the past 25 years, I have followed with interest the evidence for and against the thesis that cigarette smoking has a causal relationship with cancer of the lung. This interest has been stimulated by my operation on more than 8,000 patients, many of whom suffered from cancer in various parts of the body, including lung cancer.
In 1955, I submitted statements to the House of Representatives and the Senate in which I said that I did not believe that lung cancer had been shown to be caused by cigarette smoking, and that any conclusion to the contrary was not justified. Last year, I submitted statements to the House and the Senate stating that my opinion on this matter remained firmly the same.

I am still of the same opinion today. There are numerous complexities in the behavior and cause of cancer in all parts of the body. I believe that, when we learn how and what causes cancer, we might well find that cigarette smoking has little or nothing to do with the genesis of carcinoma of the lung.

That the smoking and health controversy continues is demonstrated by many studies and findings that cannot be explained by a smoking causation hypothesis. I would like to mention just a few of these.

1. Persons who have never smoked get cancer of the lung that is indistinguishable from those cancers that are reported in smokers. A recent study has shown a significant and increasing incidence of cancer of the lung in non-smokers.

2. In the early 1960’s researchers, without regard to smoking in the population, predicted that the death rate in lung cancer would level off in the next decade or so. Studies since then have supported their prediction.
3. Cancer is rarest in those parts of the body most in contact with cigarette smoke, for example, lip, tongue, trachea and larynx. Tracheal cancer continues to be an extremely rare disease, even though the trachea, which is part of the respiratory tract, is more exposed to tobacco smoke than the lung. The larynx is also more exposed to tobacco smoke than the lung; nevertheless, the incidence of laryngeal cancer has remained relatively stable during the last quarter century and has not followed the reported increase in lung cancer.

4. Lung cancer often originates in areas of the lung that have little or no contact with tobacco smoke, that is, in the peripheral areas of the lung, rather than in the hilar, or central, region.

5. Although there is no reason to suppose that there is greater exposure of tobacco smoke at the site of any given cancer in the lung, lung cancer usually appears as a solitary lesion. Metastatic cancer, on the other hand, such as that which spreads from the breast, may produce hundreds or thousands of minute lesions in the lung field. Moreover, once a lobe containing cancer of the lung has been removed, a second primary cancer of the lung rarely develops, although the lung fields have been exposed to the same amount of inhaled smoke.

6. The vast majority of heavy smokers don’t get lung cancer. One theory that is being investigated is that the immune system has a lot to do with the development of this disease.
7. Efforts have failed to produce the kind of lung cancer reported to be statistically associated with smoking in tests on animal exposed to smoke. One study has shown that a group of hamsters exposed to cigarette smoke lived longer than those not exposed.

8. Some studies find that in workers exposed to certain chemicals the smokers have a lower incidence of cancer than the non-smokers. For example, this has occurred in workers exposed to chloromethyl ethers.

9. A 1982 study of British male civil servants and other residents found that there was a lower rate of lung cancer mortality among the inhalers than among the non-inhalers. This confirms previous findings in a large study of British doctors.

It is clear that the smoking and health controversy has not been resolved. It is also clear that cigarette smoke cannot be the total answer to cancer of the lung.

All of us who are interested in this controversial subject, have one thing in common – we are all anxious to see a discovery for the prevention of cancer, which inexplicably claims so many lives today. The problem won’t be solved by legislating the cause of disease. The problem can only be solved by continued exploration of the various theories and factors suggested by the research to date.
The CHAIRMAN. Thank you, Dr. Sommers.

Dr. Fisher?

STATEMENT OF EDWIN R. FISHER, M.D., PROFESSOR OF PATHOLOGY, UNIVERSITY OF PITTSBURGH, AND DIRECTOR OF LABORATORIES, SHA DysIDE HOSPITAL, PITTSBURGH, PA.

Dr. Fisher. Thank you, Mr. Chairman.

I am Edwin R. Fisher of Pittsburgh, Pa. I am currently professor of pathology at the University of Pittsburgh School of Medicine and director of laboratories at the Shadyside Hospital in Pittsburgh, Pa.

In addition, I am a consultant in pathology at the Veterans Administration Hospital in Pittsburgh and the Brownsville General Hospital in Brownsville, Pa. I am a 1947 graduate of the University of Pittsburgh School of Medicine. I received postgraduate training at the Cleveland Clinic, Cleveland, Ohio, and the National Institutes of Health, Bethesda, Md.

I was certified by the American Board of Pathology in both anatomic and clinical pathology in 1952. I am a member of the honorary medical society Alpha Omega Alpha and Sigma Xi, the honorary society for scientific advancement.

I was the recipient of the Parke-Davis Award in Experimental Pathology in 1963 and the Man of the Year in Medicine in the city of Pittsburgh in 1966.

I am a member of many scientific societies, including the American Association of Cancer Research and the American Society for the Study of Atherosclerosis.

I am the author of 494 scientific publications in American and international journals and textbooks.

I have served on the editorial boards of the Journal of Cancer and of the American Journal of Clinical Pathology, as well as the board of scientific directors of Ellis Fischel Cancer Hospital, Columbia, Mo., and the board of reviewers for the American Society of Atherosclerosis. I am also the project pathologist for the national surgical adjuvant, breast and colon projects of the National Cancer Institute.

I should first like to direct my remarks to personally conducted experiments concerning the possible atherogenic effects of nicotine per se. Atherogenesis is the process that results in the disease which we commonly regard as hardening of the arteries, or more technically called atherosclerosis. We have also conducted such experiments with actual cigarette smoke which, of course, includes the relationship of carbon monoxide to the atherosclerotic process.

Reprints describing these experiments are being submitted as part of this statement. In these experiments it was clearly demonstrated in the rabbit that realistic doses of either nicotine or cigarette smoke failed to initiate, exacerbate, or otherwise influence the atherogenic process in that species.

You will notice that I used the designation "realistic doses." I think that is very important. There have been some studies that have exhibited minor or highly questionable changes with the use of an equivalent dose of 600 or more cigarettes a day in man. This
is such a large number that, I think man would find it difficult to find the time to smoke them.

Another point which I would like to emphasize is this. One could just say you found nothing in the rabbit, but can the experience in the rabbit apply to man? What the scientific experiment in the laboratory can do, however, is indicate where we should look and perhaps what we should look for in the clinical setting.

With those two points in mind, I should like to continue. It might be well to emphasize that there is no pharmacologic or other study of any scientific validity or acceptability to me that indicates that nicotine adversely affects coronary blood flow. Indeed, most of the studies reveal that this agent actually accentuates and enhances coronary blood flow.

Within the last month or so, the New England Journal of Medicine reported a study that dealt with myocardial infarction—commonly called heart attack—and the nicotine and carbon monoxide levels of cigarette smoke. Incidentally, the study also examined the tar yields of cigarettes in relation to heart attack but did not report these data because, according to the authors, results for tar paralleled those for nicotine. The authors found, contrary to what they expected, that the nicotine and carbon monoxide levels of cigarettes did not affect the risk of heart attack. This finding supports the view that neither nicotine nor carbon monoxide, as found in cigarette smoke, causes or contributes to coronary heart disease.

I have reviewed the scientific literature on the subject of coronary heart disease and smoking and find other studies that support this view. For example, studies of workers exposed to carbon monoxide over long periods of time find that the exposed workers do not have an increased incidence of atherosclerosis compared to the general population.

Other studies raise serious questions about the smoking causation hypothesis. For example, studies of twins in Sweden are very illuminating on this subject. When the researchers looked at monozygotic twins—that is, identical twins—they found that there was no increased rate of coronary heart disease in the smoking twin as compared to the nonsmoking twin. This led the researchers to conclude—and I might add justifiably—that their studies can be interpreted as showing that both the development of IHD (ischemic or coronary heart disease) and death from it are under a relatively strong genetic influence.

Likewise, a 1979 study of cardiovascular disease in Switzerland found that while Swiss women have increased their smoking over the last quarter century, their rate of cardiovascular disease has declined significantly during the same period.

Quite recently the Framingham study in this country received attention in the lay press in connection with a study dealing with cardiovascular disease and hormones. In this study the authors found that in older men a statistically significant association existed between cardiovascular disease and elevated levels of certain hormones. The researchers, however, found no association between cardiovascular disease and smoking. This study followed a 1976 study of younger men, age 32 to 42, by one of these researchers in which he found a statistically significant association between elevated hormone levels and heart attack. There was, however, no sta-
tistically significant association between smoking and heart attack. Although the relationship of hyperestrogenemia and coronary heart disease is somewhat of a scientific paradox, the data relating to cigarette smoking indicates that in these cohorts of patients smoking is unrelated to coronary heart disease.

S 772 purports to find that cardiovascular disease accounts for nearly one-half of the deaths in the United States and that one-third of these deaths are associated with smoking. The fact is, however, that no studies have been done that demonstrate scientifically that smoking is a cause of coronary heart disease. Nor has it been demonstrated that quitting smoking lowers the incidence of this disease.

Recently, there has been a good deal of publicity in the lay press about a study dealing with multiple risk factor intervention trials, popularly called MR_FIT. This program grew out of a 1970 initiative by the National Heart, Lung, and Blood Institute to conduct broad-based intervention trials with regard to smoking, cholesterol, and blood pressure. The trials got underway in the early seventies and to date have cost over $100 million. The results were published last September in the Journal of the American Medical Association.

In the introduction to the study, the authors state that “convincing demonstrations of the favorable effect of risk factor modification on CHD morbidity and mortality were not on hand by the 1960’s.” Obviously, I might interject, they were not on hand in the 1970’s and just became on hand in 1982 with the report of the results of the MR_FIT study.

Simply put, when this study was planned, the scientific evidence then available did not demonstrate that cessation of smoking or a lowering of blood pressure and cholesterol levels would have any impact on lowering the incidence of coronary heart disease. Yet, if one reads the testimony of last week’s hearing, he cannot be helped but impressed with the statements that, “We know that smoking causes coronary heart disease. We’ve known that for decades.” $100 million worth of decades.

After 6 years of followup, however, there was no significant difference in the overall rate of cardiovascular mortality between the intervention group and the usual care group, even though there was a 2-to-1 reduction of smoking in the intervention group compared to the usual care group.

Moreover, the experience of the United States is consistent with a recently published intervention study in Norway. That study found that reduction in smoking had no statistically significant effect on the incidence of coronary heart disease. In addition, a United Kingdom study found no statistically significant differences in total mortality or in coronary heart disease mortality between the smoking intervention group and the normal care group.

In short, these intervention studies find that cessation of smoking will not lead to a reduction of coronary heart disease. Moreover, they are inconsistent with the hypothesis that smoking causes coronary heart disease.

I would now like to turn briefly to the so-called passive smoking issue. My careful review of the literature, confirming the conclusions based upon my own experimental data and the related work
discussed, reveals again a lack of scientific information which would allow me to conclude that atmospheric tobacco smoke or its constituents represent a health hazard to nonsmokers.

Accounts relating adverse effects of cigarette smoke on angina patients, that is, persons who suffer chest pain, probably as a result of arteriosclerotic heart disease, should not be interpreted as indicating that cigarette smoke is etiologically related to the arteriosclerotic process. One might again interject that even the Surgeon General's own reports indicate that it is unlikely that smoking is an influential factor in angina pectoris.

This study, which involved 10 or fewer patients—that is, the study that is commonly cited as evidence that passive smoking is a danger to the nonsmoker, particularly the one with heart disease—was based on 10 or fewer patients with angina pectoris—hardly a scientific or valid sample. It led the author to conclude that atmospheric tobacco smoke aggravates the condition of persons with preexisting heart disease. This conclusion, however, must be evaluated in light of the fact that the end point of the study was highly subjective, that the stress factor was not controlled, and that sham smoke or other environmental impingement was not used. In other words, not only was the sample small, but the scientific design was exceedingly poor. Yet, this work—reemphasize—is still cited as one of the bases for the claimed hazard of smoke to nonsmokers.

In summary, my own experimental work and review of the scientific literature lead me to the conclusion that cigarette smoking has not been scientifically established to be a cause of cardiovascular disease. As to the so-called passive smoking issue, there is a lack of scientific information also incriminating atmospheric tobacco smoke as a health hazard.

Thank you very much.

[The prepared statement of Dr. Fisher follows:]
Comment by Edwin R. Fisher, M.D. on Sen. Hatch’s Staff Analysis

Senator Hatch’s staff has submitted for comment an analysis of the study entitled “Association of Hyperestrogenemia and Coronary Heart Disease in Men in the Framingham Cohort,” published in the May 1983 issue of The American Journal of Medicine.

The analysis points up the differences between retrospective and prospective studies, indicating the inherent methodological weaknesses in retrospective studies. (It should be noted that those who claim that cigarette smoking has been established as a cause of coronary heart disease do not hesitate to cite retrospective studies without ever mentioning these weaknesses.) The analysis also emphasizes the need to follow retrospective studies with prospective ones and quite properly calls for further research to explain the reported association between hyperestrogenemia and cardiovascular disease, which is somewhat at odds with other research findings.

The analysis further indicates that a risk factor does not necessarily play a role in the causation of disease. Rather, the identification of risk factors helps the scientist formulate hypotheses to be tested by further research.

Some studies have reported smoking to be statistically associated with coronary heart disease; other studies -- both prospective and
In order to test scientifically the hypothesis that smoking, elevated cholesterol, and increased blood pressure are causally related to coronary heart disease, the federal government funded a prospective intervention study popularly called MRFIT. After 6 years of follow-up at a cost of more than $100 million, the study found no significant difference in the overall rate of cardiovascular mortality between the intervention group and the usual care group, even though there was a 2 to 1 reduction of smoking in the intervention group compared to the usual care group. Intervention studies in Norway and the U.K. also showed no statistically significant relationship between reduction in smoking and, respectively, CHD incidence and mortality. In short, all of these prospective studies are inconsistent with the hypothesis that smoking causes heart disease.

Having explained why a risk factor does not necessarily imply causation, the analysis makes the unfounded statement that "smoking must be considered a causative risk factor of heart disease." This statement ignores the studies discussed above, as well as other studies such as the Swedish Twin study which demonstrates the presence of a strong genetic or constitutional influence. To draw the unfounded conclusion that smoking has been shown to be a cause of cardiovascular disease would tend to foreclose further research in this important area.

In conclusion, scientists do not know the cause or causes of coronary heart disease. Nor do they understand which, if any, of the cardiovascular risk factors plays a role in the causation of that disease. My own experimental work and review of the scientific literature lead me to the conclusion that cigarette smoking has not been scientifically established to be a cause of cardiovascular disease.
The CHAIRMAN. Thank you, Dr. Fisher.
Let me just start left to right.
Dr. Fisher, do you smoke cigarettes?
Dr. FISHER. Yes, I do smoke.
The CHAIRMAN. Dr. Sommers?
Dr. SOMMERS. No.
The CHAIRMAN. Dr. Blau, you quite a number of years ago.
Dr. Blackwell?
Dr. BLACKWELL. No.
The CHAIRMAN. Mr. Judge?
Mr. JUDGE. Yes.
The CHAIRMAN. I figured you would as the chairman of Lorillard.

[Laughter.]
Mr. JUDGE. That is not why I smoke them. I enjoy them very much.
The CHAIRMAN. Well, I think most people who do smoke enjoy smoking.
How many packs a day do you smoke, Doctor?
Dr. FISHER. One pack a day, sir.
The CHAIRMAN. One pack a day. You have done it, I take it, most of your life?
Dr. FISHER. Since the war.
The CHAIRMAN. I see.
Mr. Judge, you cite the article in the New York Times as scientific proof to suggest that heart disease would be primarily a hormonal disorder which, if true, would affect treatment and prevention. I might just as well direct this to Dr. Fisher because he covered it—or any of the other witnesses. All of you can chat with us about this.

I have been advised that that study, however, compared 61 men with heart disease with 61 men without heart disease. Those with heart disease had had it for at least 10 years before being studied. Correct me if I am wrong, Dr. Fisher.

Thus, what the study showed in my opinion was that the people with heart disease still alive when studied had increased levels of the sex hormone. What the study did not prove is that the presence of the sex hormone was a risk factor for heart disease. Do you disagree with any of that so far?

Dr. FISHER. It is not conclusively proven. I do not believe that the study in essence is a conclusively demonstrated example of indicating a— I think we are looking for a factor which may cause heart disease, when anybody who has had much experience with the disease knows obviously it is a multifactorial situation. Multiple risk factor intervention reflects that.

I think that there are certain difficulties with that study on the hyperestrogenemia. If I recall correctly, the authors gave some reasons which were, as you suggest, Senator, not very strong reasons why they did not believe that the heart attacks caused the hyperestrogenemia. I believe that is what you are referring to.

The CHAIRMAN. For all we know, it could even mean that the increased sex hormones protect against fatal heart disease.

Dr. FISHER. That is correct. That is what I referred to as the paradox of the study, because we know that one of the reasons advanced why younger women are protected, at least relatively
speaking, to their male counterparts is because during young reproductive life they do have a relatively high estrogen content, whereas as the menopause the risk is as great for women as is men. It is believed that this is the reason. Now that is why I said it was paradoxical.

I think it is an interesting, nibbling type of study. I would not say that that pulls the curtain down in conclusively demonstrating anything.

The CHAIRMAN. We have made some comments on the New York Times article and this particular study. I will submit those to you, Mr. Judge, and would ask you to ask your scientists to analyze our comments to see if they can dispute, restate, or otherwise give us your best understanding and knowledge about it.

Mr. JUDGE. We would be delighted to do that.

The CHAIRMAN. We will give that to you.

Dr. Sommers, now you have indicated that in your testimony you state that your own study suggests that hormonal factors protect women from lung cancer. Could you briefly discuss for the committee what your study entails?

Dr. SUMMERS. Examination of autopsy cases of men and women with lung cancer, a demonstrated evidence in the endocrine glands and target organs of women that female sex hormones, estrogens, seem to be increased in their middle and later years, and that in men with squama cell carcinoma of the lung there was evidence from morphologic findings of increased testosterone more than found in age matched controls without lung cancer.

The CHAIRMAN. I see. In your testimony you state that mothers who smoke have smaller babies with lower birth weights. With that conclusion that you reached, I wonder how you can support the statement that these babies won't be deficient in any way but they will grow and develop normally.

Dr. SOMMERS. The approximately 500 grams less weight on the average of the babies of smoking mothers does not render them premature by definition of weight. In terms of their growth after birth, it is reported that they grow and put on weight faster, and after a short period of time are indistinguishable by these criteria from other infants.

The CHAIRMAN. If your wife or your daughter were pregnant and they smoked, would you advise your wife or your daughter to discontinue smoking during the pregnancy?

Dr. SOMMERS. No.

The CHAIRMAN. You would not?

If people in general ask you the question as a medical doctor, do you think that I should smoke or not smoke, what would be your response to them?

Dr. SOMMERS. I have no general advice on the subject. I would let the people make their own decision.

The CHAIRMAN. Basically what you are saying is that if a woman is pregnant it is up to her whether she smokes or whether she doesn't, even though your findings show that she has a greater propensity to have a smaller baby with a lower birth weight?

Dr. SOMMERS. No, sir. My point is that smoking mothers' smaller babies do not represent a deficiency and that shortly after birth they gain weight and fall into the normal weight limits of all
babies. In general, I am in favor of moderation in all things, including smoking.

The CHAIRMAN Dr. Fisher, in your testimony you mention again the article in the New York Times linking sex hormones to heart disease. According to which, there was no statistically significant association between smoking and heart disease. With all your professional experience, can you actually say that the data presented in that paper is relevant and statistically significant when the report was based on data collected from only 112 men in a study where it is well known that there 5,127 men and women subjects followed for three decades?

Dr. FISHER. If I may be brief and frank—

The CHAIRMAN. Sure.

Dr. FISHER. I am not overly impressed with that study. It is, I think an example that does show that at least they came to the conclusion they found no relationship to smoking. I am not using that as a positive index of my argument, so to speak.

I think the MR FIT study is the only extant study as well as the Norwegian study in which prospective randomized clinical trial technique was performed. The only way to solve a problem in a clinical setting or a clinical problem is by the prospective randomized clinical trial, so that the hundred million was, indeed, worthwhile, because, from a scientific standpoint, it was a correct approach to a problem.

The points that I make about MR FIT, however, and the thing that I found very disquieting and disconcerting in reading the testimony of last week's activity was the two points that came across to me—that we knew that smoking caused heart disease for decades. Well, that is untrue. We did not need the prospective clinically randomized trial if we already knew this. That was a falsehood.

Now the other thing that is disquieting that conclusive evidence relating smoking as a high risk factor in coronary heart disease was proven by the MR FIT study. That is totally incorrect. That is totally incorrect. You can read right from MR FIT. If I may quote one little short paragraph:

There is also the increased likelihood of over interpreting normally significant differences resulting from the examination of multiple comparisons, some of which were defined post hoc. However, the subgroup findings need exploration, especially to provide insight into the overall result and to indicate areas for further investigation.

Now that is far from being conclusive. Of course, there was a response—a large volley of letters to editors by some pretty well-recognized scientists, which indicated that in essence this is a good study. It might have been surprising to those who thought that it would turn out differently, but it tells us—at least it tells me in 1983—that again where is the evidence indicating that smoking, the intervention of the smoking factor in these people, actually influenced coronary heart disease? It didn't. It has not as yet.

Those are my points.

The CHAIRMAN. I take it as a pathologist you have examined a lot of different lungs of various people.

Dr. FISHER. Yes, sir.

The CHAIRMAN. Last week we had a very dramatic presentation by one of the leading carcinoma specialists in this country. He indi-
cated that you can definitely tell the difference between a smoker's lungs and, say, somebody who lives a normal life, say, Pittsburgh, Pa., or Shadyside.

Dr. Fisher. We have been accused of having—we have pretty bad looking lungs in Pittsburgh, Pa., but they are not functionally bad. They look bad, but they are not functionally bad.

The Chairman. Is that the same with regard to those who have been chain smokers over a long period?

Dr. Fisher. Senator, I have to disagree with that. The reknown cancer—was he a pathologist? No, he wasn't a pathologist.

The Chairman. No, he was a thoracic surgeon.

Dr. Fisher. Oh, yes. He couldn't be a pathologist. No pathologist would make that statement.

The Chairman. I see. Well, I am not sure I am quoting him right.

Dr. Fisher. Well, with the advent of the industrial revolution, Senator, it became well recognized that you could tell the lungs of a city dweller—

The Chairman. Well, he indicated that.

Dr. Fisher. From a non-city dweller. That was known by the old French pathologists in 1830 and appeared in McCollum's Textbook of Pathology, the First American Textbook of Pathology in 1900. We all know that soot, fly ash, and the industrial environment, which we practically all live in—not only humans, but, even pets in industrial environments—will show a black appearance to their lungs.

The Chairman. Well, he indicated that and did show us the carbon specs, and so forth.

Dr. Fisher. Aside from that, I know of no—none whatsoever—telltale feature which would allow me to say that was a smoker and that wasn't.

The Chairman. In other words, you would have difficulty distinguishing between two people who have lived in Pittsburgh all their lives, and one had never smoked and the other one chain smoked all his life?

Dr. Fisher. Unless somewhere along the line I must have missed something, but as far as I am concerned it is an impossibility. I would challenge him. I would like to wager with him on that.

The Chairman. All right.

Dr. Blau, if you feel that cigarette smoking is not addictive, why is it that the majority of Americans, the majority of people, have had such a difficult time quitting smoking? I agree with you that there are some who do not have that difficult time, but it seems to me the vast majority do.

Dr. Blau. My work is with people and their behavior. People in our society have a difficult time quitting a lot of things. For instance, some recent studies suggest that consistent television viewing may deteriorate schoolchildren's study habits. Therefore, frequently in cases where the child is not doing well in school, I tell the parent, "You must omit television from Sunday evening until Friday evening." For about 3 days there is a good deal of fussing. I often get calls and the parent says, "Do I have to give up the TV, also?"—very plaintively.
The difficulty in giving up pleasurable things is a great difficulty, whether it be Coca-Cola, Twinkies, hamburgers. They are things which we become acclimated to which are reinforcing——

The CHAIRMAN. Surely, you are not comparing smoking to hamburgers or jogging or affiliations with the opposite sex?

Dr. BLAU. In many ways I am.

The CHAIRMAN. You really are?

Dr. BLAU. It is a reinforcement. It is a reduction of tension, and there are many things in our society which serve the purpose of reducing stress. We are living today in what is perhaps the most stressful society that ever has existed.

The CHAIRMAN. I agree, but would you say there are really no differences between somebody who has been a chain smoker and wants to quit and someone who wants to quit eating hamburgers or wants to quit jogging, or does not want to quit jogging but for some reason has to do so.

Dr. BLAU. There are differences, Senator. Most theorists ascribe these differences to quantity. A cigarette smoker who smokes over a pack a day will be receiving over 70,000 reinforcements a year for their behavior. To withdraw the reinforcement or the pleasure or the relief is a very powerful kind of behavior. So I say it is a matter of quantity rather than quality.

It is similar to other tension-relief agents, such as television, favorite objects, routines. When people's routines are disrupted, they show very mild forms of the same kind of distress as the smoker who has to stop smoking.

The CHAIRMAN. I see.

Dr. BLAU. But there is no way you could bring an overdosed smoker or a smoker on withdrawal into an emergency room and have the staff identify this as similar to an overdose or a withdrawal on opiates, an overdoses or a withdrawal on amphetamines, or an overdose or withdrawal on alcohol. There is just no similarity.

The CHAIRMAN. Apparently, you smoked for 24 years and you quit at 35, so that would have made you 11 when you started to smoke.

Dr. BLAU. Yes, sir, I started at 11.

The CHAIRMAN. I see. When you did stop smoking after 24 years of smoking two to three packages of cigarettes a day, could you tell us the reason? Did you stop smoking out of just plain willpower or because of the hazards of smoking or the alleged hazards of smoking?

Dr. BLAU. Well, I quit the year before the Surgeon General's report so the only information we had were the childhood conundrums about these are coffin nails. In my day that is what they called cigarettes.

I quit because I suddenly decided that I wanted to take up a later-day athletic career. I became an underwater diver and a fencer in my 35th year, which was rather late, I found that smoking interfered with my competitive edge. I found that it was particularly difficult underwater diving where one is breathing compressed air, and I simply gave it up to increase my skill facility and ability in these areas.

