DOCUMENT RESUME

ED 322 004

SE 051 512

AUTHOR

Crosser, Gail Hoddlebrink; Molleson, Ann L.

TITLE

Questions about Common Ailments. Nutrition in Health

Promotion Series, Number 26.

INSTITUTION Ohio State Univ., Columbus. Dept. of Family

Medicine.

Health Resources and Services Administration SPONS AGENCY

(DHHS/PHS), Rockville, MD. Bureau of Health

Professions.

PUB DATE

CONTRACT

240-83-0094

85

NOTE 47p.; See SE 051 486 for "Comprehensive Guide and Topical Index" to Modules 1-26. See SE 051 487-502 for Modules 1-16, "Primary Care Series" and SE 051

503-512 for "Nutrition in Health Promotion"

series.

Guides - Classroom Use - Materials (For Learner) PUB TYPE

(051)

EDRS PRICE MF01/PC02 Plus Postage.

DESCRIPTORS *Dietetics; Disease Control; Health Education; Higher

Education; Independent Study; *Medical Education; Medicine; *Nutrition; *Nutrition Instruction;

*Patient Education; Physiology; Preventive Medicine;

Science Education; *Special Health Problems

ABSTRACT

Nutrition is well-recognized as a necessary component of educational programs for physicians. This is to be valued in that of all factors affecting health in the United States, none is more important than nutrition. This can be argued from various perspectives, including health promotion, disease prevention, and therapeutic management. In all cases, serious consideration of nutrition-related issues in the practice is seen to be one means to achieve cost-effective medical care. These modules were developed to provide more practical knowledge for health care providers, and in particular primary care physicians. This module provides information concerning the role of nutrition in the development and/or prevention of common ailments. Emphasis is placed on ailments about which patients have concerns related to nutritional issues. Included are learning goals and objectives, a self-check of achievement with regard to goals, resources for patients and physicians, and references. Appendices include: (1) discussions of the dietary treatement of acne, gout, constipation, and hypercalciuria; (2) a table of fiber in foods; and (3) a suggested low-calcium diet. (CW)



Reproductions supplied by EDRS are the best that can be made

from the original document. ************************

The Nutrition in Primary Care Series Contains These Modules:

- 1. Nutrient Content of Foods, Nutritional Supplements, and Food Fallacies
- 2. Appraisal of Nutritional Status
- 3. Nutrient and Drug Interactions
- 4. Normal Diet: Age of Dependency
- 5. Normal Diet: Age of Parental Control
- 6. Normal Diet: Adolescence
- 7. Normal Diet: Pregnancy and Lactation
- 8. Normal Diet: Geriatrics
- 9. Dietary Management in Obesity
- 10. Dietary Management in Diabetes Mellitus
- 11. Dietary Management in Hypertension
- 12. Dietary Management in Hyperlipidemia
- 13. Dietary Management in Gastrointestinal Diseases
- 14. Dietary Management for Alcoholic Patients
- 15. Nutritional Care of Deteriorating Patients
- Ar Office Strategy for Nutrition-Related Patient Education and Compliance

The Nutrition in Health Promotion Series Contains These Modules:

- 17. Individual and Social Factors
- 18. Metabolic Principles
- 19. Risk Factors and Disease Prevention
- 20. Decoding Fad Diets
- 21. Protecting Bone and Teeth
- 22. Exercise and Physical Activity
- 23. Vitamins and Trace Minerals
- 24. Behaviorial and Neurological Disorders
- 25. Preventing Hospital and Home Malnutrition
- 26. Questions About Common Ailments

Faculty Guide (includes comprehensive index for Modules 1-26)

Department of Family Medicine College of Medicine - The Ohio State University 456 Clinic Drive - Columbus, Ohio 43210



26 Nutrition in Health Promotion: Questions About Common Ailments

Gail Hoddlebrink Crosser, M.S., R.D. Clinical Dietitian Nutrition Services Riverside Methodist Hospital Columbus, Ohio

Ann L. Molleson, Ph.D., R.D. Nutrition Consultant Columbus, Ohio

Project Editor
Lawrence L. Gabel, Ph.D.