Also, I found it was a pretty filthy habit. My fingers were brown, and my dentist kept saying, "Well, I think we have to see you.
every 3 months rather than every 6 months. My wife didn't like the way it smelled and tasted. I said, "Well, this is the time when these things have come together. I'm going to dump it."

The CHAIRMAN. However, in your case you did not have any real difficulty dumping it, then?

Dr. BLAU. No, other than that—

The CHAIRMAN. But you had plenty of motivation to do it.

Dr. BLAU. The greatest difficulty was my fingers. I needed something to hold in my hand. Smoking three packs of cigarettes, about 60 times a day you have something in your hand. It is always a wonderful thing such as it a tense point in psychotherapy where the patient asks something and I do not know the answer, instead of saying, "Hmmmmm, I would reach for a cigarette and put it on the desk, you know.

The CHAIRMAN. It gives you a little time to pause.

Dr. BLAU. I missed it as a part of the everyday paraphernalia of pacing oneself's.

The CHAIRMAN. But you don't think that has anything to do with the so-called addiction to cigarettes that some of the doctors last week, or all of them basically, testified to?

Dr. BLAU. I understand that many of my colleagues are quite enamored of the concept of cigarettes as being addictive, but I think that they have taken a concept with very severe and important manifestations and diluted it in order to fit a desired outcome. They want people to stop smoking. I think that is fine, but to dilute the concept of addiction because that might be a good way to stop people from smoking I think begins to smack of deterioration of respectable science—and may have very serious long-range consequences.

The CHAIRMAN. Dr. Blackwell, in your testimony you have said that the decision to smoke is not based on advertising. Could you just elaborate a little bit more on that?

Dr. BLACKWELL. The decision to smoke is a complex sociopsychological process. I have not conducted studies to determine that myself, but the ones that I have seen indicate that it is peer pressure, social pressure, that primarily causes people to be interested in that.

I can testify from my own experience and as the parent of a teenager I have observed that. I also noticed in the testimony of Dr. Lipsett, when he said that a number of studies have indicated that the presence of a best friend who smokes is the single most important predictor of smoking in children from the 5 through the 12 grades, he went on to say that cigarette smoking is perceived by youngsters in the early school years to be bad for health. They know it can cause cancer and has been associated with lung and heart disease. They concede it is unhealthy and habit forming; but neither knowledge nor fear is adequate to counteract the pressures that surround children as they reach puberty and enter adolescence.

From the other studies that I have seen and observed, I believe that it is the social situation—and I am active in a group that works with high school and young people with smoking issues, and the educational materials, and so forth, that we have developed
there stress the fact that primarily social issues and pressures cause people to start smoking.

As a behavioral scientist, you usually come to the conclusion of adage that the reason you start would probably be rather important in why you stop, also. It is social pressures as well as personality and psychological reasons.

The CHAIRMAN. Dr. Fisher, do you recognize any disadvantages healthwise to a person who smokes? Are you saying there are no disadvantages that you know clinically or otherwise, through research or otherwise, to smoking, from a health standpoint. We all know that they cost money. We all know that there may be other practical disadvantages, but you can talk about those, if you wish, also.

Dr. FISHER. I think that, as I address myself to coronary heart disease—and to reemphasize, I find no firm scientific evidence—it does not even have to be that firm; there just isn't any—

The CHAIRMAN. I understand your testimony there, but do you find any other areas in the health of human beings where smoking is deleterious?

Dr. FISHER. Not that I can recognize.

The CHAIRMAN. Not any? Not cancer? You do not believe that smoking causes cancer? 

Dr. FISHER. Again, not demonstrated.

The CHAIRMAN. You do not believe that it causes respiratory diseases or illness?

Dr. FISHER. I have not investigated the more common types of chronic obstructive lung disease in that regard. Whether it does or not, I cannot state.

The CHAIRMAN. Do you believe smoking has a deleterious effect on emphysema or the cause of emphysema or even a preexisting emphysema condition?

Dr. FISHER. It may have on an individual who has emphysema. That is obvious. Any agent inspired in an individual with preexisting emphysema is going to cause difficulty, but whether it actually can be stated to be the cause of emphysema, I am not willing, as yet, to accept.

The CHAIRMAN. But aren't you willing to at least consider that it might be a cause of emphysema?

Dr. FISHER. I am not ashamed to say that we do not know. You see, we have a lot of statements being made. Last week, in particular, as I read the testimony and as one makes little notes, there is nothing to be ashamed of in saying you don't know. It seems that people are willing to accept an answer even if it is wrong. They will be willing to accept that, but they will not accept no answer.

The CHAIRMAN. Are you saying that all these health professionals—the National Institutes of Health, the National Cancer Institute, and others—are wrong when they say that they do know?

Dr. FISHER. No. Well, I think that the evidence that they use to support their argument is highly questionable and, indeed, very spurious. Indeed, let's take heart disease again. I want to emphasize it doesn't take much to recognize that, why did we spend $115 million on a clinical trial if we already had this evidence? That would be like frosting on the cake. We didn't know. We have not known. We still do not know.
In 1982, the results came out that you can interpret as indicating, contrary to expectations, that smoking does not represent an important risk factor in heart attack.

The CHAIRMAN. Dr. Fisher, there have been, as I understand it, and heard testified to before our committee, over 30,000 studies that relate smoking to disease, and you are saying that none of these studies is definitive or conclusive with regard to the relationship between smoking and disease?

Dr. FISHER. Thirty thousand studies?

The CHAIRMAN. Thirty thousand studies was the figure that was mentioned.

Dr. FISHER. One could spend a lifetime reading those studies. My only experience to the 30,000 figure is I know a book at home—it is a red book, and I think it was put out by the Government on smoking and health. You read the abstracts. There are not 30,000. There may be several thousand. You could split them right down the middle—those pro and those con.

The point that I am trying to make, Senator, is that as far as I am concerned MR FIT was an excellent study. It was worth the money. It showed me something that I can say, "This is a good approach. This had scientific validity to the approach."

The CHAIRMAN. You also have to say that the MR FIT study involved subjects who were put on nutritional value—

Dr. FISHER. That is correct.

The CHAIRMAN. They were put on an exercise program.

Dr. FISHER. That is correct.

The CHAIRMAN. They were not the normal, average human being who does not always eat nutritionally, does not always perform to the right exercise quotient, does not live certain respectable and good health laws. Mr. FIT was really not the study of the average American human being.

Dr. FISHER. Well, but the design of the study—you see, the advantage of the study was taking people at risk and then randomizing them. The reason for randomizing them, of course, is that you have then—by randomizing, prospectively randomizing them, then you are, in essence, controlling certain other unknown variables which may participate in causing an effect. That is the purpose of randomization.

Scientifically, it was done well. The results were a little unexpected, but that is the way results are. Never be surprised at results.

The CHAIRMAN. You are familiar with the Framingham study?

Dr. FISHER. That is correct.

The CHAIRMAN. As I understand it, it had 5,127 men and women who were followed through three decades, and that was one of the many studies which has conclusively shown that cigarette smoking, high blood pressure, and elevated blood cholesterol are powerful and independent predictors of heart disease risk. Do you disagree with that statement?

Dr. FISHER. Oh, yes. I do not think that is conclusive whatsoever.

The CHAIRMAN. OK. Those who wrote it feel that it is conclusive.

Dr. FISHER. That is why they needed to do a MR FIT. We must not have been too satisfied with it. The same people who were—
apparently, they modeled the MR FIT after, to some degree, the Framingham study.

The CHAIRMAN. MR FIT involved 112 people who were put on an extraordinarily good diet, who did extraordinary exercise, and who really lived superior health laws, whereas this other was a study of 4127 basically normal people. They concluded that cigarette smoking results in high blood pressure, elevated blood cholesterol, and that cigarette smoking is a powerful and independent predictor of heart disease risk. If you pay attention to the risk factors, you are going to be able to predict heart disease probabilities more in smokers than you can in others.

You don’t agree?

Dr. Fisher. I do not agree. Let's just look at MR FIT here at page 1472 in the discussion of the findings and showing the difficulty—how difficult it is to define subgroups and to collect your cohorts of people. It says here, "Nonsmokers have on the average higher blood cholesterol and blood pressure levels than smokers." That has been known, too. In other words, nonsmokers have higher blood cholesterol and blood pressure levels than smokers to start with.

I do not see anything wrong in—there are large numbers of people here in this study, the MR FIT study. I think it was money well spent. I think it shows the right approach. Instead of relying on that 30,000 anecdotes that you refer to, Senator Hatch—anecdotes, stories—I could read you a story that Mark Twain said about smoking: I would not say it is very scientific. It is humorous. It is philosophical, but those are anecdotes that refer to certain studies dealing with carbon monoxide—5 patients, 10 patients. In the hyperestrogenemia study there were only 15 patients here against 11 patients here. That is anecdotal. This is problem solving, clinical problem solving done by a prospective randomized clinical trial. It is the only scientific method.

We have not had that until MR FIT. Now MR FIT produces some results that people do not like. Well, that's too bad. It is like looking at a picture. how do you interpret results? Beauty is in the eye of the beholder. Some like them, some don't like them. However, when you get down to the bottom line, they do not tell us anything about cigarette smoking.

They have extra bonuses. It shows us that maybe the way we are treating high blood pressure is not so good. That is an extra bonus. I think it is wonderful to do a study like that.

The CHAIRMAN. I have no problems with the MR FIT study. I am just saying it can be distinguished from other studies that conclude differently from the way you have here today.

Be that as it may; let the ask just one other question.

Dr. Fisher. Yes.

The CHAIRMAN. Do you know of any difficulties to women who are chain smokers who are pregnant? Do you know of any difficulties prenatally to the infant or postnatally, or to the woman herself?

Dr. Fisher. No. I have not investigated that particular matter, sir. I know that again, I could be anecdotal about it, but I will not because it is unscientific.

The CHAIRMAN. All right.
Dr. Sommers, do you know of any—do you agree there are no studies or there are no conclusive conclusions that determine that cigarette smoking is a disease causer in any human beings and under any circumstances? Do you agree basically with what Dr. Fisher has said about this?

Dr. Sommers. I basically agree with Dr. Fisher with regard to disease causation. Smokers show functional differences from non-smokers, just as they show weight and blood pressure differences. Some of these may be constitutional.

The Chairman. However, you would not say that smoking probably causes cancer?

Dr. Sommers. I do not believe that it has been proved scientifically that cigarette smoking causes lung cancer.

The Chairman. I see. Do you feel that cigarette smoking is an aggravating aspect to a preexisting emphysema condition or do you believe that it can cause emphysema?

Dr. Sommers. As Dr. Fisher stated, a person who already has emphysema does suffer problems with any inhalants, whether it be diesel fumes or smoke of any source. On the other hand, I do not believe that it has been demonstrated that cigarette smoking causes emphysema.

The Chairman. OK. I have taken too much time. We will turn to Senator East.

Senator East. Thank you, Mr. Chairman.

I would like to yield, if I might, to Senator Dodd, who has a markup to attend. I would be happy to yield to him.

Senator Dodd. I thank the Senator. I will be very, very brief. I really appreciate your testimony. I have looked over the testimony of witnesses from last week, and a lot of this gets very arcane.

Let me ask you a layman's question. The chairman obviously is getting into the sophisticated medical issues and knows a lot about them. Let me ask you what I think all of us want to know, particularly, those of you from the scientific field. Mr. Blackwell, you are in marketing, so I will spare you the question.

Do you believe that smoking is injurious to one's health? Yes or no?

Dr. Fisher. I think the way I would state it, smoking may be injurious to one's health.

Dr. Sommers. Smoking might be injurious to one's health.

Dr. Blau. I don't know.

Mr. Judge. I think it is definitely an open question.

Senator Dodd. I guess that is why they call it a “practice.”

My own feeling is I think it does. I do not have very many questions about it.

I smoke, and it is hard to quit. I have quit before. I lay off them for a while, but it is not easy. Others may have an easy time, but I don't.

I feel better when I do not smoke. Again, I do not have scientific data, but I can say how I feel on these things.

I was intrigued about the marketing question. The issue is a bit confusing to me. I would like to spend some time going over that myself, quite honestly, Mr. Chairman. The marketing, the aware-
ness questions are the ones that intrigue me the most because that
is, in effect, part of the bill that matters.

Are we increasing awareness of people who do smoke or who do
not smoke as to what I believe is the case, that it is generally injuri-
sous to your health. Whether or not it causes or exacerbates a par-
ticular illness or another is something medical people can argue
about, and I will try to become more knowledgeable about that.
However, the general proposition of whether or not people under-
stand smoking is bad for their health is the issue that I find in-
triguing.

The FTC study, of course, we have. There is a Roper study done
in 1980 and a Chilten study as well. Then I do not know which wit-
ness talked about the Gallup poll; is that correct?

Dr. BLACKWELL. Yes.

Senator DODD. What other organizations have done those sur-
veys? Who do they do them for? Who did the Roper study? Who
paid for that study?

Dr. BLACKWELL. Those studies are studies cited in the FTC. They
were mostly done at Government expense for the purposes of the
FTC investigation. I can supply those specific references for you, if
you would like me to do so.

Senator DODD. Do you have them with you today?

Dr. BLACKWELL. I do not have them with me, but I can provide
them.

Senator DODD. Mr. Chairman, I would like to see those studies
and the surveys. I would like to know who, in fact, did pay for
them, not that that necessarily would persuade me one way or an-
other as to their legitimacy. But I happen to agree with you that I
think question writing is the key. Of all of us up here are particu-
larly sensitive to polls.

[The information referred to follows:]
May 25, 1983

The Honorable Christopher J. Dodd
402 Russell Senate Office Building
Washington, D.C. 20510

Dear Senator Dodd:

This letter is in response to your request at the May 12 hearing on S. 772 that I provide you with the studies relied upon by the FTC Staff in their report on public awareness of smoking and health claims. Although the Staff Report cites to a variety of surveys and opinion polls (see FTC Staff Report pp 3-1 through 3-3), the two that the staff chiefly relied upon are the 1980 Roper Study and the 1980 Chilton Study, both of which were commissioned and paid for by the FTC Staff (See FTC Staff Report pp 3-2 and 3-3). I am enclosing copies of these studies for your review. I also am enclosing copies of two other studies also paid for by the FTC Staff -- the 1980 Burke Research Focus Group Study and the 1980 Walker Research Study -- that I discussed in my written statement to your Committee (at pp. 12-13).

As described in my testimony (at pp. 4-9) all of these surveys suffer from certain methodological deficiencies and definitional problems that, in my opinion, result in an understatement of the actual levels of awareness about the claimed health effects of smoking -- an underestimation of the survey results. But as I also mentioned in my testimony (at pp. 9-10), even these FTC-sponsored studies demonstrate an extremely high level of awareness of specific claims about smoking and health. Thus, taking into account the underestimation errors, I believe that a proper reading of the figures cited in the studies reflect virtually universal public awareness not only of the general claim that smoking is hazardous to health but of the specific health hazards attributed to smoking by S. 772.

At the May 12 hearing, you also asked whether there are other recent studies not commissioned by the FTC Staff concerning public awareness of smoking and health claims. The major polling organizations such as Gallup and Roper periodically conduct surveys on this issue, and as an example, I am enclosing a summary of the Gallup Poll for August 31, 1981. This poll confirms the findings of the FTC-sponsored studies that the vast majority of consumers believe (and thus even more are aware of) both the general and specific claims about smoking and health.

College of Administrative Science
I hope this material is responsive to your request. I would be pleased to provide any further information or comments that you feel would be helpful in your deliberations on the bill.

I also would appreciate your seeing to it that this response is included in the hearing record, so that the record will reflect that I have complied with my undertaking to supply this material to you.

Very truly yours,

Roger O. Blackwell
Professor of Marketing

cc: The Honorable Orrin G. Hatch

College of Administrative Science
SMOKING LEVEL DECLINES AS
MORE PERCEIVE HEALTH HAZARD

by George Gallup

PRINCETON, N. J. -- Cigarette smoking in the U.S. has
declined during the last decade, apparently because growing
numbers of Americans have become convinced there is a causal
relationship between smoking and such diseases as lung
cancer, throat cancer, heart disease and birth defects.

As reported yesterday, the percentage of smokers is
the lowest recorded in 37 years of regular Gallup Poll
audits. In the current survey, 35 percent say they have
smoked within the past week, reflecting a continuing
downward since 1972 when 41 percent of adults were smokers.

In addition, the proportion of heavy smokers (two packs a
day or more) has decreased over the last four years.

As the smoking level has trended downward -- eight
points nationally since 1972 -- an upswing has been recorded
in the proportion of Americans who see a link between
smoking, lung cancer and other health problems.

The proportion who believe smoking to be a cause of
heart disease has increased from 62 percent in 1969 to 68 percent in 1977 and to 74 percent in the current survey. And the percent who perceive smoking as a cause of lung cancer have grown from 71 percent to 83 percent since 1971.

Additionally, the percentage who believe smoking is a cause of birth defects has increased from 41 percent in 1977 to 83 percent today, and the proportion saying smoking is a cause of throat cancer was 79 percent four years ago and is 81 percent today.

Following are the questions on attitudes toward smoking and the results with a comparison of the views of smokers and non-smokers:

"Do you think that cigarette smoking is or is not harmful to your health? Is or is not one of the causes of lung cancer? heart disease? cancer of the throat? birth defects?"

<table>
<thead>
<tr>
<th>CIGARETTE SMOKING HARMFUL TO HEALTH?</th>
<th>Yes</th>
<th>No</th>
<th>No opinion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NATIONAL</strong></td>
<td>90%</td>
<td>7%</td>
<td>3%</td>
</tr>
<tr>
<td>Smokers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-smokers</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

420
<table>
<thead>
<tr>
<th>CAUSE OF LUNG CANCER?</th>
<th>Yes</th>
<th>No</th>
<th>No opinion</th>
</tr>
</thead>
<tbody>
<tr>
<td>NATIONAL</td>
<td>62</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>Smokers</td>
<td>69</td>
<td>21</td>
<td>10</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>93</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CAUSE OF THROAT CANCER?</th>
<th>Yes</th>
<th>No</th>
<th>No opinion</th>
</tr>
</thead>
<tbody>
<tr>
<td>NATIONAL</td>
<td>81</td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td>Smokers</td>
<td>69</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>87</td>
<td>5</td>
<td>8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CAUSE OF HEART DISEASE?</th>
<th>Yes</th>
<th>No</th>
<th>No opinion</th>
</tr>
</thead>
<tbody>
<tr>
<td>NATIONAL</td>
<td>74</td>
<td>14</td>
<td>12</td>
</tr>
<tr>
<td>Smokers</td>
<td>59</td>
<td>25</td>
<td>16</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>62</td>
<td>9</td>
<td>9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CAUSE OF BIRTH DEFECTS?</th>
<th>Yes</th>
<th>No</th>
<th>No opinion</th>
</tr>
</thead>
<tbody>
<tr>
<td>NATIONAL</td>
<td>53</td>
<td>23</td>
<td>28</td>
</tr>
<tr>
<td>Smokers</td>
<td>30</td>
<td>20</td>
<td>27</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>68</td>
<td>10</td>
<td>23</td>
</tr>
</tbody>
</table>

Growing numbers favor harder line on smoking
GALLUP POLL 4/30/41

Over the last four years the proportion who favor stricter measures to discourage cigarette smoking has increased:

- 41 percent think federal and state taxes on cigarettes should be increased, compared to 36 percent who held this view in 1977.
- 43 percent think there should be a complete ban on cigarette advertising compared to 36 percent four years ago.

At the same time, however, similar majorities in both the 1977 and 1981 surveys oppose a complete ban on the sale of cigarettes.

Here are the questions and results for both smokers and non-smokers and for the nation as a whole:

"Do you think federal and state taxes on cigarettes should or should not be increased?"

INCREASE TAXES ON CIGARETTES?

<table>
<thead>
<tr>
<th></th>
<th>Should increase</th>
<th>Should not</th>
<th>No opinion</th>
</tr>
</thead>
<tbody>
<tr>
<td>NATIONAL</td>
<td>46%</td>
<td>46%</td>
<td>8%</td>
</tr>
<tr>
<td>Smokers</td>
<td>23%</td>
<td>73%</td>
<td>4%</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>59%</td>
<td>12%</td>
<td>9%</td>
</tr>
</tbody>
</table>

"Do you think there should or should not be a complete
BAN CIGARETTE ADVERTISING?

<table>
<thead>
<tr>
<th></th>
<th>Should ban</th>
<th>Should not</th>
<th>No opinion</th>
</tr>
</thead>
<tbody>
<tr>
<td>NATIONAL</td>
<td>43 pct.</td>
<td>31 pct.</td>
<td>6 pct.</td>
</tr>
<tr>
<td>Smokers</td>
<td>27</td>
<td>68</td>
<td>5</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>57</td>
<td>41</td>
<td>6</td>
</tr>
</tbody>
</table>

"Do you think the sale of cigarettes should or should not be banned completely?"

BAN SALE OF CIGARETTES?

<table>
<thead>
<tr>
<th></th>
<th>Should ban</th>
<th>Should not</th>
<th>No opinion</th>
</tr>
</thead>
<tbody>
<tr>
<td>NATIONAL</td>
<td>20 pct.</td>
<td>74 pct.</td>
<td>6 pct.</td>
</tr>
<tr>
<td>Smokers</td>
<td>10</td>
<td>89</td>
<td>1</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>26</td>
<td>66</td>
<td>3</td>
</tr>
</tbody>
</table>

The results reported today are based on in-person interviews with 1,325 adults, 18 and older, contacted in more than 300 scientifically-selected localities across the nation during the period June 26-29.

For results based on a sample of this size, one can say with 95 percent confidence that the error attributable to sampling and other random effects could be as large as 5 percentage points in either direction.
61 PERCENT OF CANADIANS ARE SMOKERS

A higher proportion of Canadians (45 percent) than Americans (33 percent) can be classified as smokers—that is, have smoked cigarettes in the past week—according to the Gallup-affiliated Canadian Institute of Public Opinion.

A long-term downward in the rate of smoking is noted in Canada as in the U.S. In 1973, when the audit was started in Canada, 52 percent of Canadians reported that they smoked in the test week.

A total of 54 percent of Canadians think all advertising for cigarettes should be banned. The comparable figure among Americans, as reported today, is 83 percent.

A solid majority of Canadians today (58 percent) also favor separate areas in restaurants for those who smoke, a higher figure than favored four years ago, 49 percent.

(COPYRIGHT 1981 FIELD ENTERPRISES, INC.
FIELD NEWSPAPER SYNDICATE 8/12/81)
The Chairman. Who did pay for it?
Dr. Blackwell. Those were FTC-sponsored studies.
The Chairman. We will be happy to provide those.
Dr. Blackwell. I will get those specific references for you, Senator.
Senator Dodd. That, to me, is a pivotal question on the multiple labeling question, as to whether or not—how necessary—
The Chairman. Would the Senator yield again?
Do you mean the FTC paid for them?
Dr. Blackwell. Yes.
The Chairman. They actually ordered them through you and paid for them?
Dr. Blackwell. Say it again, sir.
The Chairman. They actually ordered these studies and paid for them?
Dr. Blackwell. Yes.
Senator Dodd. Have you looked at other ones that have not been ordered by the FTC? Are there a lot of surveys done on smoking and people's awareness?
Dr. Blackwell. There are studies done which look at who smokes, why, by market segments. Some of those are done for advertising purposes, agencies and tobacco companies, and some done by academics, cases that they write. These were the only studies that I know of that specifically investigated the issues as they relate to these warnings and the information that might be given to consumers for the purposes such as this bill.
Senator Dodd. If there are any other ones, I would like to get them. I do not want to be buried in them, but to the extent there are some recently done—any earlier than 1980, I would not worry about—I would be interested.

Mr. Chairman, you have asked a lot of health questions, and I have listened attentively to what you are saying. It is my understanding we are going to have a markup on this tomorrow?
The Chairman. We have one scheduled for tomorrow.
Senator Dodd. Are we going to have a report by tomorrow?
The Chairman. No. The report will not be done, assuming we can get this marked up tomorrow—but, I think a lot depends on whether or not we are in tomorrow.
Senator Dodd. I agree. I would like to see these things.
The Chairman. Let me ask you this, Senator: Are you saying that you need more time?
Senator Dodd. I would like to look at these reports because this is a new issue for me. The findings issue particularly. I would like to look into it.
The Chairman. It is a complex issue. We had a witness testify last week who rebutted a lot of what Dr. Blackwell has said. However, Dr. Blackwell has an excellent reputation. There are some contradictory findings. Every witness last week contradicted Dr. Fisher and Dr. Sommers, and they are the most eminent people in the field—against smoking and find smoking is deleterious to the human being.
Senator Dodd. My inclination substantively is I sort of agree with the bill. That is substantively where I come from on the thing, but I am curious about this—
The CHAIRMAN. Let me ask you this: Is the Senator saying that you feel you need to examine all of these materials—

Senator Dodd. I would like to see those—

The CHAIRMAN [continuing]. Before we have a markup?

Senator Dodd. I would like to see the surveys and who paid for them, and I would like to look at that awareness question. I am saying if the markup is tomorrow, it is a little fast without a report.

The CHAIRMAN. Well, it is fast. We do have the report of last year's bill, but this record made this year is additional to last year. If the Senator feels that we need more time, I would certainly take that under consideration.

Senator Dodd. Would you? I would appreciate it.

The CHAIRMAN. Yes. Let me chat with staff and try to get back to you today and make a determination as to whether we should give some more time. However, we do have an extensive report from last year which goes into every one of these questions. On the other hand, that was last year.

Senator Dodd is a new member of our committee, and, of course, he has a right to see these materials. He cannot be expected to know what all of us went through last year on this.

Senator Dodd. I appreciate that, Mr. Chairman, very much. I found this very interesting. I was particularly intrigued with the questions you had, Mr. Chairman, on the health issues as well. I thank you.

The CHAIRMAN. Thank you.

Senator East?

Senator East. Thank you, Mr. Chairman.

Senator Dodd. Thank you, Senator East, for yielding.

The CHAIRMAN. Thank you, Senator Dodd, for being here.

Senator East?

Senator East. Mr. Chairman, I would like to make a unanimous consent request with regard to some materials that I would like entered into the record.

First of all, I have written eight individuals, medical doctors, regarding their comments on this bill. To date, I have received statements from three of the eight. I would like, please, to request that their responses be made a part of the record.

In addition, I would like, please, to request that the record be left open so that five additional individuals' statements from medical doctors and scientists might also be made a part of this record. That is the first part of my unanimous consent request.

Second, if it has not already been included, I would like to ask unanimous consent that the Castelli study that has been referred to here today, and also previously, be made a permanent part of the record.

The CHAIRMAN. Without objection.

Senator East. Thank you, Mr. Chairman. I appreciate your consideration.

[The statements and study referred to follow:]
Dear Senator East,

I have the honour to enclose a critique of S.B. 5772, before the State and House of Representatives of the United States of America. Some supporting reprints, which present my arguments in greater detail, are also enclosed.

Yours sincerely,

P.R.J. Burch
Professor

Encs.
In this memorandum I criticize the statements made in the Bill, S. 772, about the effects of cigarette smoking on overall mortality, lung cancer and heart disease. These are issues that I have studied personally in some detail.

Under FINDINGS, SEC 2, we are told that the Congress finds various hazards associated with smoking. The manner in which these "findings" were made is not described but I presume that the reports of the Surgeon General of the United States have served as a basis for the allegations.

Mortality from All Causes

"Finding (1)", at the top of page 2 of the Bill, is ambiguously worded: "cigarette smoking is the largest preventable cause of illness and premature death in the United States and is associated with the unnecessary deaths of over three hundred thousand Americans annually." (Emphasis added.) I infer that "associated with" is meant to be understood as "causes" although, in scientific parlance, the two terms are far from synonymous. Logicians, statisticians and competent scientists are familiar with the aphorism: "association does not necessarily imply causation." The general tenor of the Bill leaves little doubt, however, that we are intended to conclude that cigarette smoking causes at least 300,000 unnecessary deaths in the United States. Nevertheless, the use of the expression "is associated with" in the Bill is more than pedantic interest because the frequent identification in epidemiology of association with causation is a source of many misunderstandings and fallacies. When analysing epidemiological findings it is of the utmost importance that the distinction between association and causation should always be borne clearly in mind. Those who appreciate the distinction will be careful to observe it in their written statements. In discussing "association", Sir Austin Bradford Hill wrote in his "Principles of Medical Statistics": "Merely to presume that the relationship is one of cause and effect is fatally easy; to secure satisfactory proof or disproof, if it be possible at all, is often a task of very great complexity." The degree of complexity is commonly.
if not invariably, underestimated in the Surgeon General's reports.

The figure of "over 300,000" appears to be based on: (a) the Surgeon General's (1979) conclusion: "The overall mortality ratio for all smokers of cigarettes is about 1.7 compared to nonsmokers"; and (b) the assumption that this association between cigarette smoking and overall mortality is causal in origin. Given this kind of association - and the others implicit or explicit in the Bill's (2) to (4) of SEC.2 - we have to all instances to consider the following possibilities:

(i) Smoking causes the morbidity or fatal disorders. This is the straightforward causal hypothesis.

(ii) The associated diseases, or one or more connected pre-disease conditions, cause the smoking. (I call this the "reverse causal" hypothesis.)

(iii) A third factor causes, or predisposes to, both the smoking and the associated diseases. This is sometimes described as the "common cause" hypothesis and it is often discussed in genetic terms: the person's genotype predisposes both to the smoking and to the diseases and the association arises at the genetic level. In this latter form, the common cause hypothesis is often referred to as the "constitutional" hypothesis.

(iv) Because (i), (ii) and (iii) are not mutually exclusive, any combination of them may be needed to explain the overall association, whether it be negative or positive.

Our ultimate scientific objective should be the estimation of the separate contributions (i) to (iii), together with their respective confidence limits. The Surgeon General's reports do not acknowledge, and appear to be unaware of, these obligations.

I have studied the relation between smoking and overall mortality in England and Wales and a reprint of my paper on the subject is enclosed (Smoking and Mortality in England and Wales, 1950 to 1976, Journal of Chronic Diseases).
The paper contains an analysis of the temporal trends in sex- and age-specific mortality (all causes) in relation to the corresponding trends in cigarette consumption. An important cause of the consistent decline in mortality, which accounts, in effect, for virtually all of the fall over the age range 20-44yr in both sexes, is shown to be the near-eradication of tuberculosis. There is no indication that the temporal changes in cigarette smoking have modified this fall.

My analysis of temporal (secular) trends is one of the very few approaches to the problem that addresses itself to the causal/constitutional issue. Another is the randomized controlled intervention trial in which smokers are allocated randomly to either of two groups: (a) the "intervention group", which is subjected to intensive pressure to give up smoking; and (b) the "usual care", or "normal care" group, which is not subjected to special pressures. Randomization is a device that eliminates the bias of self-selection.

One such trial has been carried out in England by Professor Rose and Dr. Hamilton and the results of another, the Multiple Risk Factor Intervention Trial (MRFIT) carried out in the United States, were reported in the Journal of the American Medical Association on September 24, 1982. Neither trial has given any indication that quitting smoking reduces the risk of death but numbers have been too small to provide a definitive test.

In the London study, 128 deaths were recorded after 10 years of follow-up among the 731 men in the "normal care" group and 123 deaths were recorded among the 714 men in the "intervention" group. In the United States, MRFIT study, 260 deaths were reported among the 6,438 men in the "usual care" group, while 265 deaths were reported among the 6,428 men in the "special intervention" group, which was also given dietary advice and stepped-care treatment for hypertension. Combining the results from the two studies, we see that the "normal-usual-care" groups suffered a 5.41% mortality, while the "intervention" groups, at a substantially lower level of smoking, suffered a mortality of 5.43%. These mortalities are virtually identical, but the difference would need to be as much as 14% to be
The final paragraph of my own Conclusions, written before the publication of the results of the HR1T Study, reads as follows: "This paper has shown that secular trends in overall mortality in England and Wales give no consistent indication that they were appreciably influenced by changes in cigarette consumption. The two other types of critical evidence reviewed here - the preliminary results of a randomized controlled intervention trial and findings from MZ and DZ twins discordant for smoking - both suggest that the observed mortality ratios are more likely to result from a constitutional than a causal association. But so far, both studies suffer from small numbers and definitive conclusions on this issue cannot yet be drawn from them. On scientific grounds there can be little doubt that the conclusions drawn by the Royal College of Physicians and the Surgeon General of the United States about the lethality of smoking are precipitate and unwarranted."

In the light of all the critical evidence that is free from the bias of self-selection, I am unable to find any scientific justification for the assertion in the Bill that cigarette smoking causes in the United States over 300,000 unnecessary deaths annually.

Heart Disease

Following mortality from all causes it is convenient to consider heart disease because, as the Bill states, it accounts for nearly one half of the deaths in the United States. The Bill adds, with characteristic ambiguity: "one third of the deaths attributable to heart disease are associated with smoking." I strongly suspect that "are associated with" is intended to be understood as "are caused by".

My recent paper published in this field is: Ischaemic Heart Disease: Epidemiology, Risk Factors and Cause (Cardiovascular Research, 14, 307-338, 1980), a reprint of which is enclosed. Among the various so-called "risk factors" discussed in the paper is cigarette smoking. I analyse the sex- and age-specific mortality from ischaemic heart disease in six populations, including U.S. Whites and U.S. Non-Whites, and give special attention to the intriguing fall in mortality from the disease in the United States over the
Period, 1968 to 1975. The data from all populations have certain special features, including a particular relation between the sexes that facilitate analysis. These features allow conclusions to be drawn that do not depend too critically on errors of death-certification. J.C. Kleinman, J.J. Feldman and K.A. Hook had previously observed (American Journal of Public Health, 69: 195-802, 1979): "Smoking changes among women were not generally consistent with declines in CHD mortality." My analysis agrees with theirs and it indicates that any causal influence of smoking on the fatal disease Process is either small or non-existent. The factors responsible for the fall in mortality in the United States in both sexes are unconnected with smoking over the age range 40 to 64yr they make an effectively equal impact on white males and females and are therefore ubiquitous. They are likely to be either microorganisms, or viruses, or allergens.

The randomized controlled intervention trials in England and the United States have failed to demonstrate any significant differences in mortality from coronary heart disease in the "intervention" and "usual care" groups but numbers are, of course, markedly fewer than for "all deaths" and they do not provide a definitive test. Intervention with dietary habits and the treatment of hypertension further complicate the WHFIT study. My own studies are based on the statistics for both sexes in the national populations of England and Wales, and the United States and they enable me to reject confidently the claim implied in the Bill that one third of the deaths attributable to heart disease are caused by smoking.

Lung Cancer

Partly because of past and present errors of clinical diagnosis and death certification it is impossible to derive precise quantitative conclusions about the role of cigarette smoking in the pathogenesis of lung cancer. I have published three major papers on this subject, including an invited one presented to the Royal Statistical Society in London on May 17, 1978 (reprints enclosed).

In addition, a substantial portion of Chapter 10 of my book, The Biology of Cancer,
A new approach (University Park Press, Baltimore, 1976), is devoted to the relation between smoking and lung cancer.

Recognizing that simple associations and evidence for lung cancer in voluntary ex-smokers do not resolve the causal/constitutional issue, and bearing in mind the Royal College of Physicians (1971) stricture: "The chief reason for rejecting the genetic hypothesis is its inability to account for the enormous rise in death rates from lung cancer in the past half century," I have given special attention to the sex- and age-specific death rates recorded for lung cancer in England and Wales. (Thanks to the work of the late Professor A.P. Passey in England and that of Dr. K. Herrold in the United States it is now apparent that the converse causal hypothesis — for example, a pre-cancerous inflammatory condition of the lungs and bronchi caused the smoking — has little or no relevance to this context.)

Careful study of the recorded increases in mortality from lung cancer in England and Wales shows that, although the rises have been generally larger in men than in women, the fine structure of the changes has been extraordinarily synchronous in the two sexes. It follows that the main causes of the increases have also been synchronous in men and women. Smoking cannot have been a main cause because the sharp rise in cigarette smoking by women occurred some 30 years after that by men. More detailed calculations, including those of the sex-ratio of the recorded changes in mortality described in my Royal Statistical Society paper, confirm that smoking cannot be implicated as the main cause of the recorded rises in either sex. Indeed, it is readily shown that those rises are many times greater than those predicted from the causal hypothesis and the data for tobacco consumption. It is now widely agreed that changes in diagnostic practice have been responsible for a substantial part of the recorded increases. However, because they are so large they could readily conceal a genuine component attributable to smoking.

In an attempt to explore this latter possibility and to minimize the complications that arise from errors of death certification I have studied the
details of recorded sex- and age-specific mortality from lung cancer in relation to those of cigarette consumption for the relatively recent period, 1950 to 1975 (Journal of Chronic Dis. 33, 221-233, 1980) and the still more recent period, 1974-1978 (Cancer Epidem. 1, 1651-1657, 1980). To test causal hypotheses methodically we have to make specific assumptions about mechanisms; none are based on evidence independent of that being examined in the papers cited in this paragraph. The precipitator (causal) hypothesis emerges as being consistent with a wide range of evidence that refutes initiator and promoter hypotheses. Unfortunately, the precipitator hypothesis is not supported by the analysis of post-1950 trends. However, recent direct post-mortem studies of the accuracy of certification in England (West Midlands) and Scotland (Edinburgh) show that substantial errors in the clinical diagnosis of lung cancer persist. Probably many of the anomalies in the epidemiologic data should be attributed to them. In the abstract of my Journal of Chronic Disease paper I concluded: "In view of the dubious reliability of the potentially critical data analysed here and elsewhere, it is questionable whether any firm conclusions can as yet be drawn about 'dose-response' relations for cigarette smoking and lung cancer."

The criterion of "repeatability" is usually applied to a causal relationship. In epidemiology it is impossible to replicate observations as in an experimental science because of difficulties, for example, in characterizing and standardizing for the relevant parameters of smoking, drinking, etc. It is seldom possible to allow adequately for errors of diagnosis. Nevertheless, in commenting on the 1964 report of the Surgeon General, Professor Mervyn Susser was able to write in his Cancer: Smoking in the 1960s (1973): "Thus 28 of 29 case-control studies and all seven cohort studies reported in Britain and elsewhere found an association between smoking and lung cancer and roughly the same relative risks." Had investigation stopped at that point the causal hypothesis could not have been faulted because of inconsistencies over relative risks. The early studies were, however, confined to Western, largely Caucasian.
populations. During the past decade reports on Oriental populations have been published and they reveal a very different picture. The differences are so large it is difficult to avoid the conclusion that some of them should be attributed, in one way or another, to genetic factors.

In the American Cancer Society study the mortality ratio for cigarette-only smokers, versus those who never smoked regularly, was found to be 10 for men and 2.6 for women; for British male doctors the ratio was found to be about 14. However, the corresponding ratios in Japan are 1.8 for men and 2.4 for women; among Chinese in Singapore they are 3.8 (men), 2.6 (non-Cantonese women) and 1.6 (Cantonese women); in Bombay, India, they are 2.2 (sexes combined, all cases) and 1.7 (sexes combined and cases were confirmed by histology or cytology), in Northern Thailand they are 1.8 (men, univariate analysis), 2.1 (women, univariate analysis), but only 1.65 and 1.63 respectively (neither significantly greater than unity), in a multivariate analysis that allowed for several "risk factors". A mortality ratio of only 1.574 (sex unspecified) is reported indirectly from the Chinese mainland where, it is said, scientists do not accept that smoking is the main cause of lung cancer. The incidence of lung cancer in Chinese women in Hong Kong is among the highest for the world's women but the risk in cigarette smokers relative to nonsmokers was found to be only 1.74; 53.4% of patients had never smoked cigarettes.

A particularly interesting study has been made in Hawaii of lung cancer in women of Hawaiian, Japanese and Chinese origin: relative risks of 10.5, 4.9 and 1.8, respectively, were found. The 95% confidence limits for the Hawaiians, 6.1 to 18.3, fail to overlap with those for the Chinese, 0.69 to 4.8. The authors concluded: "Cigarette smoking is clearly not the only cause, nor even the major cause, of lung cancer in all populations of women." Although proper standardization between different populations cannot be carried out, the wide contrasts in mortality ratios between male Caucasoid populations on the one hand, and male Oriental populations on the other, suggest that this
Another aspect of consistency can be studied by relating mortality from lung cancer to previous per capita cigarette, or total tobacco, consumption in different countries. When this is done (see Figs. 10.33 and 10.34, p. 352 in my book) a very wide scatter of points is obtained in contrast to the approximately linear relation predicted by the causal hypothesis. The wide scatter of points again suggests that factors other than cigarette smoking are likely to play an important part in determining mortality from lung cancer. In view of the direct evidence for genetic factors in lung cancer it would be most surprising if they made no contribution to these many inter-population differences.

In the London randomized controlled trial 26 deaths and registrations for lung cancer were recorded in the “normal care” group and 22 in the “intervention” group. In the PRF1 study, 28 deaths from lung cancer were recorded in the “usual care” group, but 34 in the “intervention” group, where dietary advice and treatment for hypertension were unlikely to be complicating factors. Overall, 0.74% of men developed lung cancer in the “normal-usual-care” groups and 0.78% in the “intervention” groups. Hence, no support is given to the idea that quitting smoking reduces the risk of lung cancer but, again, numbers are too small to provide a definitive test. Nevertheless, my own conclusions, from different types of evidence, are corroborated.

There can be little doubt that constitutional factors make some contribution to the strong association between cigarette smoking and lung cancer in Western Caucasian populations but it is not yet possible to estimate its extent. In the words of Dr. P.D. Oldham (Journal of the Royal Statistical Society, 141, 460-462, 1978): “... we still do not know how cigarettes cause lung cancer, nor even, if we are particularly rigorous in our use of scientific logic, whether they do.”
Conclusions

According to Alvan Feinstein, Professor of Medicine and Epidemiology at Yale University, and himself a distinguished clinical epidemiologist, "a licensed epidemiologist ... can obtain and manipulate the data in diverse ways that are sanctioned not by the delineated standards of science, but by the traditional practice of epidemiologists." It will be clear from my critique that I fully endorse Professor Feinstein's condemnation. The combination of powerful and admirable humanitarian impulses with an invalid methodology — in the absence of data — has generated many aetiological fallacies and often substituted certainty for agnosticism. Although I have every sympathy with the desire to inform the public of genuine dangers to health I can in no way condone the abandonment of "the delineated standards of science".

If cigarette smoking causes "unnecessary deaths" they form too small a proportion of the total to be detectable by rigorous methods. In short, the Bill S.972 contains spurious assertions about the dangers of smoking and is misguided. Messrs Match, Packwood and Kennedy have been misled; they should be persuaded to withdraw their Bill.

Philip R.J. Burch.
May 16, 1983

The Honorable John P. East
United States Senate
5313 Dirksen Building
Washington, D.C. 20510

Dear Senator East:

Thank you for your letter of April 21, 1983, asking for my comments on Senate Bill S. 772. I am pleased that my statement submitted last year on similar proposed legislation was helpful to you.

I am concerned that the emphasis placed on smoking by both public health officials and federal legislators may divert attention from other factors that require serious study. The "findings" in the current Bill, in my view, are disturbing examples of such undue emphasis. I have prepared a short critique (enclosed) directed to the proposed finding in S. 772 about smoking and the outcomes of pregnancy. My comments are based on my analysis of the research literature and my own investigations.

I appreciate the opportunity to address these serious scientific matters.

Sincerely,

T.D. Sterling
Professor
STATEMENT OF PROFESSOR T.D. STERLING

My current position is University Research Professor at Simon Fraser University, British Columbia, Canada. My teaching career has included appointments at the following universities: Alabama, Cincinnati, Michigan State, Princeton and Washington University. I have served as an advisor to the Food and Drug Administration, the Federal Trade Commission, the National Science Foundation, the National Security Agency, the National Academy of Sciences and the U.S. Veterans Administration. I have been elected a fellow of the American Association for the Advancement of Science and of the American Statistical Association. My work has covered a variety of areas ranging from computer science to statistical analysis and occupational health.

As a scientist, I have devoted much effort to evaluating the quality and analyses of data from epidemiological studies, especially those concerning occupational and environmental exposures. My publications include extensive critical reviews of the scientific literature on such topics as indoor pollutant levels, the possible relationship between smoking and sexual performance and the evidence bearing on smoking and health. I have conducted my own investigations into the potential health effects of occupational exposures on workers and their families. Currently, I am studying health problems which are apparently associated with working in modern air-tight office buildings.
I will specifically address the Congressionally "finding" in S.772 about smoking and the outcomes of pregnancy. I have reviewed much of the related literature and I do not find convincing support for the claims that maternal smoking is responsible for miscarriage, stillbirths, premature births and child weight deficiencies. In my view, this "finding" is highly questionable and represents an uncritical interpretation of the available research data.

Many of the studies which are used to support this "finding" reported only statistical associations between smoking and adverse pregnancy outcomes. It is very important to realize that such data can never be used to establish cause and effect relationships. Further, most investigations failed to take into adequate account other factors which may have bearing on pregnancy outcomes. In particular, occupational exposures and socioeconomic factors must be carefully controlled in studies of smokers and non-smokers. It is recognized that smokers differ from non-smokers in many of these aspects. The results of studies focusing solely on smoking habits have suspect scientific validity.

The claim that at birth infants of smokers weigh less on the average than infants of non-smokers has appeared often in the literature. There are many factors however which might influence birth weight and thus confound any relationship with
smoking. Individuals in lower socioeconomic groups (who are also reported to smoke more) are often smaller in stature and thus would be expected to have smaller offspring. This observation could at least partly explain the association of lower birth weight babies with maternal smoking. It also may be related to the work of the late Dr. Yerushalmy, who compared the birth weights of infants born to women before and after they started smoking. Dr. Yerushalmy found that smoking women tended to have smaller infants even before they began smoking. He also discovered that smoking and non-smoking mothers tended to differ not only in their smoking habits, but in a number of biological and behavioral aspects as well. He concluded that his findings "raise doubt and argue against" the proposition that maternal smoking affects the fetus and suggested that the higher tendency to low birth weight infants in smokers may be due to "the smoker, not the smoking."

My own work indicates that low birth weight may be related not only to social class but also to the occupations of some of the fathers, who may bring toxic materials home on their hair, skin and clothing. My analyses so far have borne out this hypothesis and the data are being prepared for publication.

I also have grave reservations about the scientific validity of the "finding" that smoking increases the risk of miscarriage. This claim, too, is based mainly on epidemiological
studies, which for the most part have not given adequate con-
sideration to occupational exposures or the socioeconomic class
of the women involved. In addition, it is becoming increasingly
clear that the modern working environment of women must be taken
into account in any study of pregnancy outcome. For example,
office buildings in Fort Worth, Michigan, and in Quebec were
studied by some of my colleagues after a number of miscarriages
had occurred amongst the female employees. During my discussions
with the investigators, it became apparent that indoor environmental
conditions, independent of smoking, may have influenced the
unfavorable pregnancy outcomes. Other studies of office buildings
in North America and Europe have indicated that workers have many
complaints ranging from headache and eye irritations to reproductive
system and pregnancy problems.

...itis...has been suggested that hermetically sealed, mechan-
ically ventilated office buildings are like submarines standing on
end—with the result that indoor pollution levels become quite
high. "Photochemical smog" may be formed from the interaction of
these indoor pollutants with ultraviolet light (a component of
artificial "sunlight" light) and might well be responsible for
many of the workers' complaints. Ultraviolet light may in some
way also cause difficulties in pregnancy. The possible relation-
ship of this indoor working environment to health problems deserves
further study. To this end, I have recently designed a work
environment survey questionnaire to obtain information from
workers in modern office buildings about miscarriages, stillbirths, premature infants and low birth weight infants. I believe that this information is crucial in any investigation of pregnancy outcomes.

In summary, I believe the "finding" in S.772 on smoking and pregnancy outcome ignores a complexity of factors which confound the problem. This simple finding does not even take into account the consistent and dramatic shifts in women's employment patterns since the 1940's. Women have moved increasingly into blue collar occupations, where they are exposed to the types of dusts and fumes which have been associated with health problems. Many also work in modern air-tight office buildings. In addition, neither the socioeconomic conditions of women nor their husbands' occupations are considered.

As a private citizen, I am concerned that simplistically placing the blame for unfortunate pregnancy outcomes on the mother's smoking might result in strong feelings of guilt, the consequences of which cannot be estimated. As a scientist, I must conclude that the "finding" on smoking and pregnancy in S.772 could discourage much-needed objective research in this area.

T.D. Sterling, Professor
Simon Fraser University
Burnaby, B.C. V5A 1S6
Canada
May 9, 1983

The Honorable John P. East
United States Senator
Committee on Labor and Human Resources
United States Senate
Washington, D. C. 20510

Dear Senator East:

Thank you for your letter of April 21, 1983 in which you invited me to comment upon S. 772 - the "Smoking Prevention Health and Education Act of 1983" - currently under consideration by the Committee on Labor and Human Resources. In response to your request, I have prepared the enclosed written statement for inclusion in the printed record of the hearings regarding S. 772. I hope the enclosed statement will be of assistance to you and other members of your Committee. This statement should be viewed as that of a private citizen and does not represent the official opinion of the University of California, San Diego.

Yours sincerely,

C. M. Schauer

C. M. Schauer, Ph.D.
CONCERNING THE "SMOKING PREVENTION
HEALTH AND EDUCATION ACT OF 1963"
(S. 772)

P. N. Schrauzer, Ph.D.
La Jolla, California

I am G. N. Schrauzer, Ph.D., an a chemist by training
and profession. I hold a Ph.D. degree in chemistry from the
University of Munich and am a Professor at the University of
California, San Diego. I am a member of several scientific
societies, including the American Chemical Society, the
Association of Clinical Scientists, the American College of
Nutrition and the American College of Toxicology. I am the
author of 250 research publications and have edited two books.

Apart from work in several areas of experimental
chemistry and biochemistry, my current research interests are
centered around cancer prevention, cancer epidemiology, the
study of the roles of trace minerals in human and animal health
and the effects of environmental toxicants. I have done
pioneering work on the prevention of cancer by the essential
trace element selenium. As an acknowledged specialist in this field I have organized and hosted three international conferences on Inorganic and Nutritional Aspects of Cancer during the past six years.

As a scientist and concerned citizen, I wish to comment upon S. 772 - the "Smoking Prevention Health and Education Act of 1983". In my opinion, there is not sufficient scientific evidence to justify the proposed "finding" regarding lung cancer set forth at page 2 of the bill. That "finding" is based, in large part, upon unwarranted extrapolation of ambiguous epidemiological data.

Those who claim smoking causes lung cancer rely upon reported statistical associations and ignore the inconsistencies in the smoking causation theory described in the scientific literature. For example, to date, no one has been able to produce lung cancer in laboratory animals through exposure to fresh, whole cigarette smoke. Furthermore, the vast majority of smokers never develop lung cancer and there
are serious inconsistencies in the epidemiological data. For instance, a Japanese male smoking 50 cigarettes per day is reported to have a lower risk of dying from lung cancer than a British doctor smoking only 1-14 cigarettes per day.

In addition, no ingredient or combination of ingredients, as found in fresh, whole cigarette smoke, has been shown to cause human lung cancer. Tobacco smoke is a very complex mixture of thousands of ingredients. Some of those ingredients, in isolation, have been reported to be carcinogenic in test animals. However, this does not mean that tobacco smoke is harmful to humans. Compounds that are carcinogenic in test animals, when applied in isolation, have been found to be anti-carcinogenic when applied in combination with each other. Since it is probable that the many hundreds of compounds present in smoke interact with each other, it is highly artificial to focus upon the effects of any one ingredient or combination of ingredients in isolation from the others. It has long been known that certain smoke constituents
act as anti-carcinogens in test animals. Tobacco belongs to the selenium accumulating group of plants and selenium has been shown to possess anti-carcinogenic properties. Constituents of cigarette smoke previously thought to be totally lacking in carcinogenic activity have recently been found to be anti-carcinogenic when applied with true carcinogens in test animals.

Cancer is an extremely complex, multifactorial disease. Studies report that many factors, other than smoking, are statistically associated with cancer. Some of these factors are familial predisposition, exposure to tumor viruses and other biological agents, exposure to ionizing radiation and industrial carcinogens, diet, exogenous environmental factors and stress.