Nutrition Content Editor Charlette R. Gallagher-Allred, Ph.D., R.D.

Family Medicine Content Editor Patrick J. Fahey, M.D.

Contract Number: 240-83-0094
U.S. Department of Health and Human Services
Public Health Service
Health Resources and Services Administration
Bureau of Health Professions
Division of Medicine
Project Officer: Margaret A. Wilson, Ph.D.



Acknowledgments

Project Staff

Lawrence L. Gabel, Ph.D.-Project Director, Associate Professor and Director, Graduate Education and Research Section, Department of Family Medicine, The Ohio State University, Columbus, Ohio

Joan S. Rehner-Project Assistant, Secretary, Graduate Education and Research Section, Department of Family Medicine, The Ohio State University, Columbus, Ohio

Patrick J. Fahey, M.D.-Family Medicine Coordinator, Assistant Professor and Director, Predoctoral Education Section, Department of Family Medicine, The Ohio State University, Columbus, Ohio

Charlette R. Gallagher-Allred, Ph.D., R.D.-Nutrition Coordinator, Nutritionist, Riverside Methodist Hospital, Columbus, Ohio

John S. Monk, Ph.D.-Evaluation Coordinator, Assistant Professor and Coordinator, Research and Evaluation, Graduate Education and Research Section, Department of Family Medicine, The Ohio State University, Columbus, Ohio

Independent Study Package Consultant

Tennyson Williams, M.D., Professor and Chairman, Department of Family Medicine, The Ohio State University, Columbus, Ohio

Nutrition Consultant

Jil Feldhausen, M.S., R.D., Nutritionist, Department of Family & Community Medicine, University of Arizona, Tucson, Arizona

Editorial Consult

Chester E. Ball, M.A., Assistant Professor Emeritus, The Ohio State University

Technical Assistants

Annette M. Battafarano, M.A., Graduate Research Associate, Graduate Education and Research Section, Department of Family Medicine, The Ohio State University, Columbus, Ohio

Richard E. Doty, M.S., Graduate Research Associate, Graduate Education and Research Section, Department of Family Medicine, The Ohio State University, Columbus, Chio



Criteria/Assessment Committee

Mark T. Winders, M.D., Resident, Department of Family Medicine, The Ohio State University, Columbus, Ohio

David R. Rudy, M.D., Director, Monsour Family Practice Residency Program, Monsour Medical Center, Jeannette, Pennsylvania.

Maria Steinbaugh, Ph.D., Associate Director, Nutrition Services, Ross Laboratories, Inc., Columbus, Ohio

Wilburn H. Weddington, M.D., Family Physician, Columbus, Ohio

A special note of appreciation is extended to persons in family practice residency programs and universities throughout Ohio for reviewing the materials and to the faculty and residents of the Central Ohio Affiliated Family Practice Residency Programs where the materials were piloted:

Grant Hospital, Columbus, Ohio Riverside Methodist Hospital, Columbus, Ohio University Hospital, Columbus, Ohio Mt. Carmel Hospital, Columbus, Ohio

Composition, Camera Work, Reproduction and Binding: Lesher Printers, Fremont, Ohio Library of Congress Catalog Card Number: 85-62199

Copyright © 1985 by the Department of Family Medicine, The Ohio State University. All rights reserved.



Contents	Page Introduction
Contonts	Goal
	Objectives
	Acne
	Alzheimer's Disease. 3
	Arthritis
	Constipation 6
	Epilepsy
	Food Allergy 8
	Gallstones
	Hemorrhoids
	Kidney Stones
	Migraine Headaches
	Multiple Sclerosis
	Psoriasis
	Sexual Potency
	Varicose Veins
	Summary20
	Evaluation20
	References
	Resources for Physicians
	Resources for Patients
Tables	26-1. Purine Content of Food
	26-2. Infant Formula Other Than Cow's Milk
	26-3. Summary of Selected Ailments Where
	Dietary Treatment Is Effective in Health Promotion18
Appendices	A. Dietary Treatment for Acne
• •	B. Dietary Treatment for Gout
	C. Approximate Fiber in Commonly Used Food Portions
	D. Dietary Treatment for Constipation
	E. Low-Calcium Diet (600 mg Ca)
	F. Dietary Treatment for Hypercalciuria