Further, studies indicate that a number of agents normally present in foods may have activating effects on tumor viruses and that there is an association between lung cancer mortalities and other variables of life-style and diet. Thus,
lung cancer mortalities are correlated with the per capita intakes of sugar and milk, and with the consumption of seafoods. It also has been suggested that the consumption of diets rich in pro-vitamin A (carotene) may have lung cancer protecting effects. In summary, there is not sufficient scientific evidence to justify the proposed "finding" regarding lung cancer set forth in S. 772.

Speaking as a private citizen, I do not believe the federal government should interfere with the life-style preferences of the American people. Passage of S. 772 would set the stage for further crusades against other, imagined "culprits" in American life. It would only be a matter of time until legislation concerning the claimed health effects of beer, ice cream and eggs would have to be considered. For each of these, arguments similar to those voiced against cigarette smoking may be formulated and Congress would soon have to decide whether eggs should carry a label, "Warning: Eating Eggs -- causes high blood pressure -- is a major cause of DEATH."

Selection of a life-style is, and must remain, a constitutional right of the individual and must not be dictated by government.
May 11, 1983

The Honorable John P. East
United States Senate
Washington, D.C. 20510

Re: S.772

Dear Senator East,

Thank you so much for your letter of May 4. Inasmuch as I consider myself somewhat of a punctualist, I am doubly embarrassed that your letter of April 21st apparently was lost.

Allow me to thank you for your keen interest in this matter which has concerned me for a period now of over twenty years. I must say that the matter is no more clear than it was twenty years ago.

I am enclosing a recent copy of my comments which I hope will be pertinent to S.772.

Sincerely yours,

Jack Q. Farris, M.D.
Professor of Surgery, Emeritus
Associate Dean
School of Medicine

Enclosure
The Honorable John P. East  
United States Senate  
1939 Dirksen Building  
Washington, D.C. 20510  

Dear Senator East:  

In reply to your letter of 21 April, I have read Senate Bill S. 772.  
As a scientist interested in heart disease epidemiology for many years, I have  
strong concerns regarding certain claims made in the bill with respect to cigarette  
smoking. These concerns I have expressed in the enclosed statement.  

Sincerely,  

[Signature]  

CC:rs  

Enclosure
I am Dr. Earl C. Seltzer, Honorary Research Associate at the Peabody Museum, Harvard University and Professor of Nutrition, Tufts University. I was formerly Senior Research Associate at the Harvard University School of Public Health. My work in the smoking and health area is extensive and I have published over thirty-five (35) articles since 1964 on the subject, many dealing with the relationship between smoking and coronary heart disease (CHD). I was a consultant to the Surgeon General's Advisory Committee on Smoking and Health (contributed a section to the 1964 Report) and am presently a Fellow of the Council on Epidemiology of the American Heart Association. My investigative work in the heart disease field includes participation in studies involving the Framingham Heart Study, The American Medical Association, the Veterans Administration and the Kaiser-Permanente Foundation.

S. 772 proposes that a label appear on cigarette packages which states the Surgeon General's conclusion that "Cigarette Smoking is a Major Cause of Heart Disease." This proposal is apparently based on a claim in the "findings" part of the bill.

Such a bald statement is not based on consistent, valid, demonstrable scientific evidence, and is without established proof. Apparently, it is based on the views of a succession of Surgeon Generals subsequent to the 1964 Report, on the statements of the American Heart Association, the Framingham Heart Study and others.
The primary basis for these views can best be characterized by statements from the 1981 Surgeon General’s Report and by the Framingham Heart Study. The 1981 report stated: “The effect of smoking on CVD risk fulfills many epidemiological criteria for a causal association: powerful, independent, dose-related, and reversible.” Kennel of the Framingham Heart Study stated: “Causal inferences are supported by the fact that the relationship is strong, consistent, demonstrated prospectively, independent of associated risk factors, and can be explained by the known effects of cigarette smoking on the cardiovascular apparatus. Finally, it has been shown that those who quit smoking have only half the risk of those who continue to smoke. Epidemiologic data have...tended to indicate an independent, transient, noncumulative, reversible, triggering effect of cigarette smoking.” (JAMA, 1981)

Accordingly, the main evidence advanced for a causal relationship between cigarette smoking and CVD is essentially epidemiologic, to the effect that the association is (1) strong, (2) consistent, (3) dose-related, (4) independent of associated risk factors, and (5) reversible by stopping smoking. (6) In addition, the association is said to be explained by known effects of cigarette smoking on the cardiovascular system. Let us examine these claims in turn.

Claim 1. Is the relationship strong? The 1964 Surgeon General’s Report stated that in the U.S. the mortality ratio of smokers to nonsmokers is 1.7. But, this is not a strong relationship, according to Professor Hutchison of the Harvard School of Public Health. (Bull NY Acad Med, 1968) This independent observer feels that a mortality ratio of two or less is
actually indicative of a weak relationship which may possibly be explained by confounding factors.

Can the relationship be characterized as "strong" when the 1979 Surgeon General's Report, although discussing smoking and health studies throughout the world, limits its conclusion to "men and women in the United States," and Kannel of the Framingham Study talks about the excess CHD death rates of smokers "in most of Western societies"? (AHJ, 1981) If the relationship between smokers and CHD is "strong," why is it not present in all Western societies or in non-Western societies?

Claim 2. Is the relationship consistent? Since no significant statistical associations between smoking and CHD have been reported in Finland, the Netherlands, Italy, Greece, Yugoslavia, Japan and Puerto Rico, it is clearly incorrect to claim that the relationship is consistent.

The relationship is not even consistent in the Framingham Study. It appeared to be consistent in initial analyses, but when standard risk factors and such psychological factors as Type A personality were controlled, it was found that "cigarettes smoked per day" were not predictive of CHD and myocardial infarction incidence in men, or of CHD and angina pectoris incidence in women. (Am J Epidemiol, 1980)
assocation of smoking with the incidence of angina pectoris is not clear? The Framingham Heart Study also reports that "the relationship to angina pectoris is modest, if it exists at all" (JAMA, 1981).

Is the relationship consistent when the relative risk progressively declines with increasing age? Selzer demonstrated and Kannel noted that the benefits of quitting smoking do not extend beyond the age of 65 as regards heart attacks. (Am J Med Sci., 1978; JAMA, 1981). Furthermore, the 20 year follow-up data of the Framingham Study show no excess incidence of CHD in men or women 55 years and over. (Framingham data made possible by Selzer as consultant.)

Can the relationship be characterized consistent when the Framingham Heart Study reports an absence of a relationship of smoking and CHD in women?

Claim 4. Is the relationship dose-related? CHD rates do not consistently show a rising gradient in relation to an increased amount of cigarette smoking. (JAMA, 1968). In some studies, the gradient is actually reversed. (JAMA, 1968). I found that data from the Framingham Study do not show a consistent rise in CHD risk with increasing amounts of cigarette smoking because of the "heterogenous" character of its "none" category, a deceptive procedure. The "none" category in Framingham consists of a combination of never smokers, ex-smokers, pipe and cigar smokers. A Framingham Study report confirmed that "there is no distinct..."
dose-response relation of CHD and death rates with increasing amounts of cigarette smoking over an 18-year surveillance period. (Lancet, 1974)

There is no significant dose-response relationship between smoking and CHD in studies of Finland, the Netherlands, Greece, Italy, Yugoslavia and Japan. (Circulation, 1970)

No claims have been made lately about the alleged association of CHD with duration of cigarette smoking. In the combined Albany and Framingham Heart Studies (JAMA, 1962), no association was found between duration of heavy cigarette smoking and risk of myocardial infarction. In a study of Canadian veterans, no consistent gradient occurred between CHD mortality and years of cigarette smoking. (JAMA, 1968) In Kahn's analysis of Dorn's U.S. veterans data, there was also no significant relationship between duration of cigarette smoking and risk of death from CHD. (JAMA, 1968)

Claim 4. Is the relationship independent of associated risk factors? Smoking has been found to be associated with CHD independently of associated risk factors in some studies when only cholesterol and blood pressure were considered. But this generally has not been the case when other risk factors were investigated, such as Type A personality, and other psychological factors (as seen above in the Framingham Study).
Claim 5. Does stopping smoking reduce the risk of CHD? This claim is based on studies from some Western countries which report that CHD rates of ex-smokers are lower than those of continuing smokers. The 1974 Lancet article on the Framingham study is the most widely quoted source for claiming that the rate of CHD among those who stop smoking is reduced to one-half that of continuing smokers. When I was provided with the CHD rates of Framingham ‘never-smokers’ (which had been omitted in the article), however, I was able to show that the CHD rates of ex-smokers were far below those of “never” smokers,” while the rates of “never smokers” were not far below those of continuing smokers. (Selzer letter to Lancet, 1977)

Additional evidence about the fallacy of the claim that stopping smoking reduces the rate of CHD comes from two other sources. The first is a study challenging the assumption by epidemiologists that ex-smokers and continuing smokers are alike in all pertinent characteristics, except for their smoking habits. If this assumption is false, which I believe it to be, then comparisons of the two groups would be biased as to their eventual CHD rates. Friedman, Selzer, and others have shown that ex-smokers, when they all smoked, were not a representative sample of the population of continuing smokers. The data showed, in fact, that ex-smokers were at lower heart-disease risk (and healthier) before they stopped smoking, than those persons who continued to smoke. (Iz Carpe Dia, 1979). This would appear to account for the fact that ex-smokers have lower CHD rate outcomes than continuing smokers, not
Because they stopped smoking but because they are different kinds of persons to begin with through self-selection.

In a subsequent article published in the NLM, Friedman and others tried to negate the results of that paper by applying theoretical correction techniques through multivariate analysis to the lower CHD-related characteristics of the ex-smokers. They claimed that the lower CHD-related characteristics usually made little difference, that ex-smokers still showed lower CHD outcomes than continuing smokers. Although this paper was criticized by Burch and Seltzer (Burch and Seltzer letters to NLM, 1981), Friedman evaded virtually every criticism in his reply (Friedman letter to NLM, 1981). Friedman did not explain how he could accept the value of CHD-related characteristics (risk factors) in one paper and deny their importance in a second paper. Nor did Friedman explain why the mortality data in his later paper showed that never smokers exhibited higher CHD rates than the ex-smokers (the same anomaly as that found in the Framingham study). Does this mean that it is better to begin smoking and then to quit than never to have smoked at all?

The second source of evidence which points to the fallacy of the claim that stopping smoking reduces the risk of CHD is the first large-scale "intervention" trial of the effect of smoking cessation in middle-aged London civil servants at high risk of cardiovascular disease. (Epidemiol Comm Health, 1978, 2:92) After nearly eight years of
surveillance, the group subject to intervention for smoking cessation showed no significant improvement in its rate of overall mortality and heart disease mortality over the group which was not urged to stop smoking. Thus, the alleged reversibility of the risk to the smoker's life was not demonstrated.

This result is consistent with a more recent intervention trial from Norway which examined the effects of both smoking and cholesterol reduction. (Lancet, 1981) Although reductions in dietary cholesterol produced a statistically significant decrease in CHD, reductions in smoking did not. This led the authors to conclude that past studies may have "overestimated the decline in risk of CHD" following reduced cigarette consumption.

Even more recently, the results of the massive Multiple Risk Factor Intervention Trial have appeared. This intervention trial was designed to show that reductions in "risk factors," including smoking, would reduce risk of death from heart disease. It failed, because of the complexity and importance of this study I have provided an extended discussion of its findings in the Appendix.

Claim A. Kannel of the Framingham Study claims that: "causal inferences can be explained by the known effects of cigarette smoking on the cardiovascular apparatus." (JAMA, 1981) There is no satisfactory evidence for this claim, and, in fact, there are contradictions. The cold
reality is that the mechanisms by which tobacco smoking allegedly causes CHD have not been established. Much of the evidence to support this statement is contained in Seltzer's article, "Smoking and Coronary Heart Disease: What Are We to Believe," published in the American Heart Journal as an editorial in September, 1980. This editorial discusses those statements of the 1979 Surgeon General's Report which illustrate the absence of firm evidence of mechanisms by which smoking affects CHD, such as: (1) little is known about the mechanisms by which smoking enhances atherogenesis; (2) further research is needed to show the mechanisms of sudden death and its precursor states; (3) the data on the effect of smoking on blood lipids are not very uniform; (4) the association between cholesterol and smoking is minimized; (5) the acute and transient effects of smoking are to increase heart rate and blood pressure to a minor degree; (6) smoking is not a risk factor for hypertension; (7) the association of smoking with necropsy manifestations of atherosclerosis, shown principally by Strong's group in New Orleans, now has been contradicted by a study by Holm, Strong and others who found that smoking did not show a significant association with coronary artery lesions. (Atherosclerosis, 1981)

More evidence comes from other sources. An editorial in the August 1980 British Medical Journal states that the mechanism by which smoking affects coronary heart disease is unknown. The American Heart Association Heartbook states: "The mechanisms by which cigarette smoking is
associated with higher rates of coronary heart disease are not yet fully understood.

The call of S. 712 for required disclosure of levels of nicotine and carbon monoxide on cigarette packages is without justification with respect to coronary heart disease because their pertinence to this disease has not been adequately established. For many years, there have been claims and counterclaims by a number of investigators as to whether nicotine or carbon monoxide in cigarette smoke exerts a harmful effect on the coronary system and on atherosclerosis; sometimes roles for both nicotine and carbon monoxide have been claimed by investigators. On the other hand, Dr. Wald and associates (Lancet, 1981) asserted succinctly that their data strongly suggested that nicotine does not cause coronary heart disease. Dr. Russell and associates (British Medical Journal, 1981) conceded: "It is not known whether nicotine or carbon monoxide is the major culprit responsible for cigarette-induced coronary heart disease."

This subject was examined in an international workshop conference in Berlin, and the following quotations illustrate the state of knowledge on this problem. Drs. Astrup and Kjeldsen (Preventive Medicine, 1979) stated: "It is very unlikely that nicotine is the responsible agent in tobacco smoke for the increased incidence of atherosclerosis in smokers." In the same conference, Dr. Astrup also admitted that the results of his previous publications that carbon monoxide was
responsible for increased atherogenesis were not confirmed since the laboratory techniques were faulty. Dr. Doyle, the former director of the Albany Heart Study of the U.S. Public Health Service asserted: "How cigarette smoking exerts its vasculotoxic effect remains undetermined despite intensive investigation." Dr. Schievelbein, in summing up the role of carbon monoxide and nicotine in the etiology of atherosclerosis and cardiovascular disease, concluded that: "Neither substance has any influence comparable to human arteriosclerosis, on the development of CVD (cardiovascular disease)."

In a recent article published in the New England Journal of Medicine (1983), Dr. Kaufman and coworkers, of Boston University and Harvard, reported on their attempt to evaluate the roles of carbon monoxide and nicotine levels on risk of heart attacks. They said that their results showed that heart-attack risk "did not appear to be related" to the amounts of nicotine or of carbon monoxide in the cigarettes smoked by the subjects in their study.

From the Surgeon General in his report titled The Changing Cigarette stated: "Estimation of the impact of varying cigarettes on coronary heart disease risk is difficult, because the exact etiologic agent(s) have not been identified." And finally, Sir Richard Doll in a February 1983 issue of the British Medical Journal acknowledges that it has not been established that either nicotine or carbon monoxide is the responsible agent in coronary heart disease.
This is also shown by the conclusion of the 1981 Report. Despite radical changes in the composition of cigarettes over the last 10 to 15 years (filter, tar, nicotine), there was no clearly demonstrated effect on cardiovascular disease. This surprising result is clearly consistent with the proposition that amounts of tar, nicotine, and carbon monoxide in cigarette smoke have no demonstrable effects on CHD rates, and that these components of tobacco smoke are not proven etiologic agents.

It is clear that any possible roles of nicotine and carbon monoxide in coronary heart disease are neither certain nor established. Accordingly, the call for required printing of the levels of these substances is to place a scientific imprimatur on this subject by a legislative body when the problem itself has not been satisfactorily delineated by medical science.

As a final observation, since most cardiologists believe that atherosclerosis is the most important factor in coronary heart disease development, they claim that smoking enhances atherosclerosis, despite the lack of definitive evidence. On the other hand, the Framingham Heart Study has disavowed the atherosclerotic argument and claimed that the effect of smoking on the heart is acute (not progressive), and disappears promptly when smoking is stopped. These are contradictory claims and illustrate the lack of definitive knowledge in this field.
In conclusion, it is reasonable to believe that stopping smoking does not reduce the risk of coronary heart disease. This has been principally indicated by the MRFIT program, by the randomized controlled trial of smoking cessation in middle-aged civil servants of high coronary risk, by the bias of selection (comparability bias) inherent in the many observational studies as shown by the results of the Kaiser-Permanente ex-smoker study.

It is also reasonable to believe that cigarette smoking has not been established as a direct cause of coronary heart disease. This is evidenced by the negative results of the MRFIT program; by the fact that contrary to conventional wisdom from observational studies, the association of cigarette smoking is neither strong, nor consistent, nor consistently dose-related, nor independent of associated confounding factors (as seen in the Framingham Heart Study); and by the fallacy of the claim that stopping smoking reduces the risk of heart disease. In addition, the mechanism(s) by which cigarette smoking is alleged to enhance atherogenesis or affect coronary heart disease is admittedly not established, nor does medical science know the etiologic basis of coronary heart disease.

The claims made against cigarette smoking relative to coronary heart disease are either wrong, inconsistent, selective, unsubstantiated, and, in many instances even contrary to statements of the Surgeon General, the Framingham Heart Study, and the American Heart Association.
It is clear from the above that extensive research data do not support the claim that cigarette smoking is a major cause of heart disease.

Signed Carl Vintage

Date July 1, 1987
Appendix

There is danger both to the public interest and to the integrity of congressional bodies in legislating alleged scientific dogma (in this instance, that cigarette smoking is a major cause of heart disease) when the evidence for such a dictum is clearly in flux and in doubt. This is manifestly illustrated by the results of the massive, expensive, and lengthy clinical investigation termed Multiple Risk Factor Intervention Trial (MRFIT) sponsored by the National Heart, Lung and Blood Institute of N.I.H. This 10-year, $115,000,000 investigation involving 12,864 subjects was the combined product of 22 clinical centers across the country, several coordinating centers and more than 250 investigators. The results of the MRFIT program have recently been published in the Journal of the American Medical Association (September 21, 1982), and showed that very substantial reductions in the levels of three so-called major "risk factors"—blood pressure, cholesterol and especially cigarette smoking—in the intervention group did not result in the expected reductions in deaths from coronary heart disease. The significance of this finding is such that it bears detailed discussion.

MRFIT was a randomized primary prevention program designed to demonstrate the effect of interventions on hypertension, high cholesterol levels, and cigarette smoking in high-risk men (aged 35-57 years) in the United States on mortality from heart disease. The rationale for this program was the stated certainty on the part of the sponsors that reductions in the levels of the so-called major "risk factors"—blood pressure, cholesterol, and cigarette
smoking — would result in a substantial reduction in deaths from coronary heart disease. Apparently, this would be the ultimate demonstration that these so-called "risk factors" were causally related to heart disease.

The design of the study was as follows: from a first screening of 361,662 men who volunteered for the study, ultimately 12,866 were selected. Each was identified as being of high-risk for heart disease on the basis of combinations of high levels of blood pressure, high levels of cholesterol, and of cigarette smoking. These 12,866 men were then randomized into two groups of approximately the same size, one group receiving continuous "special intervention" (SI) over the length of the program aimed at cessation of cigarette smoking, reduction of levels of blood pressure by medication and other means, and lowering the blood cholesterol levels by changing their dietary habits. The men in the other group, termed "usual care" (UC), were not subject to intervention but were left to their usual sources of health care, a sort of control group. The two groups were then followed for 6-7 years for mortality outcomes.

Before discussing the actual results, it should be pointed out that the key risk reduction goals were essentially met. Over the follow-up period the SI group showed a 44% greater decline in diastolic blood pressure over the UC group, a 38% greater decline in serum cholesterol, and a 100% greater decline in cigarette smoking over that seen for UC men. The WHIPIT reported that the smoking cessation in the SI group was "much more successful than had been expected," the blood pressure reduction "exceeded" the desired drop, and the effect on cholesterol lowering was "considerable." On the basis of these risk factor
Incidentally, it is important to note that the failure of MR fit results to fulfill the predictions of the Framingham Heart Study makes it clear that the Framingham Heart Study data are now out of date, and no longer apply as satisfactory guidelines to physicians as to their patients' risk for heart disease.

The failure of the MR fit program is not just an aberration. We have noted that Rost and Hamilton's randomized controlled trial of smoking cessation
in middle-aged civil servants did not show significantly decreased mortality from smoking reduction. With respect to hypertension, the Veterans Administration Study found that while treatment was effective in connection with stroke, there was no demonstrably significant benefit for coronary heart disease mortality. The Food and Nutrition Board of the National Research Council found no evidence that a reduction of cholesterol in diet will help prevent heart disease. In the Oslo intervention study on cholesterol and smoking, initial cigarette smoking was a non-significant risk factor for acute myocardial infarction incidence. Dr. Mehl, an eminent Dutch cardiologist stated: "... modification or treatment of risk factors... does not demonstrably or predictably affect coronary artery disease." An editorial in the prestigious British Medical Journal summed up the matter as follows: "Such as we might like to think otherwise, it is not yet possible to prevent coronary heart disease in the community—let alone in an individual."

The matter of cigarette smoking cessation in the MRFIT program deserves special attention. We have noted above that MRFIT reported that smoking cessation in the special intervention group was "more successful than had been expected,"—in fact 100% greater than that which occurred in the usual care group. Accordingly, it is highly meaningful that even under such a condition the MRFIT report was forced to conclude that their overall results failed to show a beneficial effect on coronary heart disease or total mortality. For, if there was any one potential "beneficial effect," it was believed by the sponsors of the experiment that it would come from smoking cessation. Even the Task Force on Atherosclerosis of the NHLBI (which recommended the MRFIT program) was
somewhat sanguine about the possible "beneficial effect" of the lowering of blood pressure and serum cholesterol in reducing heart disease mortality, by they were very positive in their expectations of a "beneficial effect" of smoking cessation. This is why the absence of any "beneficial effect" on heart disease mortality was completely "unexpected" and caused "surprise" and consternation among the MPH study investigators. Their favorite risk factor -- "smoking cessation" -- did not work.

Instead of the MPH report concluding that smoking cessation did not work, it attempted to circumvent the basic study design of the experiment, and to suggest that smoking cessation was beneficial. As explained below, such analyses are fallacious.

The basic design of the MPH study involved randomized controlled simultaneous reductions of the three so-called major "risk factors" -- blood pressure, cholesterol and cigarette smoking. It was stated in advance of the trial, and even after the trial, that it would not be possible to assess the effects of any of the "risk factors" separately. Furthermore, as stated by the MPH investigators, it was necessary that comparisons of mortality rates for 61 and 62 men than subgroups defined by pretreatment characteristics preserve the comparability provided by randomization. But the MPH report ignored its own stipulations of randomization. It compared the subsequent death rates of men who quit smoking in the first year with the rates of those who continued to smoke. And found that the quitters had significantly lower rates of coronary heart disease mortality. This was done outside of randomization. The
MRFIT recognized this fallacy in its statement: "It must be emphasized that this kind of analysis does not preserve the randomized controlled design of the MRFIT, and must be interpreted with regard for the possibility of confounding by many factors. . . .) There must be caution in reaching conclusions from such subgroup data."

In other words, this analysis of quitters and continued smokers was improper, and it cannot be stated from MRFIT that smoking cessation reduced the rate of coronary heart disease mortality. Obviously, the MRFIT report was improperly attempting to show a "beneficial" effect of smoking cessation, and admitted it was improper. Anyone who refers to the MRFIT analysis as suggesting that quitting smoking reduces coronary heart disease, is lifting the MRFIT statements out of content unless it is also pointed out that the analysis fails to preserve the randomization comparability design and is therefore questionable.

The absent failure of the MRFIT program to demonstrate "beneficial" effects on heart disease mortality has reluctantly forced some to seek intervention evidence and to return to their reliance on the old observational data with its inherent bias of selection. Editorials in The Lancet and the British Medical Journal appear to take this position.
Hon. John P. East
United States Senator
from North Carolina
Washington D. C. 20510

Dear Senator East:

Thank you for your letter requesting my comments on the pending bill S. 772. I have been interested in the questions of smoking and health for many years, even before the first Surgeon General's Report. Although I have retired from active laboratory research, I still keep up with the published literature in this field.

My statement is enclosed. If you have any questions or if you wish any clarifications, please feel free to contact me again.

Very truly yours,

Arthur Purdy, Ph.D.
Distinguished University Professor Emeritus

UNIVERSITY OF SAN FRANCISCO
INSTITUTE OF CHEMICAL BIOLOGY
R. James Brown, Ph.D., Director
Arthur Purdy, Ph.D., Director Emeritus

4 May 1983

465

HARNEY SCIENCE CENTER
SAN FRANCISCO, CALIFORNIA 94117
415/664-6445

472
STATEMENT OF ARTHUR FURST, PH.D.

I am Arthur Furst, Ph.D., Distinguished University Professor (Emeritus) and Director (Emeritus) of the Institute of Chemical Biology at the University of San Francisco. As the attached copy of my curriculum vitae shows, I have been a professor of pharmacology at Stanford University School of Medicine and a (visiting) clinical professor of pathology at the College of Physicians and Surgeons of Columbia University. I am listed in Who's Who in the World and Who's Who in Science. I am a consultant (temporary) to the World Health Organization. In fact, I was a member of the international working group that wrote IARC Monographs Volume 2 and 23 on Evaluation of the Carcinogenic Risk of Chemicals to Humans. I just returned from a meeting at the World Health Organization in Lyon, France, where I spoke on the mechanism by which nickel causes cancer. I also consult frequently with governmental agencies and private industries on a variety of environmental health problems. For example, last year I was asked by the EPA and the American Water Works Association to chair a conference which will bring together experts to discuss water quality standards.

I have read S. 772 with great interest, for I have spent over thirty years in scientific research, much of it studying the issue of smoking and health. My original laboratory work on carcinogenesis antedates the first Surgeon General's Report on Smoking and Health. Over the years, I have studied the possible effects on animals of both whole smoke and various smoke components. I have also investigated the carcinogenicity of a wide variety of substances, particularly the heavy metals, and I have carefully monitored the
Perhaps this background will help explain my concerns about § 772. I question the scientific basis of this proposal to amend the Public Health Service Act and the Federal Cigarette Labeling and Advertising Act, because it makes such flat, dogmatic statements about a scientific area which I know to be fraught with uncertainties.