Introduction

"Doctor, will changing my diet aid in the prevention or treatment of this certain illness as reported in the newspaper?" How will you, as the physician, answer this frequently asked question from patients? Many answers may be found in popular women's magazines, newspapers, and books that patients read. The long-time interest in nutrition and health has grown to the current wide-spread emphasis on promoting the billion dollar health food industry and vitamin-mineral supplement sales to prevent or cure practically every illness known to man. Are you able to assist patients by providing information or recommending articles written by reputable individuals providing information based on scientific research? Most reports of fantastic success of various nutrients in curing some illness are based on anecdotal research. Do these reports provide legitimate facts to share with patients?

Fraud in autrition information abounds. Each year millions of dollars are spent in advertising the need for supplemental vitamins, minerals, and other non-nutrient substances (such as choline and lecithin). However, few claims, if any, reveal the real and probable dangers of ingesting these substances. Health food faddists often create false claims for various nutrients and appear to consider profit more than health motives. The cost of supplementation is not covered by third-party payments and is thus an additional cost to the patient.

Goal

The goal of this module is to provide information concerning the role of nutrition in the development and/or prevention of common ailments. Emphasis is placed on ailments about which patients have concerns related to nutritional issues.

Objectives

Upon completion of this module, you will be able to:

- 1. Answer patients' questions about the role of diet in the prevention and/or treatment of the following: acne, Alzheimer's disease, rheumatoid arthritis, gout. constipation, epilepsy, food allergy, gallstones, kidney stones, migraine headache, multiple sclerosis, psoriasis, and varicose veins.
- 2. Counsel patients regarding dietary modifications appropriate for the r medical care.
- 3. Identify patients at risk, monitor clinical signs to aid in the diagnosis of, and develop a management plan for the dietary prevention and treatment of acne, constipation, food allergy, gout, kidney stones, and migraine headache.
- 4. Discuss the scientific basis, or lack thereof, for the wide-ranging recommendations related to nutrition management of the ailments discussed in this module.
- 5. Identify methods which may be employed in examining the quality of nutrition recommendations related to diseases and ailments not covered in this module.



Acne

Do Foods Exacerbate Acne?

Dietary treatment of acne is controversial. Controlled studies have not shown that chocodate, sweets, or cola drinks should be removed from the diet. Alcohol in large amounts may worsen acne by increasing the inflammatory reactivity of the skin. Evidence for limiting fat is based upon observations that acne may improve after fasting or following a vegetarian diet.

The role of diet in acne is unclear. The most important factor in the pathogenesis of acne is probably the increased androgen production of puberty. Androgens lead to increased sebum production and may influence the keratinization and bacterial flora of the pilosebaceous duct.

Many lay persons believe that certain foods result in worsening of acne. Foods frequently implicated include chocolate, fatty foods, sweets, and cola drinks. The medical community has not reached a concensus on the effect of food on acne. The American Medical Association recommends avoidance of chocolate, sweets, soft drinks, alcohol, fatty foods, and iodine. ** Many dermatologists do not agree with this recommendation.

*Numbers refer to numbered references listed under the specific disease in the list of References.

What is the Role of Vitamins and Minerals in Acne?

Iodine, zinc, and the vitamin A derivative, 13-cis-retinoic acid, each play a role in acne. Therapeutic use of 13-cis-retinoic acid has been shown to be beneficial. Vitamin A therapy is not efficacious since toxic side effects occur before benefit is achieved. High doses of iodine can exacerbate acne and should be avoided. Not enough is known about zinc to consider use of zinc supplementation for acne treatment.

Vitamin A has been used in the treatment of acne. The beneficial effects of vitamin A therapy are usually not achieved before toxic side effects occur. Studies have shown that boys and girls with severe acne have significantly lower levels of retinol-binding protein than healthy controls. The level of retinol-binding protein is closely associated with the serum vitamin A level. Mor studies are needed to determine tissue levels of vitamin A.

Recently, a synthetic derivative of vitamin A, 13-cisretinoic acid was approved in the United States for treatment of severe, recalcitrant cystic acne. Impressive results have been reported, with prolonged remission in many individuals.² Suppression of the sebaceous glands appears to be the mechanism of action of the drug.

Side effects of 13-cis-retinoic acid are less severe than the toxic effects of hypervitaminosis A. Cheilitis, dry skin, facial dermatitis, pruritus, increased sun sensitivity, dryness of the oral and pharyngeal mucosa, headache, emotional changes, visual disturbances, and elevated serum triglycerides have been reported. All side effects are reversible once the drug is discontinued.

Several physicians have recommended that a triglyceride level be determined prior to treatment and at regular intervals during treatment with 13-cis-retinoic acid. The drug should be stopped if the triglyceride level becomes greater than 700 to 800 mg/dl to reduce the risk of pancreatitis.² Obese patients or those with a history of excessive alcohol intake should be advised to reduce caloric intake, decrease intake of saturated fat, control carbohydrate intake, and eliminate use of alcohol in an effort to control the serum triglyceride level.

Liver function tests should be monitored since 13-cisretinoic acid is metabolized by the liver. Other monitoring parameters include cholesteroi, high-density lipoproteins, and a complete blood count. One report of hypercalcemia has been published. The author suggests that serum calcium levels be monitored during therapy.³

A serious side effect of 13-cis-retinoic acid is that it is a potent teratogen. Women during child-bearing years should be tested for pregnancy prior to treatment. During therapy an effective form of contraception must be used. Women are warned to avoid pregnancy for at least 3 months after therapy has been completed.

Iodine in high doses (such as in some cough syrups) can exacerbate acne and should be avoided. There is no evidence that the low amounts of iodine in table salt or



fish are harmful. Avoidance of iodized salt and other foods which contain iodine could result in development of a goiter.

A low-zinc diet may worsen acne. This has been noted in patients receiving total parenteral nutrition solutions for several months which contained an inadequate amount of zinc. Treatment with zinc reversed the condition. In boys with severe acne, serum levels of zinc have been found to be lower than in healthy conttols. At this time, there is not enough evidence to recommend zinc supplementation or foods high in zinc for treatment of acne. Zinc toxicity can result in anemia and other problems. Appendix A, Dietary Treatment for Acne, may be used as a handout for acne patients.

Alzheimer's Disease

What is the Role of Nutrition in Alzheimer's Disease?

Considerable advances have been made in the understanding of Alzheimer's Disease, but the cause of the disorder remains unknown. The diet for patient's with Alzheimer's disease should be adequate in all nutrients, including a calorie level which will maintain a patient's weight. The efficacy of a low aluminum diet in treatment or prevention of Alzheimer's Disease has not been established.

Diet has been linked with Alzheimer's Disease. At one time it was proposed that aluminum toxicity was a cause of Alzheimer's Disease. When aluminum was injected into rabbit brains, formation of neurofibrillary tangles were similar to, but not identical to, the tangles found in the brains of Alzheimet patients.' However, others have failed to confirm the findings of elevated brain aluminum levels in patients with Alzheimer's Disease. Whether aluminum causes Alzheimer's Disease is far from proven but seems unlikely, given available evidence.²

Despite lack of supporting evidence, a low a uminum diet for Alzheimer's Disease patients has been suggested. Avoidance of medications containing excessive alum-

inum, such as antacids and buffered aspitin, used over long periods of time, has also been suggested for those with Alzheimer's Disease. Levick has recommended that the use of aluminum cookware be discontinued. However, it does not seem reasonable for the public to avoid the use of aluminum pans in food preparation, as it is unlikely the very small increase in the aluminum content of foods would be harmful to anyone, including patients with Alzheimer's Disease. Most diets average about 22 mg of aluminum daily of which less than 1 mg is likely to be absorbed. There is no evidence that a low-aluminum diet will prevent Alzheimer's Disease or be of value in treating patients with Alzheimer's Disease.