A Congressional finding that “smoking is the primary cause of lung cancer” implies a scientific certainty that I, as a scientist, believe to be unwarranted. My skepticism arises chiefly from my extensive knowledge of and first-hand experience with animal experiments on tobacco smoke and lung cancer.

For many years, I tried to induce lung cancer in animals with cigarette smoke, with no success, despite the most sophisticated smoking machines available. Not only were my colleagues and I unsuccessful, but every other investigator who attempted to induce lung cancer in animals by inhalation of fresh smoke also failed.

There have been a very small number of published reports of lung cancers occurring in experimental animals during smoke inhalation experiments. Anyone attempting to interpret these as showing that smoking causes lung cancer must understand that animals, like humans, do spontaneously develop lung cancer even in the absence of any suspected carcinogen. The key question that an investigator must ask himself is whether any cancers that develop in his animals are actually a result of the experimental exposure. For example, rodents have a definite spontaneous rate of adenocarcinomas (malignant tumors) and adenoma (non-malignant tumors) development. I have examined the data on the reports of increased adenocarcinomas in smoke-exposed animals and have found no difference between the incidence reported in the experimental animals and the normal baseline, spontaneous rate.
The lung cancer cell type traditionally associated with human smoking is squamous cell carcinoma. The spontaneous rate of this cancer is considerably lower than that of adenocarcinoma in rodents, but it too does occur naturally in these animals. There have been very few reports of a squamous cell cancer in smoke-exposed animals, and generally the authors made no comment on these findings. I assume that they concur with me that they were seeing nothing more than spontaneous production.

One of the seminal experiments that has received the most attention is that on beagle dogs conducted by Auerbach et al. Over a decade ago, they claimed to have succeeded in producing lung cancer in their beagles, but because their experimental methods were seriously flawed, I consider their conclusions to be unfounded. Because the Auerbach experiment was recently described as a positive inhalational study result, I feel compelled to repeat some of the criticisms of that study that I voiced in 1972. Any time this experiment is praised, there seems to be no emphasis on the fact that the dogs received the smoke through a tracheostomy. This in no way resembles human smoking. Unless the pictures I have seen are wrong, the smoke was initially forced into the lungs through the trachea by use of a pump! The speed of the pump was without regard to the normal respiration rate of the dogs. Further, several of the dogs died from infections, aspiration of foreign material, etc., which is unusual in a well controlled experiment. This experiment has no meaningful relationship to the human experience, and any reports of tumors should be considered in light of the fact that the experimental data were not made available to an independent panel for evaluation, as requested. Also, since beagle dogs do get lung cancer spontaneously, a discussion of this fact would have been useful to readers interested in evaluating the Auerbach work.
The design and conduct of meaningful animal experimentation require a great deal of sophistication. I have reviewed a vast number of research proposals and reports in the past 30 years, and I can assure you that a large number of the experimental designs or the conclusions drawn from the experimental data do not stand up to rigorous scientific scrutiny. One of the simplest requirements that is often overlooked is the need to select from litter mates the controls and the experimental animals. Even with exactly the same strain, the rodents' spontaneous rate of tumor appearance will vary from one supplier to another; this can cause serious problems with the interpretation of the data. Thus, even if positive results are obtained in one experiment, a replication using animals from a different litter may yield nothing unusual.

It is also extremely important to use “clean” animals. Parasites or infectious diseases in the animals can seriously affect experimental results, even if the animals are successfully treated before the experiment begins. For example, we can't be sure what effect the parasite infestation in Bentenall's Syrian Golden hamsters had on his inhalation study results. Similarly, the possibility that Auerbach’s beagles had lung worms causes concern. In my own work, I always try to avoid using unclean animals by checking them very carefully before beginning an experiment. If I discover later that my animals are not clean, I terminate the experiment and start again with new animals. That is the only way that I can feel certain that the infestation or infection will not affect the experimental findings.

In summary, based on my own research and familiarity with the literature, I have concluded that no reliable, reproducible animal studies have shown that the inhalation of cigarette smoke causes lung cancer. I might add that skin-painting experiments are inappropriate for studying the question.
of tobacco smoke and cancer. We must insist that animal experiments simulate, as closely as possible, the human experience — and skin-painting, as well as certain other experimental techniques, fail to mimic adequately human inhalation.

Various groups convinced of smoking's causal role in lung cancer development have attempted to ignore or explain away the negative results in animal inhalation studies. For example, I was once challenged in a scientific meeting about the mice I used in my smoke inhalation studies. The individual argued that this strain could not develop lung cancer, and that is why I failed. But this is not true, because we have documented well the production of lung cancer in these animals. Over a period of years we published a series of papers describing our technique of instilling carcinogens in the lungs of mice, the sensitivity of various strains of mice to carcinogens, and the response of the animals to different hydrocarbons. Yes, development of all histological types of lung cancer is common in animals treated with various carcinogens. It is even possible to rate experimentally the potency of carcinogens as inducers of lung cancer.

In experimental carcinogenesis, replication by independent investigators is essential to establish the validity of the findings. Other researchers have been successful in replicating my experimental production of lung cancers. For example, in a 1982 issue of Cancer Research (vol. 41), Henry, et al., published a complete confirmation of my research. They used the same technique, strain of mouse, chemical, and reported the same time of lung cancer appearance. It is in this context of successful lung cancer induction techniques that the failures of smoke inhalation studies should be judged.
In general, the major health claims against cigarette smoking are based mainly on epidemiological studies. From the standpoint of science, epidemiological studies can only point out statistical associations between a factor (such as smoking) and disease; they cannot prove a causal relationship. Yet, the findings in S. 772 are stated as causal relationships, as though all is proven. Thus, as a scientist with over 30 years experience in laboratory research, I must disagree with those findings on the grounds that they are overstatements of the scientific knowledge — all is not proven in the smoking and health area.

I heartily agree with the recommendation that more research must be funded and conducted. Yet, I must emphasize that negative results can also be very important. Scientific knowledge (in this case, of lung cancer causation) advances only by publication of both successes and failures. This is why I have been troubled that so little information is given in the reports by the Surgeon General about the negative experimental results in the lung cancer area. A case in point is Dr. A. Wetter's work which showed that hamsters, as a result of their exposure to cigarette smoke, not only failed to develop lung cancer, but also lived longer! Yes, the mandate that the Secretary of the Department of Health and Human Services "collect, analyze, and disseminate" smoking and health information is excellent if both positive and negative findings will be reported. Only with this unbiased approach can the needs of both science and the public in general be met. I feel compelled to say that past performance by official agencies has been characterized by highly selective reporting.

I am particularly disturbed by the 1982 Report of the Surgeon General Health Consequences of Smoking. Like so many of its predecessors, it often omits references to material contradictory to the positions it has adopted.
Why the omissions? Why should a government document selectively present only that part of the evidence that supports its position?

In general, this Report provides no new information that would make me change my mind about the lack of good scientific information on smoking and health.

I agree with the Report's recommendation that major research efforts should be devoted to elucidating the mechanisms of lung cancer development. At this point, we have a number of theories, not proof, and in many instances not very good theories.

The writers of the Report assume that the case against cigarette smoking is proven, and that the lack of knowledge about the mechanisms involved is only the finishing touch — not really essential to prove the point. I cannot disagree more strongly. Unlike the writers of the Surgeon General's Report, I believe that until we have that proof, any conclusions regarding causation are premature.

I disagree with the Report's discussion of the animal inhalation experiments using Syrian golden hamsters (many of them conducted by Donnenwirth, et al.) A summary section of the Report says that "studies on smoke inhalation with the hamster now appear suitable" for evaluating the "tumorigenic potential" of cigarette smoke. This seems highly incompatible with the Report's earlier comment that "why these inhalation experiments with hamsters did not induce carcinoma of the lung remains to be elucidated." Lung cancer can be induced in hamsters, by using a variety of substances. Also,
significant amounts of cigarette smoke do reach the hamsters' lungs. So why don't the hamsters get lung cancer after exposure to tobacco smoke?

The 1982 Report concludes that inhalation studies have basically failed to induce lung cancer in animals, but then it recommends that further research efforts in this area should receive limited priority. I find that incomprehensible. The animal data are significant negative evidence. They basically contradict the popular interpretation of the epidemiological data. Why? We must have good research to find out.

The 1982 Surgeon General's Report lists routes of administration for chemicals that are being tested as carcinogens. The intratracheal route (the means by which agents can be deposited in the lungs without the use of surgery) is not mentioned. Is that because when tobacco condensates were instilled by this route, no cancers were induced?

I am not in any way advocating smoking as a scientist, I am concerned with presenting the best information to the public, both positive and negative. I am also very concerned that if all the diseases noted in this bill were assigned by federal law to smoking, there will be a severe decline in research on and attempted control of many known or suspected environmental causes of disease. For example, this federal law can only have an adverse effect on efforts to clean up our environment, to control smog controls in cars, and to remove the particulate matter and noxious gases in the atmosphere.

I have lectured throughout California for over five years in programs on the Biology of Cancer sponsored by the American Cancer Society. Such contact has led me to believe that the public is well informed on what the
preamble in S. 772 refers to, as the "health consequences of smoking." Every package of cigarettes, every magazine ad, and every billboard ad carries the Surgeon General's warning. Furthermore, the American Lung Association, the American Cancer Society, and the March of Dimes are continually funding radio and TV announcements against smoking.

I cannot agree, therefore, with this legislative requirement that the government spend even more time and effort to inform the public as though they are ignorant of all the warnings that have been broadcast over the past nearly 20 years. It seems to me that the public is indeed well informed, and that perhaps they have simply decided that they wish to make their own decision. I would prefer to have these millions of dollars spent on good research, and not on more publicity campaigns.

Again, I respectfully suggest that Congress should encourage good research; I have been advocating this for 30 years. If we had received adequate funding of good research years ago, then many of the questions raised today might have been answered. Science, not publicity campaigns, will solve our problems.
May 10, 1983

John P. East
United States Senator
United States Senate
Committee on Labor and Human Resources
Washington, DC 20510

Dear Senator,

In response to your letter of April, 1983, I am sending you a note regarding my work on cigarette smoking by pregnant women regarding bill S. 1929, with reference to bill S. 772.

I hope the information is helpful to you and the other members of your Senate Committee.

Yours sincerely,

Ber J. van den Berg, MD
Director, Child Health and Development Studies

Enclosure
May 10, 1983

CIGARETTE SMOKING BY PREGNANT WOMEN

A note regarding S. 772 - the "Smoking Prevention Education Act of 1983"

My name is Bea J. van den Berg. Currently, I am director of the Child Health and Development Studies, a research unit of the School of Public Health of the University of California at Berkeley.

I received my medical degree in the Netherlands and in 1965 I joined the research staff of the Child Health and Development Studies which was designed and directed by the late Dr. J. Yerushalmy, professor of Biostatistics at the University of California at Berkeley. My curriculum vitae accompanies my statement.

I am writing to express my concern about the statements made in S. 772 regarding an increased risk of miscarriage, stillbirth, premature birth, and child weight...
deficiencies in pregnant women who smoke.

For many years I have been involved in research regarding pregnancy and pregnancy outcome. The results of that research do not support the statements in St. 7/12.

Our studies were among the very first to report the lower birthweight of babies born to smoking mothers as compared to that of non-smoking mothers. However, our studies did not indicate an increased risk of abortion and stillbirths among smoking pregnant women.

For almost two decades, at the Child Health and Development Studies, we have studied the pregnancy outcome of women who smoked cigarettes during pregnancy, in comparison to women who never smoked or who stopped smoking before or early in their pregnancy. These studies were based on interviews, early in their pregnancies, of some 15,000 women who were members of Kaiser Foundation Health Plan, and who enrolled in the Child Health and Development Studies. Extensive information was obtained from the medical records of the
mothers during their pregnancy and delivery and from the medical records of the children from birth to at least age five.

Our studies are prospective and observational; prospective because the smoking data were assembled before the outcome of the pregnancy was known. This approach avoids a possible recall bias that might occur when the mothers are interviewed after the termination of the pregnancy. An unfavorable pregnancy outcome might affect the mother's recall of any event that occurred during her pregnancy, including smoking habits. Our studies are observational and not experimental; this signifies that the women themselves, and not the researcher, decided whether or not they would smoke cigarettes. The women in these separate groups might be different, not only in their smoking habits, but also in various other known or unknown characteristics. This methodological problem, coined by Dr. J. Yerushalmy as the problem of "self-selection," has to be taken into consideration...
If we compare the pregnancy outcome of women who do smoke with the pregnancy outcome of women who do not smoke.

I would like to summarize the results of our studies to date that relate to the statements concerning pregnancy in the S. 732.

On birthweight. As early as 1962, Dr. A. A. and others reported an increased proportion of low birthweight infants among the offspring of smoking pregnant women as compared with the offspring of non-smoking pregnant women. The observed association may indicate a cause-and-effect relationship, but it may also be influenced by differences between smokers and non-smokers in characteristics other than smoking. A later study indeed showed that a variety of other maternal characteristics, such as socio-economic status, maternal age, and ethnic background, influence the association between smoking during pregnancy and birthweight of offspring. Controlling for a number of these variables reduces the crude
difference in the birthweight of the offspring of both groups of women.

Yerushalmy's later work on maternal smoking was aimed at developing methodologies and designing studies to test the causal hypothesis against the alternative hypothesis that the increased incidence of low birthweight is due to the differences between smoking and non-smoking pregnant women. One such study evaluated the frequency of low birthweight among the offspring of mothers who began smoking after the birth of the infants. It was found that women who subsequently became smokers also had a high incidence of low birthweight babies during the period before they started to smoke. This finding cannot be explained by the causal hypothesis and underscores the need for a larger study to confirm or refute this finding.

On spontaneous abortion and stillbirth. Our statistical study, comparing for each week of gestation, the risk of fetal death for smoking and non-smoking pregnant women, failed to find a difference between the two groups. These
results, based on the pregnancy outcome of 15,000 pregnant women, provide evidence that smoking during pregnancy does not raise the risk of spontaneous abortion and stillbirth.

We recently published the results of our evaluation of the incidence of congenital anomalies (or birth defects) among the 14,735 children born to women who never smoked, to women who were past smokers or to women who smoked during pregnancy. The children were offspring of mothers enrolled in the Child Health and Development Studies. Our data showed there was no difference in the incidence of severe congenital anomalies among children of smoking women and children of women who never smoked. We also looked at the incidence of non-severe congenital anomalies. We found that, while the incidence of these non-severe birth defects among children of heavy smokers (more than 20 cigarettes per day) was slightly more than among children of non-smokers, the incidence among children of light smokers (less than 20 cigarettes per day) was equal to that of the children of non-smokers.
Recently, my colleagues have been working with data from the Child Health and Development Studies, focusing on the problem of self-selection that may cause bias in observational studies relating cigarette smoking to status of health. Utilizing longitudinal data, these studies indicate that adolescents who smoked cigarettes differed significantly from their non-smoking peers in many aspects of family background at the time of their birth, as well as in psychosocial characteristics assessed about 5 years before they started to smoke. These observations indicate that associations between smoking and health problems need to be controlled for preexisting differences between smokers and non-smokers.

In conclusion, our data do not support the statements in S. 772 regarding smoking and pregnancy.
John P. East
United States Senator
UNITED STATES SENATE
Committee on Labor and
Human Resources
Washington, D.C. 20510

Dear Senator East:

I have and thank you for your letter to me of April 21, 1983 and your request for my comments regarding the upcoming bill S. 171. My statement concerning the "Comprehensive Smoking Prevention Education Act of 1981" as appended and please feel free to use it in any manner you deem appropriate.

If I may be of further assistance, please do not hesitate to contact me.

Sincerely,

Rodger L. Bick, M.D.

Enc.

Encl.
STATEMENT

RODGER L. BICK, M.D.

My name is Rodger L. Bick. I am the Medical Director of San Joaquin Hematology Oncology Medical Group, and Director of Hematology Oncology at San Joaquin Community Hospital. I am also an Assistant Professor of Medicine in Hematology and Medical Oncology at the School of Medicine of UCLA Center for the Health Sciences in Los Angeles. In addition, I hold adjunct teaching positions at Wayne State University, Specialized Center for Thrombosis Research (Associate Professor) and Am Associate Professor of Allied Health Professions, California State College, Bakersfield.

I received my Medical Degree from the University of California at Irvine School of Medicine in 1970 and subsequently obtained Internal Medicine, Hematology Oncology, and Coagulation Fellowships. In 1973 and 1974, I was the Director of the Hemostasis/Thrombosis Research Laboratory and Chief of Hematology/Medical Oncology and Director of Medical Education at Kern County General Hospital in Bakersfield, California. From 1974 until 1977 I worked for the Bay Area Hematology Oncology Medical Group and Bay Area Hematology Oncology Clinical and Research Laboratories in Santa Monica, California.

I am a member of numerous Professional societies, including the International Society on Thrombosis and Haemostasis, the American Society of Hematology, National Director of Coagulation Workshops, Commission on Continuing Education of the American Society of Clinical Pathologists, and serve as editor for several journals in my field. In addition, I have written over 100 published articles on various aspects of the field of hematology and am currently President of the American Society of Coagulationists.

I have long been interested in the subject of blood coagulation and its possible relationships to heart attacks and cardiopulmonary disease in general. As I pointed out in previous statements, reports have appeared in the literature describing alleged isolation of biological properties of certain brown pigments from cured tobacco leaf, cigarette smoke condensates, and saline extracts of cigarette smoke. These pigments have generally been referred to as "tobacco glycoprotein" (TGP). Becker and coworkers have reported that TGP produces allergic skin reactions, induces the formation of antibodies, and activates early clotting factors. Based upon these rather simplistic experiments, with respect to early blood clotting factors, Becker and coworkers have drawn far reaching conclusions that TGP therefore must also activate the remainder of the clotting system, clot lysis system, and complement system. However, there is no evidence for this sequence of events in either the Becker articles or in subsequent, more
careful work. In spite of this lack of evidence, these authors have hypo-
thesized that these possible sequential pathological events represent a
pathophysiological link between cigarette smoking and cardiopulmonary
disease which has been reported to occur statistically.

To investigate more carefully these claims by Becker, et al., our group,
in collaboration with Dr. Stedman, extracted TGP by the previous published
methods of Becker and coworkers. Our group found that the final product
obtained by the isolation procedure of Becker contains and consists primarily
of a contaminant that is introduced by the methods of separation and appears
to be a side product of the photo-polymerization reaction used to form the
gels for separation. (Relatives of this contaminant, polymethylmethacrylates,
are widely used in orthopedic and dental surgery and traces of these compounds
have been implicated in allergic reactions and clot formation in man.) When
we subjected the contaminant to the aforementioned coagulation, clot lysing,
complement, and kallikrein generation assay systems, we found that the contaminant,
complexed with small amounts of an early coagulation factor, Factor XII,
giving the superficial and completely erroneous impression that activation
was occurring. However, it was found to have no activity on thrombogenic
assays currently used by the Bureau of Biologies for detecting activation
of the blood clotting system. Nor was there any activation of numerous of
the early coagulation factors tested. Likewise, there was no activation of
the fibrinolytic system; no effect on the complement system, and no effect
on the kallikrein generating system. Further, we found that there was no effect
of this contaminant on the normal clot inhibiting proteins found in human
blood.

Our studies strongly suggest that TGP isolated by the general published
procedure of Becker and coworkers consists of a contaminant which is intro-
duced during the separation process and contributes to, or more likely, is
entirely responsible for the biological activity as noted by Becker and
coworkers. The presence or absence of a substance in tobacco smoke with
biological properties supposedly affecting the coagulation, clot lysing,
complement, or kallikrein system remains controversial and certainly unProven.
It is obviously of importance to examine the significance of any of
the tobacco glycoprotein; however, a good deal of controversy does exist and
it has been impossible to duplicate previous findings. In one instance
it appears that erroneous and superficial observations have led to far
reaching hypotheses which can not be confirmed by carefully performed
studies. In this light, it should be noted that other subtle changes in
the blood clotting system, such as the noting of circulating platelet
aggregates (platelet clumps) and increased platelet aggregation (platelet
stickiness) has been noted in cigarette smokers. However, this work also
remains quite controversial and many authors have been unable to duplicate
these findings as well. Additionally, in considering the above minor changes
reported by some, but not confirmed by others, with respect to the effect of
cigarette smoking on the blood clotting system it should be realized that rather profound and confirmed changes in the blood clotting system have been reported by numerous individuals with respect to the effect of caffeine, aspirin, and glyceryl guaiacolate (common cough syrup). Thus, the meaning of these findings is, at best, completely unclear. In addition, it should be noted that there is an inverse correlation between smoking and venous thrombosis (blood clots in the legs), i.e., patients who smoke have a lower incidence of blood clots in the legs than non-smokers. Thus, the implication of all of the above findings remains totally unclear at best if, indeed, there is any meaning to these findings.

Again, this author's plea would be for adherence to confirmed scientific fact, rather than emotion, in attempting to study and delineate the biological effects, if any, of cigarette smoking on the blood coagulation system, complement system, clot lysis system, or kinin system. Our own research, extensive review of the scientific literature, and the work of others have led me to a conclusion that the effects of cigarette smoking, with respect to changes in the clotting system and a possible correlation between heart attacks or cardiopulmonary disease remains totally open to question and controversial.
The CHAIRMAN. Would the Senator yield to me?

Senator EAST. Yes.

The CHAIRMAN. I have been very concerned because I have had members of the Tobacco Institute come to me and indicate that they feel that they are being pushed a little fast on this. Now we have had really 4 years or more of these considerations.

Senator Dodd has indicated to me that he would like more time to study this. He has not been on the committee. He and Senator Matsunaga are the only two members who are on the committee now who were not there in the past 2 years.

Senator QUAYLE. And Senator Grassley.

The CHAIRMAN. And Senator Grassley, that is right.

It is a complex issue. I do not want anybody to feel like they are being shoved too hard. I see that frankly you have completely diverse testimony. We have those here today who basically find no real causal connection between smoking and many of the diseases that last week we heard testimony linking them to smoking from some of the leading doctors in the world in these areas. That is not to denigrate the excellent backgrounds of our witnesses here today.

I am aware that all of you have excellent backgrounds.

There is a dispute here. There is a real concern. There is, I think, a desire to get to the bottom of this. I would like to allow our newer Senators an opportunity to look at this record.

It has been suggested that we have a markup next Wednesday rather than Friday. My personal preference would be not to do it next Wednesday, but to put it off until a week from next Wednesday, so that each side will have every opportunity to make their case to members of this committee.

Would you consider that fair, Mr. Judge?

Mr. JUDGE. I think it is very fair, Senator.

The CHAIRMAN. I think I have been fair right down the line with you on these matters. We have a differing perspective, a differing viewpoint, but I want to be fair.

Representing your industry, if I put this off until about—what is that, the 15th? If I put it off until June 15, would you consider that fair?

Mr. JUDGE. Yes, sir, I think you are being responsive to—

The CHAIRMAN. Will that give you adequate time to meet with members of the committee and express your concern?

Mr. JUDGE. Yes, sir, and we appreciate it.

The CHAIRMAN. Well, then, that is what we will do. I know that will not bother the Senator from North Carolina if we put this off, and he has advocated, I think very well and very hard, for the constituents that he represents.

What I want to do is have this matter fully examined, the best we can, and I want to have both sides feel that they have had ample opportunity to present their cases. Then I would like to have the markup on—I think it is the 15th, a week from Wednesday. That gives every member of this committee notice—

Mr. JUDGE. That is May 25, Senator.

The CHAIRMAN. Would that be May 25? I guess that is what it would be, May 25. Whenever that is, a week from Wednesday,
then, we will just set it as the markup of the bill and we will go to
work on it at that time.

Senator DODD. Mr. Chairman, thank you.

The CHAIRMAN. Thank you, Senator. I think that is a reasonable
request, and we will make that decision and get it out to all mem-
bers of the committee.

Go ahead, Senator East. I am sorry to interrupt.

Senator EAST. Mr. Chairman, I appreciate your consideration of
that concern, that we make sure we have the full report available
and adequate opportunity for all Senators on this committee to be
able to look at the matter very carefully.

I would make an observation or two, realizing the great time con-
straints under which we work.

I think the testimony of today and the testimony of our last ses-

sion, at the hearing, does show that there are bona fide members of
the medical community as well as the scientific community who
differ over this subject. I think it is certainly one thing I would
hope we could get some unanimous consent on.

When you get distinguished scientists or physicians on several
sides—two sides makes it too simplistic an issue—it does suggest it
is complex, it is intricate; and it needs to be carefully studied and
reflected upon.

I appreciate the chairman's sensitivity to that and the willing-

ness to delay additionally here the markup time, and also to make
sure we have time to issue the report, so that Senators as well as
all interested persons can look at it and draw their own conclu-
sions and, if you will, express them accordingly, either to individu-
al Senators or publicly, or however they wish to go about it.

I would like, in addition to one or two specific questions I would
like to ask for the record, I would like to underscore, one, that
point, which I think we have all agreed upon. There are very
strong differences of opinion among very respected members of the
scientific community. Hence, to me that suggests a caution, 'go-slow
sign, as to precisely what we do and do not know about the relative
health risks of smoking. It is not to suggest we do not know any-
thing. It is to caution against claiming we know more than we do,
and perhaps somewhere in there's the truth of the matter.

I would submit as a matter of science, not to get overly philo-
sophical about it, the discovery of knowledge and new informa-
tion as incremental and evolutionary. I fear in the emotional is-

sue of this kind there may be a tendency to simply rise up and, by edict,
proclaim that this, in fact, is the truth; it has been discovered;
there are no ands, ifs, or buts about it; period; that ends up, and
let's get on with the legislation. I am uncomfortable with that. I do
not wish it to sound self-serving but I am uncomfortable with it. I
do not think it is a good scientific attitude.

On the basis of testimony I have heard this morning and last
week—and I am a layman in this area; I will concede that—there
are strong differences of opinion, and I would like to see those dif-
ferences expressed and, again, I am grateful for the chairman's de-
laying the time of markup.

Second, just to try to maintain perspective on this, whatever the
relative health merits of smoking as a scientific question, as a
person serving in a legislative branch where we draft legislation, I
am concerned that we remind ourselves that this legislation pur-
ports itself only to be informational. It does not purport to prohibit.
It does not purport to overregulate or to be punitive.

I would hope that in looking at it we would remember that I do
think, though that may not be the intention, it does tend clearly to
overregulate. It does tend, though maybe unwittingly, to have puni-
tive characteristics to it.

In short, instead of representing a bill rooted in genuine scientif-
ic understanding and founding, it becomes somewhat of a desire-
ful goal on the part of certain groups of interests. Now they are
entitled to that and to express it and to press for legislation to that
end, but certainly those of us who do see strong scientific differ-
ces of opinion are equally free, and that is what we are doing, is
suggesting that any legislation we pass reflects the very strong cau-
sing signs we have been getting from testimony, that legislation not
be to punitive, that it not be overregulatory.

I repeat, the bill itself, if you will read it with care and give it
some careful textual analysis, probably the word that crops up the
most is "inform." The proponents of it are clearly disclaiming any idea that it is
prohibitive in tone. Well, to me, the evidence is rather compell-
ing, not only statistically but in terms of candidly commonsense
and conventional wisdom, and I would even say judicial notice, one
could almost take it.

The public generally is powerfully and strongly aware that there
are potential negatives to excessive smoking. I defy anyone to say
you do not think that is a widespread understanding among
normal thinking people. To suggest there is a low level of informa-
tion on that and, hence, the need to increase the-decibel level of it,
I submit the record will not bear it out. It is probably the highest
one on the decibel level, perhaps more so than the problems associ-
ated with drinking and alcohol-related deaths in automobiles or the
excessive use of fat in the diet, or seat belt, or salt, or sugar, and
so it goes.

I do not wish to anguish over it too much publicly, but I have
trouble convincing me just in my own commonsense examination
of this that one can sit with straight face and say that the public
seems to be unaware that there could be health risks to excessive
smoking. I simply do not accept that.

Therefore, I would hope that whatever final measure that this
committee or the Senate or the Congress may come up with in due
course, after fair hearing, will be based upon the idea, one, of a le-
gitimate understanding of where we are scientifically with it, and
it will reflect an informational tone, not an overregulatory or a pu-
nitive tone to it. I think it would be unfair to a very honorable in-
dustry, and I think it would set a very bad precedent for other in-
dustries in this country, where something takes on a very strong
eotional character and we no longer think terribly rationally
about it, we simply emote about it and pass very heavy-handed leg-
islation which ultimately we come back to regret.

I appreciate the indulgence of my colleagues in allowing me to
say that, but I would like for the record to show that is where I
stand on this matter. I am not trying to unduly obstruct or fore-
close my colleagues’ making their own considered judgment over this matter after fair hearing and understanding of the matter.

I know the Senator from Indiana has been extremely patient this morning. What I would like to do at this point is yield to him and let him pursue his line of questioning. I think I have used up at least my allotted 10 or 15 minutes, whatever rule we are operating under.

Senator Quayle?

Senator QUAYLE: Thank you very much, Senator East.

The hearings we had, I believe it was last week—Dr. Brandt, in response to a line of questioning that I pursued, indicated that a preferable approach to getting a resolution on a stronger label requirement would be to get an agreement between HHS and the tobacco industry and the people that are affected by this. A further line of questioning produced the response that there had not been any attempt by HHS to get this kind of agreement, no specific contact with the industry, and I presume no contact by the industry to HHS. I wonder if, in fact, Mr. Judge, if you may be willing to tell us whether you might—first of all, would you— I guess my question would be, which would be a preferable route if, in fact, there would be a consensus in the Congress and a consensus within the executive branch, HHS, that there should be a single—at least a single stronger labeling warning on cigarettes, as far as sitting down and discussing that particular issue with HHS? What would be your response to that?

Mr. JUDGE. Senator, we feel that agreement, open discussion, and working with the legislative branch is much more desirable than additional regulation. We voluntarily met with Dr. Brandt on the ingredients matter and reached an agreement with him which he has praised before this committee and before the House.

We would be completely willing to sit down and discuss this issue with Dr. Brandt, but I would be less than forthcoming if I did not tell you, Senator, that we are starting from a great distance apart, but we would be happy to sit down with him at any time and have an open, honest, forthcoming discussion.

Senator QUAYLE. I wonder why there has not been any discussion along this line. Is it because you are so far apart that both sides basically feel that there is no useful purpose in sitting down and discussing this and, therefore, it is going to come back into the laps of the legislative branch?

Mr. Judge. No, sir, I did not mean to say that. I do not think you could expect us to have opened a negotiation with HHS and walk into Dr. Brandt and say, “Let’s talk about a tougher warning notice.” That does not seem—it is just not human nature. However, we would be delighted to sit and discuss it with him.

I am not saying that our heels are dug in. I am not saying that at all. We did work out an agreement on the ingredients issue, and we are perfectly willing to sit down and discuss this.

Senator QUAYLE. Are there other voluntary agreements that you can tell us that you have worked out in the past with HHS or HEW or any other Government agencies?

Mr. Judge. Yes, sir, we have a long list of voluntary agreements that we have reached. While it was ultimately not voluntary, the industry volunteered to get off the electronic media back in 1969.
and subsequently it was legislated because we had an antitrust problem with the networks unless it was legislated. However, that was a voluntary effort.

Earlier than that, in 1954, to meet public demand, we began to advertise low tar and nicotine, and the Federal Trade Commission came in and said, "Don't do that any more." We voluntarily have discontinued advertising and promotional activities among youth and on campus newspapers and sampling near schools and sampling youth. We have a voluntary sampling program.

We worked with the FTC to develop a tar and nicotine testing method voluntarily. A number of things were done voluntarily relating to our advertising back in 1963 with the cigarette advertising code.

We worked with the FTC on the disclosure of tar and nicotine in our advertising.

Senator Quayle. That is enough information to—

Mr. Judge. The history of our negotiating with either branches of the Government or the legislative branch—

Senator Quayle. The reason I ask that is that I just wanted to get a feeling for the ability to reach a voluntary agreement.

I certainly want to commend the chairman in his desire to see fairness and fullness in the record. There are some disputable issues, and I think he always has been very fair, I know to this Senator, and very fair to holding hearings, whether he agrees or disagrees.

The Chairman. Thank you.

Senator Quayle. I think that is one of the true hallmarks of the way that you have handled this committee, and you certainly are to be commended.

There are about three major issues. We have this interagency committee, the additional requirements on the additives, and then the multilabeling. They are very substantive issues. It is not one that we pass over lightly.

I know you put a lot of time into your bill, and I think by putting it off until June 15 it puts everybody on notice to take a longer look at this.

You were not here, Mr. Chairman, when I had my questions with Dr. Brandt. You were at the White House. However, he indicated to us that he felt that the preference would be to try to enter into an agreement.

I do not know whether that can be done or not, but I would hope that Dr. Brandt and HHS would take seriously that testimony which indicated to me that they would be willing and wanted to sit down and discuss a single stronger warning label. The representatives today here indicated that they would certainly be willing to sit down and to discuss this.

Obviously, when you go into negotiations, we do not know what the outcome will be, but I certainly think that that may be helpful, and it might even be of particular interest to all of those who are interested in this issue.

The Chairman. Would the Senator yield on that point?

Senator Quayle. Yes.

The Chairman. I would just make a point to you. I do not have any great desire to regulate the cigarette industry. As you all
know. I have fought for free enterprise principles ever since I have been here. Therefore, I do not have any desire to see your industry overregulated.

On the other hand, in spite of the testimony today, I feel that the medical testimony that we have had in the past is pretty conclusive. I do feel that this is, as the doctors have said, the No. 1 preventable health problem in America. If we could get people to either refrain from smoking or at least reduce the levels of smoking, we would solve a great number of health problems in America.

It is apparent that you, Mr. Judge, and your witnesses do not particularly agree with that. I respect your right not to.

The best way we can come up with to get people not to smoke, or at least to consider the ill effects of smoking, is, of course, warning labels. They do work. We had testimony last week indicating that warning labels do work.

The extent, Dr. Blackwell, that people do know that smoking may be deleterious to their health, is probably because of the Surgeon General's report and the warning labels on these cigarette packages.

If we could come up with a way of resolving this that would be agreed to by both sides, I would be very happy. We do not have to have a statute here. On the other hand, if we cannot come up with a way, then I intend to have a statute that would require these alternating labels. I believe that we have a good chance of passing that through the Congress.

I am very interested if you want to negotiate with Ed Brandt and other people down at HHS and elsewhere, and hopefully with this committee. We will be very interested in working with you to see if some amicable resolution can occur that will be in the best interest of the people of this country and that will save taxpayers dollars, because we spend billions and billions of dollars in health programs throughout this country, almost $400 billion a year next year. That is a lot of money. According to the doctors, a good portion of that, a hefty percentage of that, comes from the ill effects of smoking, from diseases caused by smoking.

I am interested in that, and I hope you will keep us informed, if you can negotiate with HHS. However, what HHS decides to do is not going to determine whether or not we decide to go ahead with this bill. I think it is very important that we be part of those negotiations. It is not crucial that you ask us, but it is crucial as to whether or not we have a bill.

Mr. Judge, I would imagine, Senator, that Dr. Brandt would—

The Chairman. He would run it by us, I am sure. He would keep us advised, and I suspect you would, also.

Mr. Judge. I would like to point out that if you are having a markup in 10 days, I do not think that 10 days is adequate time for us to try to reach an agreement.

The Chairman. It will be June 15, so that gives you enough time—

Senator Quayle. June 15 is a month's time, and the reason I brought this up—

Mr. Judge. I am sorry, I thought he said a week from Wednesday.

The Chairman. I did say a week from Wednesday, but I forgot that we are not here. I was trying to give you time.
Mr. Judge, Thank you, Senator.

The CHAIRMAN. We will set it for June 15, which gives you a full month. You have had the past 2 years, too. Therefore, it is not as though it is something that is new or something that has just come on the surface.

I think throughout this process I have tried to be fair with you and I have tried—I have shown continuously that I would like to amicably resolve this rather than have the constant confrontations that we have had. On the other hand, I am not afraid of confrontations. That is what this business is all about, and I have been beaten up by the best. I can say that, too.

Mr. Judge. So have we, Senator. [Laughter.]

The CHAIRMAN. I suspect you have.

Go ahead, Senator Quayle.

Senator QUAYLE. Being the chairman of this committee, Mr. Judge, the Senator from Utah has in some respects an unenviable position because it seems as though on every issue there is a divergence of opinions. In the 2½ years that I have served on this committee, the one thing that he has been able to do time and time again is to bring these parties together and get agreement, whether it was like the child abuse—I know we were working with the Job Training Partnership Act last year, we are going to be working with health benefits this year and what we are going to do for the unemployed.

Again, you are going to be right in the center, Mr. Chairman, in trying to bring divergent factions together.

The reason I brought this up, Mr. Chairman, was because you were absent when I had that line of questioning with Dr. Wendt. What surprised me was Dr. Brandt’s first statement, that he said he would prefer this, and the second statement, that they had not even approached the tobacco industry to see if they would be willing to sit down.

I think with your expression of interest, and mine, too, that we ought to convey to Dr. Brandt to try to see if we can work something out and that may be agreeable with you and other people in the congressional branch. I would imagine that by June the 15th there may be some process that may be established. It is sort of like the Middle East negotiations quite frankly, in some respects. These sides do not agree on things, but I think it is important. If we could establish maybe some sort of a process and timing—I know how you have worked in the past—that this would certainly be of interest to you.

The CHAIRMAN. It sure would.

Would you yield on that?

Senator QUAYLE. Yes.

The CHAIRMAN. I am interested. I have told you representatives from the Tobacco Institute continuously that I have an open-door policy to you. I have told you that I am concerned about health. I am concerned about the deleterious effects of smoking on the health of human beings in not only this country, but worldwide.

I am concerned about the cost of health. I am concerned about health prevention efforts. To me the most important health issue in this country is prevention, not whether we are going to spend hundreds and hundreds of billions of dollars in medicaid and medi
care to solve problems that do not have to exist, but whether we can prevent a lot of those problems from ever occurring to begin with and save a lot of health costs that way. I think it is one of the best ways of having cost containment and cost control in the health.

Virtually every witness, except the witnesses today, has indicated that smoking is one of the major costs of medical care and treatment in this country because there are so many problems that come from smoking. I am not a scientist but I believe it to be so from listening to the testimony that I have heard in the past and listening to the testimony today.

I think if we can voluntarily resolve this so that members of this committee are happy, HHS is happy, and we have people within the National Institutes happy, that would be a step in the right direction. If it is less suppression to you, that would make me very happy, because I do not want to regulate any business except in very extreme cases where there is no other alternative than to regulate a business. I personally do not believe in it.

However, this case is the lightest form of regulation we can have, it basically requires labeling, telling people what may happen from smoking cigarettes.

What I do not want to have happen is on June 13, 14, and 15, or any date between now and the June 15, is to have people coming to me saying, "We need more time." I want to see a real effort put forward, and I will put the effort forward with you. In other words, I will open those doors for you down there, if that is what you need, or we will on this committee. We will put the effort forth, and we will negotiate, and we will see what can be done to resolve this in a manner that is more satisfactory to your industry.

Mr. Judge, we will make every effort to meet our deadline. If nothing happens by that time, I assure you it will not because of lack of trying on our part.

The Chairman. All right. Now if we cannot put it together, I understand that you will have to fight this bill. I do understand that I accept that. I respect you for wanting to do it and for doing it. What I do not want is for you to say that you have not had enough time, because I think we have been very courteous to you and, I think, fair to you throughout this process. I intend to be.

Mr. Judge. Senator, I have thanked you once, I will thank you again. I appreciate it very much.

The Chairman. OK. Well, we appreciate you, too.

We will get together and see what can be done. If this cannot be done, then I am going to push to see if we can have a bill that will help to resolve this problem once and for all.

Mr. Judge. I understand.

The Chairman. I think that may be the way to do it.

Senator Quayle?

Senator Quayle. I think you have made some good points, but I think right now, as far as getting the process moving, it is somewhat incumbent upon us and Dr. Brandt. As Mr. Judge aptly says, they would just as soon not enter into these negotiations. They would just as soon leave things alone. They are not going to go and voluntarily say, "Hey, let's do it." I think HHS really ought to
make that contact and say, "Hey, we want to sit down and discuss a stronger label."

Mr. JUDGE. We will be in touch with Dr. Brandt at 9 o'clock tomorrow morning.

The CHAIRMAN. That will be fine, and I will be in touch with him before then.

Senator QUAYLE. I think it would be very helpful if the chairman would try to make some contact with Dr. Brandt. This has come up at both hearings, so this is not going to be any news to Dr. Brandt, because he knew that we were going to pursue this. I think it would be very helpful as we try to get a process instituted on that.

I certainly thank the chairman.

I have two other quick questions. One, as you talk about a stronger labeling, I wonder if there might be also some discussion on more information and education for youth. I would presume that you would say that cigarette smoking is something that young people should not engage in, and that there might be some information that this is more of an adult custom or an adult usage. Although we do not have any specific laws as the distilled spirits and people with alcoholic beverages have—a lot of things are directed as not for young people, and also if you drink, don't drive, these kinds of messages.

I wonder if there might be some discussion with HHS in these other discussions on the youth aspect, more information on that. Could you enlighten us on what your feelings would be on that?

Mr. JUDGE. Senator, this industry has always stated that smoking is an adult habit. I mentioned earlier some of the self-regulation concerning sampling, concerning advertising in campus newspapers, in our advertising not to appeal to youth.

We have recently run a series of ads that I mentioned in my statement saying, "Do the cigarette companies want kids to smoke? No." We have copies of these ads.

We have done a number of things to prevent this, or to prevent our appealing to children.

I would say this about discussions with Dr. Brandt in this area, you have given us a pretty full plate to negotiate by June 15, if it is possible, a stronger warning notice. For us to accomplish more than that by June 15, I think would be an impossibility.

Senator QUAYLE. I am not trying to put on any deadline because that is not even before the committee. I am just saying that as an overall discussion area—and it might be part of the situation on what the ultimate conclusion may be. If you can agree on a label—you know, when you get into these negotiations, there are all sorts of tradeoffs and they might like to see some more public advertisements to dissuade the young people from smoking, since, as you say, it is an adult habit. I do not want to specifically include it, but I do not want to exclude it, either. I just bring it up as a point of reference and an interest that might be discussed in the broad parameters.

I know the chairman would be interested in that, and I think even the Senator from North Carolina.

Mr. JUDGE. Senator, if I may—

Senator QUAYLE. I am not going to say, "Give us an answer."

Mr. JUDGE. I understand.
Senator QUAYLE. I am throwing it out as one that is sensitive to it and concerned about it and just offer it at these hearings for public consumption and public discussion.

Mr. Judge. We would like to point out that one concern that we have—and Dr. Blackwell touched on this—that is the backlash effect. My experience, raising two children, is that they like to do exactly what you tell them not to do. Every time we look at advertising of this nature, that is our primary concern.

Maybe Dr. Blackwell would like to touch on that, but it is a very, very difficult thing to do.

Senator QUAYLE. I do not think it is necessary to get into all the details. I just throw it out—

Mr. Judge. Yes, sir. Thank you, Senator.

Senator QUAYLE. As one Senator who's concerned about it. It is not even really incorporated, except in a broad basis, in this bill. It mentions education.

I just think it is an issue that at least I am sensitive to.

Mr. Judge. So are we.

Senator QUAYLE. I know that you are sensitive to it, and I throw it out for, as I say, discussion purposes, not really to debate it but just to express a concern:

One final question, Mr. Chairman. Maybe you have already answered it. If so, fine; I will read it in the record.

Have you discussed the rationale and the reasons why England went back from multilabeling to a single label?

Mr. Judge. I have no factual information on it. I know the witnesses who testified last week did not have any. Our assumption is that it was impractical and did not work, because in that case the government had rotational warnings, three warnings, and this was a negotiated agreement to go back to one. I have to assume that the government did not give up three if they thought it was better than one. That is all I— I really do not have any factual information on it.

Senator QUAYLE. I do not know if there is any way we can get somewhat of a clarification on that, but I know the witnesses last week could not answer that question, either. I do not know if there is any way we could pursue it to find out what the thinking was on that negotiated agreement.

Mr. Judge. It is a recent occurrence, Senator, quite recent. I understand within a fortnight, and we will do our best to get you any information that we can on that subject.

Senator QUAYLE. I would think it would be very relevant to the discussion that we are having here, which I think it one of the substantive issues, whether you are going to have multilabeling or single labeling.

I thank the indulgence of the Chair and his very appropriate remarks.

The Chairman. Thank you.

We will leave the record open, inasmuch as we have some extra time now, to submit questions anyone on this committee would care to submit to you. We would appreciate your answers being made as expeditiously as possible.

Senator East, do you have any other comments?
Senator East. I would simply like to thank the chairman for his understanding of the complexity of this and the willingness to postpone any further action on this bill until there can be a full report and a greater opportunity for members of the committee to study it. That is the first observation I would make.

Second, I wish to thank the Senator from Indiana and also the chairman for their both underscoring here the value of discussion and dialog between the potentially affected industry and the appropriate Federal agency. I think the idea of voluntarism, of trying to discuss and dialog and reach some sort of reasonable conclusion is a good thing to encourage in any situation. I think it makes for sound political velocity, if you will, rather than the Government heavyhandedly imposing edicts on particular individuals or industries. This reflects what I am infinitely more comfortable with philosophically, and I know the chairman is historically with seeing if these things can be worked out in some constructive, positive way.

I wish to publicly thank him for his attitude and spirit on that. I do think Mr. Judge has responded in a very positive way to that, which is good. I think he raises a valid point that, even though the June 15 deadline is a very valuable and helpful one, and we deeply appreciate the chairman’s understanding there, if we look upon that as being a date that could yield up all mysteries and solutions on this problem, I do not know whether that would be possible. Maybe it would. I am not trying to prejudge it.

I would like for these discussions to be serious and positive and upbeat and try to get to the heart of what would be a good solution. On the other hand, I would simply like to state for the public record that I would not want this industry, or any other, to have to work under a threat that unless they produce in a certain fashion, they could expect some sort of punitive legislation, because obviously then that does erode away the idea of serious discourse and voluntarism, of trying to reach an accord.

I know the distinguished chairman does not mean it in that spirit. I think what he is saying—I do not mean to put words in his mouth, he is extremely articulate and eloquent—is that obviously Congress will always want to reserve the right, if voluntarism is not productive and effective, as they see it, then obviously they are going to reserve the right to move independently on their own. I do not quarrel with that observation.

However, I would like again to say for the record I hope that this dialog and discussion will be creative and productive and that we can reach a final solution to it, or at least one that will tide us over as much as any policy solution can for the near term, and continue to work in that spirit—the ultimate goal being, I would like to contend, that the American people be honestly and fairly apprised along the way of the relative health merits of smoking or not smoking, as we might do in any other area with problems affecting national public health.

Again, I thank the chairman for his attitude and approach. I think the session this morning was very constructive and positive. I would like for the record to show that, not that I need to say it. I think that the reading of the record would indicate that is the case.

The CHAIRMAN. Thank you, Senator East.
I am interested in stronger labeling. I am interested in voluntary approaches that will help our youth to understand the problems associated with tobacco smoking.

I will work with you. I will make sure, if you have any difficulties chatting with anybody at Health and Human Services, that I will help you with everything I have to be able to make the contacts and have the people meet with you that you need to meet with.

However, I would like to see it resolved that way. I have no secrets about that. It would be preferable to me to have you feel that the process has served you as well as my feeling the process will serve the people.

Let's see what can happen. The only thing I ask is that, come June 15, my colleagues show up and we resolve this matter one way or the other, if we cannot resolve it on an amicable voluntary basis.

Mr. Judge. We will do our best.

The Chairman. I want to thank you, each of you, for coming. We are grateful to have you before our committee. We will look forward to working with you in the future. Thanks so much.

Mr. Judge. Thank you, sir.

The Chairman. Our last panel consists of Mr. Eric Rubin, an attorney with Rubin, Winston & Dierks. He is counsel for the Outdoor Advertisers Association of America in Washington, D.C. Mr. Rubin is accompanied today by a great friend of mine, William K. Reagan, who is president of Outdoor Advertising, Inc., in Salt Lake City.

I am happy to welcome both of you before the committee.

These people really know how to put the pressure on a committee chairman, by bringing one of my best friends in here to chat with me.

Go ahead, Mr. Rubin.

STATEMENT OF M. ERIC RUBIN, ESQ., RUBIN, WINSTON & DIERKS, COUNSEL FOR THE OUTDOOR ADVERTISERS ASSOCIATION OF AMERICA, WASHINGTON, D.C., ACCOMPANIED BY WILLIAM K. REAGAN, PRESIDENT, OUTDOOR ADVERTISING, INC., SALT LAKE CITY, UTAH

Mr. Reagan. Mr. Chairman, other committee members. I appreciate the courtesy you have extended to me and to the Outdoor Advertising Association of America in light of the request that we may testify before this committee.

The Outdoor Advertising Association of America is concerned about the impact of this bill on advertising.

I am accompanied by Eric Rubin, as you have already recognized, who is counsel to the Outdoor Advertising Association of America. He will deal specifically with the concerns of the association.

Thank you.

The Chairman. Thank you, Mr. Reagan.

Mr. Rubin. Thank you, Mr. Chairman.

I will summarize my testimony and request that the text be accepted for the record.
Mr. RUBIN. Thank you, sir.

The OAAA is the trade association of the standardized outdoor advertising industry.

The OAAA is seriously concerned about S. 772 because its broad impact on cigarette advertising has not been recognized in these hearings. At the time S. 772 was introduced, the sponsors were careful to state that the bill was not intended to affect cigarette advertising regulation and that the predominant concerns which it addresses are public health issues.

It is true that this legislation does not contain specific language that would require a rotational warning system in cigarette advertising and, in fact, draws a distinction, a very major distinction, between package labeling and advertising. Nonetheless, enactment of S. 772 would have just that result. The legal machinery which makes this essentially inevitable is already in place and could be set in motion by the passage of this bill.

The basic restrictions on cigarette advertising, as Mr. Judge alluded to, are established in the 1972 Federal Trade Commission consent order with the six cigarette manufacturers. This FTC order requires that the Surgeon General's warning must appear in all cigarette advertising. Less than 2 years ago, the consent order was modified by further agreement of the parties in a series of U.S. district court consent judgments. These recent amendments altered the manner in which the warning is disseminated in certain media and clarified a number of ambiguities in the 1972 order.

The FTC cigarette consent order is not carved in stone. A specific legislative enactment is not required in order to change the current advertising restrictions. The FTC's procedural rules empower the Commission to unilaterally alter an order where the Commission finds such modification is required by a change of law, a change in fact, or the public interest. The FTC's authority in this regard is very broad. It has been held that the FTC even has the power to change a consent order over the objection of formerly consenting respondents. These procedures would become crucial if S. 772 were enacted.

Although the current restrictions on cigarette advertising are not themselves legislative, the FTC orders are intertwined with the provisions of the Federal Cigarette Labeling and Advertising Act, which are at issue in S. 772. The initial FTC complaint which gave rise to the 1972 consent order specifically alleged that the failure of cigarette advertising to incorporate the Surgeon General's warning "negates and overcomes any tendency or capacity which a warning on the cigarette package itself may have to impress on the public the dangers to health which accompany the smoking of * * * cigarettes." As a result, the FTC order specifically compels that the warning required on package labels by the Federal Labeling Act must also be incorporated in all advertising.

The Commission has already established the policy of requiring advertising disclosures paralleling the Labeling Act. The proposed amendment of that act to require a rotational warning system could be construed as a change of law and change of fact under the
FTC rules. This is all that would be required to support a modification of the consent order to include the rotational warning in advertising as well.

The congressional findings in section 2 of S. 772 provide further impetus for such action. The fifth congressional finding proposed in the bill includes a determination by the Congress that "present Federal, State, and private initiatives have been insufficient to convey the information" summarized in the four rotational warnings. Standing alone, this finding, this congressional finding, could be viewed as a summary determination by Congress that current advertising regulation restrictions are inadequate. Thus, even when stripped of specific advertising provisions, S. 772 is still an advertising bill.

Finally, it must be pointed out, as it has been earlier, that the FTC seems primed to impose the rotational system in cigarette advertising. Two years ago, the FTC issued a staff report to the Commission recommending that the consent order be replaced with a rotational system like that proposed in S. 772. Last September, the Director of the Federal Trade Commission's Bureau of Consumer Protection wrote directly to Senator Packwood reaffirming the FTC staff's report for legislation which would implement a rotational warning in advertising. S. 772 would make this a fait accompli.

Despite the FTC staff's view, there are serious questions as to the advisability, even the practicality, of instituting a rotational warning system in advertising. Some of those concerns were raised briefly this morning. They were extensively discussed in the House Commerce Committee earlier this year. The point was made already about the failure of rotational systems in the various European systems. In addition, a broad number of experts beyond just Dr. Blackwell have told the House committee that new detailed warnings are not going to increase public awareness when better than 90 percent of the American public already believes that cigarette smoking is a health hazard.

In addition, the OAAA, the Magazine Publishers Association, and the American Newspaper Publishers Association have all expressed the strong opinion that a Government-imposed rotational warning system in advertising would be unconstitutional.

These are important issues. They have not been explored by this committee because S. 772 has not been viewed as an advertising measure. Indeed, the Senate may not have an opportunity to review these separate advertising issues if this bill is reported out of committee based just on this hearing record. The impact of this bill on advertising has not been recognized to date, but it cannot be ignored.

Thank you.

[The prepared statement of Mr. Rubin follows:]

50
I am counsel to the Outdoor Advertising Association of America (OAAA). The OAAA is the trade association of the standardized outdoor advertising industry. I am also a part owner of two outdoor advertising businesses located in Lynchburg, Virginia and San Antonio, Texas. I am testifying before the Subcommittee today from that dual perspective. The OAAA appreciates this opportunity to testify regarding the restrictions on cigarette advertising that would result from the enactment of S.772 and to state its opposition to this bill.

The OAAA is seriously concerned about S.772 because its broad impact on cigarette advertising has not been recognized in these hearings. At the time S.772 was introduced, the sponsors were careful to state that the bill is not intended to affect cigarette advertising regulation and that the predominant concern which it addresses are public health issues. Senator Packwood stressed this point stating that the bill "retains the labelling provisions of S.1929, but drops the advertising provisions."

It is true that this legislation does not contain specific language that would require a rotational warning system in cigarette advertising. Nevertheless, enactment of S.772 would have just that result. The legal machinery which makes this inevitable is already in place and could be set in motion by the passage of this bill.
The basic restrictions on cigarette advertising are established by a 1972 Federal Trade Commission consent order with the six cigarette manufacturers. 1/ This FTC order requires that the Surgeon General's warning must appear in all cigarette advertising. The FTC order further prescribes in great detail the precise manner in which the warning must be displayed in each of the media that carries cigarette advertising. Less than two years ago, the consent order was modified by further agreement of the parties in a series of U.S. District Court Consent Judgments 2/. These recent amendments altered the manner in which the warning is disseminated in certain media and clarified a number of ambiguities in the 1972 order. For example, the format for the billboard disclosure of the Surgeon General's warning was completely revamped and substantially enlarged. The FTC's cigarette consent order is not carved in stone. A specific legislative enactment is not required in order to change the current advertising restrictions contained in the 1972 order, as amended. The FTC's procedural rules empower the Commission to unilaterally alter an order or set its provisions aside entirely where the Commission finds such modification is required by a change of law, fact, or the public interest 3/. The FTC's authority in this regard is very broad. It has been held that the Commission even has the power to change a consent order over the objection of the respondent 4/. These procedures would become crucial if S.72 were enacted.

---

1/ In the matter of Lorillard, et al., 80 F.T.C. 555 (1972).
3/ 16 CFR 3.72(b)
Although the current restrictions on cigarette advertising are not themselves legislative, the FTC orders are intertwined with the provisions of the Federal Cigarette Labelling & Advertising Act, which are at issue in S.772.

The initial FTC complaint which gave rise to the 1972 consent order specifically alleged that the failure of cigarette advertising to incorporate the Surgeon General’s warning “negates and overcomes any tendency or capacity which a warning on the cigarette package itself may have to impress on the public the dangers to health which accompany the smoking of...cigarettes.” As a result the FTC order specifically compels that the warning required on package labels by the Federal Labelling Act must also be incorporated in all advertising.

The Commission has already established the policy of requiring advertising disclosures which parallel the Labelling Act. The proposed amendment of that Act to require a rotational warning system could be construed as a change of law and change of fact under FTC rules. This is all that would be required to support a modification of the consent order to include the rotational warning in advertising.

The congressional findings in Section 2 of S.772 provide further impetus for such action. The fifth congressional finding proposed in the bill includes a determination by Congress that “present Federal, State and private initiatives have been insufficient to convey the information” summarized in the four rotational warnings. Standing alone this finding could be viewed as a summary determination by Congress that current advertising regulations are inadequate. Thus, even when stripped of specific advertising provisions, S.772 is still an advertising bill.

Finally, it must be pointed out that the FTC already seems primed to impose the rotational warning system in cigarette advertising. Two years ago, the FTC

staff issued an extensive report to the Commission recommending that the current consent order be replaced with a rotational system like that proposed in S.772. Last September, the Director of the FTC Bureau of Consumer Protection wrote directly to Senator Packwood reaffirming the FTC staff's support for a rotational warning system in advertising. S.772 would make this a fait accompli.

Despite the FTC staff's view, there are serious questions as to the advisability of instituting a rotational warning system in advertising. In recent hearings, the House Commerce Committee heard expert testimony that rotational warning systems have failed to affect cigarette consumption in every European country where they have been tried. England recently eliminated the rotation program entirely and returned to a single warning. A number of experts told the House Committee that new detailed warnings are not going to increase public awareness when better than 90% of the public already believe that cigarette smoking is a health hazard. In addition, the DAAA, the Magazine Publishers Association and the American Newspaper Publishers Association have all expressed their strong belief that a government-imposed rotational warning system in advertising would be unconstitutional. Attached to this testimony is the statement submitted to the House Committee by the APCA and a separate statement by the Magazine Publishers which detail the reasons such a measure would be unconstitutional under the First Amendment.

These are important issues. They have not been explored by this Committee because S.772 has not been viewed as an advertising measure. Indeed, the Senate will never have an opportunity to review these separate advertising issues if this bill is marked-up and reported out of Committee based on this hearing record. The impact of this bill on advertising has not been recognized in these hearings, but it cannot be ignored.
The CHAIRMAN. Thank you, Mr. Rubin.

John, do you have any questions?

Senator EAST. Just briefly, Mr. Chairman.

What you are contending, Mr. Rubin, is, that this legislation, whatever the relative merit of the public health problem involved, which you are not trying to resolve this morning—and I guess it is established no one can fully resolve it—that this legislation, at least from the standpoint of your industry, would have a very serious economic impact.

Mr. RUBIN. Yes, Senator. What I am saying basically is that there is an unresolved issue that—

The CHAIRMAN. That is applied to advertising—

Mr. RUBIN. I am sorry?

The CHAIRMAN. If this bill applies to advertising.

Mr. RUBIN. If it applies to advertising. What we are saying is that essentially there is a trip hammer that perhaps is unintended, but there is a trip hammer in this bill that will set off a series of consequences that are, shall we say, extralegislative?

Senator EAST. Therefore, it becomes a question not only of the impact; let us say, directly upon the tobacco industry as such, but it obviously has an impact upon other industries, and particularly in your case your industry. It takes on a character of business and commerce and trade activity advertising, et cetera.

Mr. Rubin. That is correct. We think there is also another very serious issue here, a very serious issue, as to the overall constitutionality of advertising restrictions. They come under a completely newly evolved number of Supreme Court cases. This could—the probabilities are—set this whole issue off without ever having been considered.

Senator EAST. Thank you, Mr. Chairman.

The CHAIRMAN. Thank you.

You have said that the FTC is likely to impose multiple warning requirements for advertising if this legislation is passed. I agree with you that the FTC may not—it may have too much power and it may exercise it injudiciously from time to time, and I think perhaps it does cause a great deal of unnecessary mischief with certain businesses in our society. However, I want to emphasize that this legislation has no requirements, as far as I know, affecting advertising. The power of the FTC is under the jurisdiction of the Commerce Committee.

How would you propose changing the legal machinery you have referred to so that the changes in labeling requirements for advertisers would not be coupled to cigarette package warnings?

Mr. Rubin. Well, I think it is—I do not want to try to dodge it. I think it is really a very technical issue because it is intertwined with a series of consent judgments, consent orders.

My concern, beyond that, is that if this bill went forward even with some provision like that, an advertising provision could easily be reinserted on the floor. Therefore, we have real concerns about that about putting it in and then the practicality of it.

The CHAIRMAN. Mr. Reagan, assuming the worst, assuming that Mr. Rubin is right in his worries about this, how would this particular bill affect your outdoor advertising business in Utah?
Mr. REAGAN. Mr. Chairman, it would not, to answer honestly, because I would not—

The CHAIRMAN. Let me make it a little broader. Would it affect outdoor advertisers across this country?

Mr. REAGAN. It would dramatically affect other—

The CHAIRMAN. You have been speaking not just for yourself but for the industry as a whole?

Mr. REAGAN. Yes. I have no interest in this at all financially because we do not display any tobacco products in the State of Utah on our boards. However, looking at it objectively as a member of this association, it would have a devastating effect on that industry—the thousands of employees that are involved and the thousands of companies, many of them very small companies. The typical ownership of outdoor advertising companies is small, owner-operator types of businesses, other than in the large metropolitan areas. It would have that impact. I do not know how it could not, objectively looking at it, without resolving this technical issue which I cannot pass—

The CHAIRMAN. I appreciate your honesty here today. Of course, I did not think it would affect you much in Utah, but you are saying it would affect, if it is construed the way Mr. Rubin is concerned it may be construed, that it would affect outdoor advertisers all over the country.

Mr. REAGAN. Yes.

The CHAIRMAN. Do you know approximately how much—or you could answer this, Mr. Rubin—what percentage of outdoor advertising is utilized by the tobacco industry?

Mr. RUBIN. I am just trying to remember. We testified as to that in the House. I think the number was about 16 percent, the national number. It is significant.

The CHAIRMAN. A significant percentage. That is what you are concerned about?

Mr. RUBIN. Yes, sir.

Mr. REAGAN. It is further significant, Mr. Chairman, because our type of business is a break-even business. You have to get to a certain level before you can succeed. To take 16 percent of the volume away from them would make most of them nonprofitable.

Mr. RUBIN. I might add that the issue on advertising, if you want to talk about the impact of this, is really not confined to outdoor advertising. In fact, I would say the outdoor advertising may be the smallest part of it. The impact on newspapers is much more substantial and the impact on magazines is much more substantial as well.

The CHAIRMAN. OK. I want to thank both of you for taking time to testify. These have been interesting hearings today. We will recess them until further notice, with the new markup scheduled for June 15.

Thanks so much.

Mr. REAGAN. Thank you, Senator.

Mr. RUBIN. Thank you.

The CHAIRMAN. At this point I order printed all statements of those who could not attend and other pertinent material submitted for the record.

[The material referred to follows:]
Statement by R. Harrison Rigdon, M.D.

I am Harrison Rigdon, M.D., a physician licensed in Georgia and Texas. I obtained my M.D. degree from Emory University in 1931 and my training in Pathology at Duke University in 1931 to 1935. I have been in the teaching department at the following Medical Schools: Vanderbilt University in Nashville, Tennessee, The University of Arkansas at Little Rock, and the Medical Branch of the University of Texas at Galveston. I was at Galveston from 1949 to 1973 when I retired. I am a member of the American Board of Pathology, the College of American Pathology and was certified in Forensic Pathology by the American Board of Pathology. I am listed as a pathologist in Marquis "Who’s Who, Inc." I am an anatomical pathologist and interested in human autopsies and experimental research. I have published 326 articles and one book.

In my opinion the purported "findings" of S.772 are not scientifically correct. It has not been scientifically established that smoking causes lung cancer or other diseases ascribed to it by S.772.

Some epidemiological studies have reported a statistical association between smoking and disease. Such studies, however, do not establish a causal relationship between smoking and diseases. Moreover, population studies are
for the most part based on vital statistics derived from death certificates.

At death, a certificate must be recorded by the physician giving the cause of death for our vital statistics records. Different diseases may produce similar clinical symptoms. Without special tests having been made, such as x-rays, histological study of a biopsy, or other specific chemical studies, the physician records the cause of death based only on the information he obtained clinically. The physician does the best he can in the care and treatment of a patient and at death he records what he then believes to be the cause of the patient's death. Studies, however, indicate that the clinician is frequently in error.

In 1963 I published a paper "Vital Statistics and the Frequency of Disease" in the Texas State Journal of Medicine, Vol. 59 p. 317-324. In this study, cases of people who died between 1945 and 1958 were studied to obtain the cause of death as recorded on the death certificates. The diagnosis of primary tumor as recorded on the death certificate and that given on the autopsy protocol were different in 71 (37 percent) of the 194 cases. In 38 cases the clinical diagnosis was different from that recorded on the death certificate.

In another study of 100 autopsy cases, also discussed in the 1963 paper, the diagnosis based on the autopsy and that
recorded on the death certificate were found to be the same in only 68 of the cases. In three cases a neoplasm was present as shown by the autopsy, however it was not recorded on the death certificate. In 32 cases the diagnosis as made from the autopsy was different from that recorded on the death certificate.

Some of the problems occurring in the coding of cases for vital statistics result from insufficient data on the death certificate. In other cases the classification of the neoplasm for vital statistics is different from that recorded on the death certificate. Further, cancer may develop elsewhere in the body and spread (metastasize) to the lung—such cases may be recorded on the death certificate as a lung cancer; without histologic study of the lung tumor it would not be known that this cancer was, for example, a primary lesion of the kidney, not the lung. These problems make it quite difficult to establish the true frequency of primary lung cancer. I concluded in 1963, and I still believe, that "statistical data obtained from vital statistics to show the frequency of a specific disease process should not be presented as 'authentic'.

In 1981, I published a paper entitled "Problems in a Statistical Study of Diseases Based on Death Certificates" in the Southern Medical Journal, Vol. 74 # 9, pp. 1104-1106. This
publication was based on a study of 5,787 registered death certificates from Morgan County, Georgia, occurring between 1927 and 1979. The cause of death as recorded on the death certificate was based primarily on a clinical diagnosis. Some death certificates cited a neoplasm but the site of origin was not given. In some a tumor was recorded but it was not coded and could not be included in vital statistics. Some of the death certificates were signed by a physician who was uncertain about the primary cause of death. In 25 of the 1500 cases obtained between 1952 and 1968 the race, sex or age were not recorded: 99 death certificates were signed by a coroner, 5 by a midwife, and one was not signed. A malignancy was present in 195 of the 1,287 deaths recorded from 1970 to 1979. In this group of 1,287 certificates, a coroner signed 240. My conclusion from this review of registered death certificates used for vital statistics by the State of Georgia and the U.S. Public Health Service was, and still is, that "such data as obtained from vital statistics would contribute no scientific data on the frequency of cancer."

Other investigators have expressed a similar opinion referable to vital statistics. In the British Medical Journal, 5563; p. 445-446, 1967 in an article "Accuracy of Mortality Statistics" it is said, "It is sometimes forgotten that the elaborate mortality statistics published annually by the Registrar-General depend for the most part on what medical practitioners write on death certificates."
In 1979, Anderson et al., in the Journal of the American Medical Association, 242, pp. 1056-1059, concluded the "many studies during the past 60 years have compared autopsy findings with clinical diagnosis. Discrepancies range from 6 to 68 percent, with a mean of 59 percent." The Surgeon General's Advisory Committee Report, DHHS, publication No. 1103. 1964, stated "the cause of death is at times difficult to establish accurately from clinical findings alone." Blot et al., at the National Cancer Institute in 1977, in Science 198, p. 51-53, said "the information obtained from death certificates may not accurately reflect incidence."

There is also clinical evidence that is inconsistent with the smoking causation hypothesis. For example, one basic problem that arises when one considers the inhalation of cigarette smoke as the cause of lung tumor is that the smoke spreads through the right and left lung. I am not familiar with any clinical or pathological study in which multiple tumors have occurred in the lungs of cigarette smokers. Furthermore, there are many people who smoke and don't develop a lung tumor.

It is my opinion that additional research based on scientific investigation is needed to determine the relation of cigarette smoking to disease. Such should be made since there
is a serious conflict of opinion referable to this problem. Many of the opinions are given by scientists who have studied these problems both clinically and experimentally over long periods of time without being able to arrive at a conclusion about cancer causation. We still do not know the mechanism by which cancers develop.

In my opinion, the purported "findings" of referable to cigarette smoking and health are scientifically unfounded.
The American Medical Association takes this opportunity to comment on S. 772. The bill states that its purpose is to "promote public health by improving public awareness of the health consequences of smoking and to increase the effectiveness of Federal health officials in investigating and communicating to the public necessary health information." The bill would accomplish this by replacing the current general health warning found on cigarette packages with four specific health warnings. One of the warnings would be required on every cigarette package. The warnings would be rotated among brands so that each brand would use all four...
warnings within a twelve-month period. S. 772 would also require that tar, nicotine, and carbon monoxide levels be disclosed on cigarette packages.

Cigarette manufacturers would be required to provide the Secretary of Health and Human Services with a list of each chemical additive used in the manufacture of its cigarettes. This information would be considered a trade secret and not disclosed to anyone outside the Department. The Secretary would be required to provide Congress with a complete list of the chemical additives used in the manufacture of cigarettes, a summary of research activities relating to such additives and findings on the health risks of such additives and any information about an additive the Secretary believes poses an additional health risk to cigarette smokers.

The AMA supports amendment of S. 772.

COMMENT

The United States Surgeon General stated in his 1981 report, The Health Consequences of Smoking: The Changing Cigarette (Surgeon General's Report), that "Cigarette smoking is the chief, single, avoidable cause of death in our society and the most important public health issue of our time."

A decision to smoke should be made with the knowledge that increased health risks are associated with smoking. For this reason the AMA is supportive of efforts to increase public awareness of the hazards.

The AMA has been involved in many efforts to increase the public's knowledge of the consequences of smoking. In 1978 the AMA published "Tobacco and Health," an account of the comprehensive research program on
warnings within a twelve-month period. S. 772 would also require that tar, nicotine and carbon monoxide levels be disclosed on cigarette packages.

Cigarette manufacturers would be required to provide the Secretary of Health and Human Services with a list of each chemical additive used in the manufacture of its cigarettes. This information would be considered a trade secret and not disclosed to anyone outside the Department. The Secretary would be required to provide Congress with a complete list of the chemical additives used in the manufacture of cigarettes, a summary of research activities relating to such additives and findings on the health risks of such additives and any information about an additive the Secretary believes poses an additional health risk to cigarette smokers.

The AMA supports enactment of S. 772.

COMMENTS

The United States Surgeon General stated in his 1981 report, The Health Consequences of Smoking: The Changing Cigarette (Surgeon General's Report), that "Cigarette smoking ... is the chief, single, avoidable cause of death in our society and the most important public health issue of our time."

A decision to smoke should be made with the knowledge that increased health risks are associated with smoking. For this reason the AMA is supportive of efforts to increase public awareness of the hazards.

The AMA has been involved in many efforts to increase the public's knowledge of the consequences of smoking. In 1978 the AMA published "Tobacco and Health," an account of the comprehensive research program on
smoking conducted by the AMA. A pamphlet called "Smoking: Facts You Should Know," a copy of which is attached, has been widely distributed along with two anti-smoking posters. Public service announcements on the hazards of smoking have been produced and distributed by the AMA. Physicians are urged to alert smokers to the risks associated with smoking.

We find it very disturbing if, as a 1981 FTC staff report alleges, approximately ten percent of the population still do not know that cigarette smoking is harmful.

Rotational Warnings

Believing a more detailed warning would better inform the public of the harm of smoking, the AMA House of Delegates, in June 1982, adopted a resolution reaffirming AMA support of the concept of explicit rotating warning labels on cigarette packages. The AMA believes S. 772 will increase public awareness of the specific harmful effects of smoking by providing more detailed warnings about the health risks of smoking. In our view, the warnings should also be required in cigarette advertisements.

The warnings provided for in this bill are appropriate. One of the warnings would be composed of three parts. The warning states that cigarette smoking 1) causes lung cancer and emphysema, 2) is a major cause of heart disease, and 3) is addictive and may result in death.

Reliable evidence links cigarette smoking to lung cancer and emphysema. In fact, cigarette smoking is the number one cause of lung cancer and emphysema in the United States. Some statistics suggest that smoking nearly doubles a person's risk of suffering a heart attack and that
one-third of all heart disease deaths are caused by smoking. These are significant health risks and should be considered by individuals who are considering smoking. The ANA believes such a warning will improve public awareness of these risks.

It should be pointed out that cigarette smoking is more correctly labeled as resulting in dependency rather than addiction. The National Drug Abuse Advisory Council has determined cigarette smoking causes a dependency. The distinction between addiction and dependence is that addiction causes an increasing need for the substance whereas a person suffering from a dependence may remain at the same level of intake for long periods of time.

The second warning indicates some of the risks to pregnant women. This warning states that cigarette smoking by pregnant women may result in miscarriage, premature birth or birth weight deficiencies. Cigarette smoking during pregnancy has been shown to have adverse effects on the mother, the fetus and the newborn infant. While the research regarding the effects of smoking while pregnant is not as conclusive as the research of lung cancer, emphysema and heart disease, the research does indicate a cause for concern.

It appears that many women may not be aware of the potential risks of smoking during pregnancy, and in our view this warning could increase public awareness. The ANA believes this warning is appropriate because it will alert pregnant smokers to the potential risks. After being informed of the potential risks, pregnant women can seek additional information from their physician and make a decision regarding their smoking during pregnancy.
The third warning states that “No Matter how long you have smoked QUITTING NOW greatly reduces the risks to your health!” The research clearly shows the longer one smokes the greater the risks to health. The literature also demonstrates that no matter how long the individual has smoked, the health risks are reduced after quitting. This message is very important to smokers, particularly those who have smoked a long time, because it indicates that they will benefit if they stop smoking.

The fourth warning provides “TEENAGERS: Smoking is Addictive. Never Starting Means Never Having to Quit.” The 1980 Surgeon General’s Report on Smoking and Women reveals that smoking is increasing among teenagers. The AMA is very concerned about teenage smoking and supports a warning directed at teenagers. Again, it should be pointed out that in the specific sense, cigarette smoking results in a dependence not an addiction.

Smoking and Teenagers

A more explicit warning, while an improvement, will not be a complete solution. Educational programs that emphasize the harmful aspects of smoking from a teenager’s point of view should be pursued, and the use of “role models” in cigarette advertisements should be eliminated. Issues of concern to an adult, like a decrease in life expectancy, may not influence a teenager’s decision to smoke. More information is needed on youth smoking, such as why they begin to smoke, why they quit smoking, and what method is most effective in urging teenagers to quit smoking. Once this information is available efforts to reduce teenage smoking can be better directed.
Because of the serious health risks involved with cigarette smoking, we believe cigarettes should not be distributed to minors. The AMA has drafted model state legislation prohibiting the distribution of cigarettes to minors. The AMA has also approved related model legislation that would prohibit distribution of free cigarette samples on public property.

Disclosure of Tar, Nicotine and Carbon Monoxide Levels

The AMA supports provisions that would require the level of tar, nicotine and carbon monoxide contained in cigarettes to be disclosed on the package. Recent trends show increasing use of low tar and nicotine cigarettes and, therefore, the information regarding the level of these ingredients is important consumer information.

A word of caution must be added. The switch to low tar, and nicotine cigarettes may be the result of information asserting a lower risk of lung cancer if low tar and nicotine cigarettes are smoked. In a 1980, public opinion poll 76% of smokers surveyed expressed a belief that smoking low tar and nicotine cigarettes did not significantly increase a person's risk of disease over that of a nonsmoker. The available research does not support this belief. The Surgeon General's report of 1981 was devoted entirely to the health significance of low tar and nicotine cigarettes. According to the report, while research indicates low tar and nicotine cigarettes are less dangerous than regular cigarettes, the benefits are minimal in comparison to not smoking. Smoking low tar and nicotine cigarettes rather than regular cigarettes reduces the risk of lung cancer but it is not clear what, if any, other risks are reduced. The Surgeon General's report states:
It is important to recognize that the largest component of excess mortality caused by smoking is cardiovascular disease deaths. There is not sufficient evidence to conclude that use of lower "tar" and nicotine cigarettes causes any reduction in this burden. The same is true of the other major diseases caused by cigarette smoking, most notably chronic obstructive lung disease and adverse effects on pregnancy.

Research also shows that many smokers increase the number of cigarettes smoked by smoking more deeply on smoking low tar and nicotine cigarettes. This can negate any potential benefits from switching to low tar and nicotine cigarettes.

In view of the limited knowledge about the reduced risk of low tar and nicotine cigarettes, care should be taken to avoid conveying to the public an impression that low tar and nicotine cigarettes are safe or even significantly safer than regular cigarettes.

Carbon monoxide is a known harmful element of cigarette smoke and may be responsible for low tar and nicotine cigarettes not being significantly safer than regular cigarettes. Research suggests that this may be true for risks related to pregnancy. This suggests that consumers, when attempting to select a safer cigarette, may want to consider carbon monoxide levels in cigarette smoke as well as tar and nicotine levels of cigarettes. The AMA believes this information should be provided.

Disclosure of Chemical Additives

The Surgeon General has expressed concern regarding the health effects of chemical additives used in the manufacture of cigarettes. The importance of additives has increased as manufacturers have modified their cigarettes to produce a low tar and nicotine cigarette. The major
obstacle to the study of the relative risks of cigarette additives is the lack of information as to what additives are used.

This bill would provide the mechanism for HHS to acquire such information and require HHS to report to Congress on the additives, the research regarding the health risks associated with such additives, and information regarding an additive that the Secretary believes poses an additional health risk. The AMA supports efforts to determine the health effects of smoking and believes that information on the additives being used is essential. The AMA supports provisions of the bill relating to chemical additives as a necessary mechanism for acquiring information necessary to determine the health effects of cigarette additives.

**Smoking Cessation**

Even those who are aware of the dangers of smoking may have difficulty quitting because of the development of a dependency on cigarettes. The Surgeon General's report shows that up to 50 percent of those who quit smoking will remain non-smokers. Assistance must be provided to the remaining 50 percent if smoking is to be further decreased significantly. The AMA developed an audiovisual presentation on "how to quit smoking" that will be available this summer for physicians to use in assisting patients desiring to quit smoking. The proposed changes in the warning, if adopted, may create more awareness of the dangers of smoking, and more smokers will want to stop smoking. Programs to assist those who want to quit smoking will be needed even more in that case.

**Conclusion**

The AMA supports efforts to increase public awareness of the hazards of smoking. We believe that the best method to decrease smoking is to
help people avoid starting the habit. One method to discourage smoking would be a clear indication of the health hazards of smoking. Thus, the AMA supports a more explicit warning on cigarette packages and in advertisements. S. 772 would provide for explicit warnings on cigarette packages and, in our view, would increase awareness of the specific risks of smoking. We urge enactment of this important public health legislation.
May 26, 1985

The Honorable Orrin G. Hatch  
U.S. Senate  
Washington, D.C. 20510

Dear Senator Hatch:

At the May 12th hearing on S. 277, the "Comprehensive Smoking Prevention Education Act," Senator Judah, speaking on behalf of the Tobacco Institute, made reference to the Multiple Risk Factor Intervention Trial (MRFIT) study conducted by the NIH. The industry stated that "this government research project cast grave doubts on the validity of the 'finding' in N.I.H. report concerning smoking cessation." The industry however seems to be seriously distorting the results of the study. As we noted in our testimony before your Committee on May 9th, the industry avoids the acknowledge of the study which showed that both groups, the "special intervention" group and the "usual care group" reduced their risk factors, including cigarette smoking.

I would like to submit for the inclusion in the hearing record a copy of the MRFIT findings as well as the statement made by Dr. Brandt at the release of the study.

If the American Heart Association can be of any assistance to you or your staff please feel free to call on us.

Sincerely,

Mary Jane Jesse  
President

(Note: In the interest of economy, the MRFIT study referred to was retained in the files of the Committee where it may be researched upon request.)
STATEMENT BY: Edward N. Brandt, Jr., M.D., Assistant Secretary for Health on the release of findings from the Multiple Risk Factor Intervention Trial (MRFIT) September 16, 1982

The National Heart, Lung, and Blood Institute of the National Institutes of Health today released the findings of a major study, the Multiple Risk Factor Intervention Trial (MRFIT). The study results contain important lessons for the health of Americans.

This clinical trial, initiated 10 years ago, was designed to demonstrate the value of special intervention to reduce health risk from smoking, high blood pressure and elevated cholesterol levels. An issue to be addressed was the extent to which intervention could affect death rates due to coronary heart disease. Approximately 13,000 middle-aged males at high risk for heart disease were enrolled in the study. Of those, about 6,500 received "special intervention" which included dietary modification, antihypertension treatment, and/or counseling for smoking cessation. Another 6,500 middle-aged white males were assigned to the "usual care" group. That is, the level of care or intervention they received was largely dependent on the quality of care in their own individual communities. Each of these volunteers was followed for a minimum of six years.

The results of this trial, released today, confirm our success in reducing death rates from cardiovascular disease in this country. Both groups of men had far fewer cardiovascular deaths than were anticipated and the differences between the special intervention group and the usual care group were not considered statistically significant.

When the study was planned, a larger difference in mortality was anticipated. At the same time, it was not anticipated that the reductions in the three risk factors under study would be so great in the "usual care" group. That observation is quite important, for it emphasizes that the "usual care" afforded to Americans results in reduced risk factors or American men, recognizing the importance of risk factor reduction, have worked to do so, or both. This is an important observation.

This study again shows the value of reducing one's risks of coronary heart disease. For example, the death rate for those who quit smoking, even for one year, was about half that of those who did not. That should stimulate middle-aged male smokers to quit.

The data we have from MRFIT is impressive; many more results will be coming out of the computers in the months to come. There are more important lessons to learn.

Let me again emphasize that both groups reduced the three risk factors under study, and both groups experienced a mortality rate considerably less
than was projected from 1970 data. In my view, the lesson from the study is that middle-aged men can reduce their risks of death from coronary heart disease and cardiovascular disease by

- Reducing high blood pressure
- Lowering elevated cholesterol levels, and
- Quitting smoking.

Every American man should take cognizance of these results and initiate the appropriate actions with his physician. I urge every physician to assist their patients in achieving the goal of a lowered risk of death.
STATEMENT IN OPPOSITION TO S.772

Smoking Prevention Health and Education Act of 1983

By

Richard W. Funk
Counsel

National Automatic Merchandising Association
On Behalf of its Members and the Vending Industry

Filed With

Senator Orrin G. Hatch
Chairman
Committee on Labor and Human Resources
United States Senate

1983 NATIONAL CONVENTION • CHICAGO • OCTOBER 11-16
STATEMENT IN OPPOSITION TO S 772

The National Automatic Merchandising Association (NAMA) is the national trade association of the merchandise vending and contract food service management business. Its over 2400 member firms include service companies, vending machine manufacturers and suppliers of vendible products. Among products its members retail are cigarettes and other tobacco products sold through vending machines.

Annual cigarette sales through vending machines in 1981 totaled 3,784,000,000 packs with a total dollar volume of $3,180,000,000. Total packages sold by the industry represents 12% of the total cigarette market in the United States. The dollar volume represents 22% of the total vended product volume of all items sold through merchandise vending machines. Clearly the vending industry has a stake in the tobacco industry generally and the retail distribution of cigarettes specifically.

On behalf of its members NAMA takes this opportunity to state its opposition to S 772, a bill to, among other things, establish a national program to increase the availability of information on the health consequences of smoking and to amend the Federal Cigarette Labeling and Advertising Act to change the label requirements for cigarettes.

At the outset NAMA takes no position in regard to the controversy over smoking and health. Serious research on the physiological effects of smoking has been conducted on a large
scale over a long period of time and there remains much to be
done to establish scientific cause and effect relationships
between smoking and various diseases and other deleterious
physiological effects. N A A joins with other segments of
the tobacco industry in support of such research. We acknowledge
also that the Department of Health and Human Services has deter-
mined that cigarette smoking is dangerous to health and based on
that determination has led government educational efforts to
discourage smoking.

New Cigarette Packaging Labeling Requirements

Government agencies have overwhelmed the American public with
constant disseminations of warnings and exhortations about the
evils of cigarette smoking. Among the many ways of informing
the public, the Federal Cigarette Labeling and Advertising Act
requires the following warning to be placed on all cigarette
packages and to be used on all cigarette advertising: "The Surgeon
General has determined that cigarette smoking is dangerous to
your health." For those who smoke, this message has been read
untold times and presumably it has changed the smoking habits of
many smokers. All nonsmokers have been exposed to the same warning
used in cigarette advertising throughout the country over a period
of many years. We seriously question whether the requirement for
rotation of four different new warning statements called for in
the bill will be more effective than the current warning label and
indeed might well diminish the impact of the warning with additional
clutter and unsupported conclusions.
Effect on Cigarette Vending

Under a current consent decree of the U.S. District Court between the Federal Trade Commission and the cigarette manufacturers, cigarette vending machines must display the warning label in close proximity to the coin slot. This is accomplished by the use of an adhesive label or a permanent label attached to the machine by the machine manufacturer.

The rotating label requirements of the bill would not allow the permanent attachment of labels at the point of machine manufacture but would require the use and change of adhesive labels many times annually on the over 800,000 cigarette vending machines on location throughout the country. This procedure would (a) obviously lessen the efficiency of bringing the warning to the public and (b) would result in substantial labor and material cost to the vending industry.

This wasteful expenditure of time, labor and money and the loss of guaranteed permanency of label placement would be the result of enactment of S 772.

The Congress ought to seriously consider the cost of the program outlined in the bill and whether this money could not be appropriated to better use. Propaganda alone will not solve the problem and therefore we suggest that appropriations to support such program and the added costs to the industry would be better spent in scientific research leading to definite conclusions about the impact of cigarette smoking on health.
Educational activities about smoking and health promulgated by the government and a wide variety of private sources have been more than adequate to alert the public about the possible health dangers related to cigarette smoking. What the public wants now is a definitive finding based on sound scientific evidence. S 772 does nothing to meet this need and would result in a waste of assets which could better be directed to intelligent inquiry.
Mr. Chairman, I am David Minton, Washington Counsel for the Magazine Publishers Association, a national organization representing approximately 200 publishing firms which publish approximately 800 consumer magazines. Most of the weekly and monthly publications which the American people read are members of MPA.

Last year, when the Senate held hearings on similar legislation, MPA appeared in opposition to those provisions of the legislation which would have imposed specific warning label requirements on all cigarette advertising. S. 772 does not include those specific requirements, but the effect of the enactment of this legislation would undoubtedly trigger FTC action resulting in imposing the package labeling notices in advertising. So our position on your new legislation is identical to our position on last year's bill: The Magazine Publishers Association opposes this legislation for two reasons: we think it exceeds the permissible limit of government regulation of advertising as that limit has been established by the Supreme Court of the United States, and we think that even if it were not unconstitutional, it will not achieve the goal you appear to be pursuing -- persuading people to stop smoking. If there is room to differ as to the correctness of our objection to the bill on constitutional grounds, the evidence indicating that labeling is not likely to help achieve your objectives should
lead you to refrain from imposing the restraint upon free speech in the first place.

"Commercial speech" is a term used to differentiate between speech which relates to economic interests and speech which does not. In the early development of commercial speech protection, the Supreme Court, in Valentine vs. Chrestensen, decided in 1942, held that the Constitution did not protect this form of speech. A New York statute prohibiting the distribution of handbills "or other advertising matter" in any "public place," was legal. The Court said, "We are equally clear that the Constitution imposes no such [First Amendment] restraint as respects purely-commercial advertising. 1/ A similar conclusion was reached in Breard vs. Alexandria, involving door-to-door salesman peddling without a permit. But since Breard, in 1951, the Court has not denied protection to commercial speech on that basis alone, and, in the words of the late Mr. Justice Douglas, the Chrestensen rule "has not survived reflection. 2/

In 1975, the Supreme Court first extended protection to advertising. In Bigelow vs. Virginia, a Virginia statute which made the newspaper advertisement of abortion referral services a crime was struck down as an unconstitutional infringement upon the First Amendment. If there were lingering doubts as to "purely" commercial speech's status under the First Amendment.

1/ Valentine vs. Chrestensen, 316 U.S. 52 (1942).
because the Bigelow case involved abortion services — a public issue transcending mere commercial speech — the Court's decision in Virginia Pharmacy in 1976 laid all doubts to rest. Justice Blackmun, speaking for the Court, defined the issue to be whether purely commercial speech was outside the protection of the First Amendment. "Our answer," Justice Blackmun said, "is that it is not."

The Court has nevertheless recognized legitimate avenues for regulation of commercial speech, just as there are legitimate grounds for the regulation of political speech. The Securities and Exchange Act, the Sherman Antitrust Act, and a number of other laws regulate commercial speech, but the interests of society in the positive benefits of those legislative aims have been considering an "overriding" public interest. Virginia Pharmacy itself prescribed that purely commercial speech could be regulated to be "clean" as well as "free." A clear exception to the protection of the Constitution is deceptive or misleading advertising.

In 1980, the Supreme Court defined in detail the constitutional protection of commercial speech in Central Hudson Gas. That case involved a New York State regulation which banned commercial advertising by a Public utility which promoted the purchase of natural gas. Justice Powell laid out the rule to be

followed to test constitutionally permissible regulation of advertising. He said,

If the communication is neither misleading nor related to unlawful activity, the government's power is more circumscribed. The state must assert a substantial interest to be achieved by restriction on commercial speech. Moreover, the regulatory technique must be in proportion to that interest. The limitation on expression must be designed carefully to achieve the state's goal. Compliance with this requirement may be measured by two criteria. First, the restriction must directly advance the state interest involved, the regulation may not be sustained if it provides only ineffective or remote support for the government's purpose. Second, if the governmental interest could be served as well by a more limited restriction on commercial speech, the excessive restriction cannot survive.

Senator, we believe that the restrictions which would result from the enactment of this legislation proposed do not meet the requirements prescribed in Hudson Gas. They do not because advertising and label warnings do not have a sufficiently effective impact upon public behavior, most particularly where personal habits are involved. Most likely, the effectiveness would be unmeasurable. Finally, the goal which you seek to achieve may be more effectively achieved by means not involving further restrictions upon the Freedom of Speech.

In determining whether the proposed restriction violates the rule enunciated in Hudson Gas, we must first determine whether cigarette advertising is free of misleading content. To mislead, Webster says, is to lead in a wrong direction or into a mistaken

action; to lead astray. See deceive." "Deceive," Webster says, 
is to cause to believe the false."

Deception in advertising usually involves affirmative claims. "Wonder Bread builds healthy bodies 12 ways." Household Finance makes "Instant Tax Refunds." That is a positive claim or something good that will happen if you buy Wonder Bread or take your tax return to Household. In fact, it was not so, so the FTC in those cases ordered the ads to be withdrawn.

Cigarette advertising does not make affirmative claims in regard to the effect of smoking upon health. There are statements of fact: nicotine and tar content, determined under FTC standards. "There are also claims that the brand advertised "tastes better," which is a matter of opinion. In recent years, tar and nicotine content have become a major advertising feature for most brands, and perhaps a reason for switching brands. That in itself demonstrates public awareness.

An advertisement which makes no affirmative claim to anything and which has a health warning in plain sight and plain words is not deceptive, and therefore is within the boundary of protection for commercial speech prescribed by the Supreme Court.

The second issue is whether the remedy proposed is likely to be effective, or whether the results will be ineffective or remote, thereby failing the Court's standard.

The FTC staff concluded in 1981 that the current cigarette warning label is ineffective. Since it appears that the FTC's staff's goal is the elimination of cigarette smoking in the United States, there may be many programs which would fail to
meet the staff's test. Advertising labels may be one of them, but the claim that the public is not aware defies common sense and the Surgeon General's most recent report. The 1982 Report of the Surgeon General showed that today 53 million people smoke, about the same number as 20 years ago. That is a significant decline in the percentage of the population. There were about 180 million people in the United States in 1962, and there are about 230 million today. The percentage of adult smokers has dropped from 42% to 33%. Public attitudes have changed; medical advice has changed. Radio and television advertising is no longer available.

In testing whether legislative control complies with the standards set out in Hudson Gas, the relevant question is whether increased restrictions will have a direct impact upon achieving the legislative goal. We believe that the correct answer is either "no" or "nobody knows." In either case, we believe that the prudent advocate of constitutional freedom should refrain from restraint upon free speech rather than impose further questionable government restrictions.

Recent studies of the effectiveness of warning advertisements and labeling show that the public tends to ignore them. In their November, 1980 Report to the President and the Congress, the Departments of the Treasury and Health and Human Services found that "the public generally is over warned" by the government and that the effectiveness of warning declines as the degree and frequency of warnings increase. Personal attitudes, experience, and habit play a highly significant role in
determining whether a person pays attention to warnings, regardless of the consequences. The 1131 Report specifically found that "fear statements" are "generally not as effective...and may cause the audience to feel overly threatened and, as a result, screen out the message." The "size" of the problem is related to the effectiveness of the warning, too. Many people switched from aerosol spray cans to carbon dioxide spray cans when alerted to aerosol's threat to the level of ozone in the stratosphere. Residents near Three Mile Island moved out quickly. The widespread fear of strontium 90 in cows' milk was a significant factor 25 years ago for banning nuclear testing in the atmosphere. Those are big threats of almost incomprehensible proportions which appear to threaten all life on earth.

When it comes down to the personal level, the effectiveness of warnings is significantly lower. The regulation of personal behavior, particularly personal habits, is extremely difficult. Many manufacturer warnings or instructions do not appear to work. Controlled experiments illustrate the problem. One recent experiment involved the use of hammers -- the device you drive nails with -- which had been carefully labeled to warn of danger, or to instruct the user not to use the tool at all. One hundred high school and college students were asked to use the hammers to drive nails in pieces of wood. They all did, and

5/ Report to the President and the Congress on Health Hazards Associated with Alcohol and Methods to Inform the General Public of these Hazards, U.S. Department of the Treasury and U.S. Department of Health and Human Services, November, 1980.
following the experiment, all were asked what the labels said. Not one out of 140 had even noticed the labels. Everyone knows how to spell "relief," not everyone knows that the label of that highly publicized over-the-counter antacid contains a 65-word warning as to the dangers involved.

It is an interesting Problem. Automatic seat belts, required on all American cars in the mid-1970s, were so unpopular with the American people that Congress repealed the requirement, despite overwhelming evidence as to the effectiveness of the belts, and the failure of people to use seat belts which require personal fastening. Whatever the reason, in personal matters -- don't smoke, don't drive when you're drinking, -- admonitions of the consequence, regardless of the evidence, don't seem to have such effect. In the specific instance of the Government’s 15 year campaign to persuade people to quit smoking, the 1980 Treasury/HHS Report concluded that “it is impossible at this time to isolate the impact of any specific communication technique on smoking behavior.”

To extend further restrictions upon the freedom to advertise a commodity the manufacture, sale, and consumption of which is legal in every state in the Union is not called for, particularly in light of substantial evidence that this legislative remedy would not be effective.

The American Newspaper Publishers Association is concerned that several provisions of HR 1824, "The Comprehensive Smoking Prevention Education Act," raise certain First Amendment issues which must be carefully considered.

ANPA is a national trade association whose more than 1,400 member newspapers represent more than 90 percent of the U.S. daily circulation in the United States. Many non-daily newspapers also are members.

Section 4 of HR 1824 is of particular concern to ANPA. It would require that all cigarette advertising contain one of three specifically worded statements warning of the potential hazards of smoking. The Secretary of Health and Human Services (HHHS) would be given the authority to develop a system whereby these warning labels appeared an equal number of times on each brand of cigarettes and reference advertisements of each brand within a 12-month period and for each succeeding 12-month period. Moreover, HR 1824 also would require the label statement to 'appear in conspicuous and legible type in contrast by typography, layout, or color with all other printed material ... in the advertisement.'

While ANPA understands the public health concerns which have motivated this legislation, we believe that Congress should proceed with extreme sensitivity and caution when it considers regulating speech as a method of regulating the sale and use of an entirely legal product.

By seeking to extend the warning label requirement to advertising, to specify the precise words to be used in three different warning messages required to be contained in such advertising, and to direct the establishment of a new regulatory system by which the government would determine, in effect when and by whom each warning message would be published, HR 1824 proposes a substantial expansion of governmental control over the commercial speech of advertisers and, indirectly, over the newspapers printing that speech. ANPA
Advertising is the central means of communicating information required for economic decision-making in our free society. In a series of decisions including Virginia State Board of Pharmacy v. Virginia Citizens Consumer Council, Inc., 425 U.S. 748 (1976); Central Hudson Gas & Electric Corporation v. Public Service Commission of New York, 447 U.S. 557 (1980), and most recently In Re R.M.J., 455 U.S. 191 (1982), the U.S. Supreme Court has recognized that advertising enjoys significant First Amendment protections. While the Court has indicated that some of the full protection of political speech may not be accorded purely commercial speech, the burden is on the government to show a compelling need for regulation and also to show that the regulation is no more extensive than necessary. As the court stated, "Although the potential for deception and confusion is particularly strong in the context of advertising professional services, restrictions upon such advertising may be no broader than reasonably necessary to prevent the deception." See In Re R.M.J., supra.

In classic First Amendment analysis, speech which is governmentally mandated is, if anything, even more suspect than speech which is restrained. See Miami Herald Publishing Company v. Tornillo, 418 U.S. 241 (1974).

Enactment of R.R. 1824 would be a significant escalation of regulation of speech in this area. It is one thing to mandate a warning label on cigarette packages, and quite another to specify statutorily the precise language of three specific messages which must be contained in advertisements as well as in packaging, and to provide for a system under which the questions of when those messages will be run and by which advertisers are determined by the government.
AMA recognizes that the First Amendment protections offered commercial speech are not absolute, and that some forms of regulation are permissible. For example, place and manner restrictions are applicable, as is regulation of commercial speech that is false or deceptive. Similarly, there may be restrictions on advertisements for transactions or products which are illegal. See, e.g., Pittsburgh Press Company v. Human Relations Comm'n, 413 U.S. 376 (1973). Some of those justifications for regulation of commercial speech apply to cigarette advertising. Although cigarette smoking may well be harmful to public health, it is not illegal, nor is cigarette advertising inherently misleading or deceptive. Likewise, the approach of NR 1824 is not a time, place or manner restriction in that it mandates publication of required content.

In Central Hudson Gas & Electric Corp v. Public Service Commission of New York, 447 U.S. 557 (1980), the Supreme Court struck down a New York regulatory ban on promotional advertising by electric utilities. In his concurring opinion in that case, Justice Blackmun expressed concern about regulating speech that is not deceptive or misleading "... in order to manipulate a private economic decision that the state cannot or has not regulated or outlawed directly". 447 U.S. at 574.

The Court recognizes that we have never held that commercial speech may be suppressed in order to further the State's interest in discouraging purchases of the underlying product that is advertised. Permissible restraints of commercial speech have been limited to measures designed to protect consumers from fraudulent, misleading or coercive sales techniques.

447 U.S. at 574.
It may be argued that the warning messages required by 45 U.S.C. 182 are simply examples of the kinds of warnings and disclaimers that the Supreme Court, several of the recent commercial speech cases such as Virginia Pharmacy and In re E.H., has indicated may be permissible forms of regulation. Uniformly, however, references to the possibility of requiring such disclaimers occur in the context of discussion of commercial speech that is deceptive or misleading. For example, in Virginia Pharmacy, Justice Blackmun states:

The [excesses of commercial speech such as the greater objectivity and hardness] may also make it appropriate to require that a commercial message, advertise such facts, or include such additional information, warnings, and disclaimers, as are necessary to prevent its being deceptive.

623 U.S. at 773, n. 24 (Emphasis added).

There is no suggestion that cigarette advertising is so inherently deceptive or misleading as to require the mandatory inclusion of specifically worded warning messages. This is not to say that cigarette advertising which is deceptive or misleading is immune from challenge. Under Section 5 of the Federal Trade Commission Act (15 U.S.C. Sec. 45), the FTC clearly has the power to deal with such advertising. Absent such a specific finding, it should not be assumed that cigarette advertising per se is deceptive without a health warning.

In the Central Hudson case, decided in 1980, the Supreme Court set out a four-step analytical framework for examining restrictions on commercial speech:

In the Central Hudson case, decided in 1980, the Supreme Court set out a four-step analytical framework for examining restrictions on commercial speech:

...
In commercial speech cases, then, a four-part analysis has developed. At the outset, we must determine whether the expression is protected by the First Amendment. For commercial speech to come within that provision, it at least must concern lawful activity and not be misleading. Next, we ask whether the asserted governmental interest is substantial. If both inquiries yield positive answers, we must determine whether the regulation directly advances the governmental interest asserted, and whether it is not more extensive than is necessary to serve that interest.

441 U.S. at 566.

When this four-part test is applied to 82 182, the first two inquiries can be answered positively: cigarette advertising, in general, is not misleading and is therefore protected by the First Amendment, and the asserted governmental interest — public health — is substantial. It is in connection with the last two questions that AMPA believes the commercial speech provisions of 82 182 do not satisfy this constitutional test: whether mandatory warning messages in cigarette advertising "directly advance[s] the governmental interest asserted" and more importantly, whether the government's intrusion into commercial speech "is not more extensive than is necessary."

Whether a series of warning messages carried in cigarette advertising "directly advance" the governmental interest in public health is open to serious question and should at least be the subject of careful debate and specific factual showings. There is already a required health disclaimer in cigarette advertising and packaging, if that has been ineffective, what evidence is there that the new multiple warnings will effectively advance the government's interest?

A more critical inquiry is whether the kind of approach represented by 82 182 unnecessarily involves the government in a pervasive system
of regulating non-deceptive commercial speech. Is it essential, for example, that the NEA be given the power to dictate which advertising will carry which message and for how long?

No matter how seemingly laudable the goal, any legislation which proposes to delegate to government the power to decide what will be published and when something will be published in constitutionally protected Private speech, equally troublesome in the proposed government requirement that specific words must be published in not just one but three different messages. APA believes this would impinge on the constitutionally-protected domain of advertisers and the press.

Commercial speech restrictions must be a "last resort" action of government. Only where there is a concrete showing that an identifiable problem cannot be solved by some other than restricting speech can the government turn to measures such as those embodied in this legislation. The process of reaching this decision must include a complete assessment of all the information now available to the public concerning cigarettes and the health risks surrounding them. Having taken that and all other factors into account, they, and only then, can restrictions on commercial speech be considered. It is not at all clear that this rigorous process has been followed in advancing these legislative measures.

The principal concern of APA about this legislation is the proposed resort to government regulation of speech as a means of regulating a product which for whatever reason, the government chooses not to regulate directly.

We believe a very troubling precedent is set whenever the federal government determines the content of the speech of private entities. HR 1824 not only proposes to do that, but also would give a regulatory agency
the power to determine what certain messages would be published, for how
long and by whom. Absent compelling circumstances and clear evidence that
other, less intrusive alternatives are ineffective, any kind of governmental
action is inimical to the preservation of free speech and a free press
in our free society, thus the case has not been articulated which would
support as harsh a statutory restriction on commercial speech as that embodied
in St 1974.
On behalf of the Health Insurance Association of America and its 340 member health insurance companies, I want you to know of our continued concern about the health hazard of smoking, and particularly our support for legislation to strengthen warnings on cigarette packaging. We believe this will more effectively alert the public to the direct link of cigarette smoking and a large proportion of fatal diseases.

The Association's Board of Directors, at its meeting last Fall, addressed this issue and approved a resolution to this effect. As a result of strong recommendations from our industry's Advisory Council on Education for Health (composed of prominent professionals in the field of health education and wellness), our companies are also being encouraged and assisted to establish smoking cessation programs and policies for their employees and work with their group policyholders to this end. All this is a consequence of our industry's commitment to health promotion in general and to smoking cessation in particular.

Sincerely,

[Signature]

James L. Moorefield

US Senate, 135 Russell Building
Washington, D.C. 20510

June 13, 1983

The Chairman. The hearing is recessed.
(Whereupon, at 12:30 p.m., the committee recessed, to reconvene at the call of the Chair.)