Considerable research in treating Alzheimer's Disease patients has focused on a method to stimulate production of the neurotransmitter, acetyl choline, necessary for cognitive function. It has been shown that the enzyme choline acetyltransferase, essential to production of acetyl choline, is dramatically reduced in Alzheimer patients by as much as 90 percent of normal. 6 Choline in a meal may lead to elevated brain acetylcholine. When lecithin has been used as a source of choline in research studies, it is in highly purified form, nor the form of most commercially available lecithin that health food stores recommend. Studies have shown that neithet choline^{7,8} nor lecithin used in the treatment of patients with Alzheimer's Disease have improved memory functions.

More studies are needed since there is still much misinformation and lack of understanding about Alzheimer's Disease and the role of nutrition in its prevention and/or treatment. Of importance in consideration for all Alzheimer's Disease patients is their physical activity level. Many such patients with Alzheimer's Disease are excessively physically active; many others are quite inactive. Calorie intake should be adjusted to meet the patient's calorie needs for weight maintenance. Sometimes, care-givers must lock up food or the patient will eat continuously. C. the other hand, some anorexic or semi-conscious patients may need to be fed via tube feedings. Attempts to make mealtimes enjoyable expetiences for patients with Alzheimer's Disease is important, but can be frustrating for care-givers. As a physician you need to anticipate problems and assist families in dealing with the day-to-day ptoblems in caring for a family member with Alzheimer's Disease.

Resources on Alzheimer's Disease for families and patients can be found in the section on Resources for Patients.



Arthritis

Is There a Special Diet for Prevention of Rheumatoid Arthritis?

Diet has not been shown by controlled studies to prevent or cure rheumatoid arthritis. Patients with rheumatoid arthritis are often anorectic and may have dysphagia. High-calorie commercial supplements may be helpful. Patients taking steroids may benefit from calcium supplementation and a diet low in salt and simple sugar.

Rheumatoid artiritis-is a systemic disease which can result in inflammation of any organ as well as the synovial membranes of the joints. Fever, anemia, and anorexia can accompany the disease. Weight loss, with inadequate intake of calories and/or protein, and development of deficiencies in vitamins and minerals are possible in patients with anorexia because of the pain of arthritis. Use of commercial dietary supplements such as Ensure, Sustacal, or instant breakfast can supply needed nutrients until appetite returns. Supplements or tube feeding may also be useful when rheumatoid arthritis is complicated by Sjogren's syndrome. This condition results in decreased saliva production which makes swallowing difficult.

Anemia is often present in those who have rheumatoid arthritis. This type of anemia is not corrected by increasing iron intake. A defect in the body's ability to re-use iron appears to be the culprit. When other signs of rheumatoid disease activity are controlled, the anemia can correct itself.

Osteopenia occurs frequently in rheumatoid arthritis patients. Loss of bone mass results when synthesis cannot compensate for accelerated lysis of bone due to inactivity and use of corticosteroids. Calcium supplementation to a total daily intake of greater than one gram/day and vitamin D supplements, not to exceed 800 I.U./day (2 x RDA), are prescribed to reduce osteopenia.

Steroids used in the long-term management of patients with arthritis have side-effects which may be successfully treated by diet. Glucose intolerance secondary to gluconeogenesis stimulation and possible insulin insensitivity due to glucocorticoids, is often well-treated by

omission of simple sugars from the diet. Frank diabetics may need counseling or the exchange system for blood glucose control. In addition, hypertension caused by the aldosterone-like effect of steroids is generally well-treated by a 4-gram sodium (no added salt) diet; a stricter diet is generally not needed, but a 2-gram sodium diet may be indicated in severe hypertension.

Is There a Special Diet for Prevention of Osteoarthritis?

No diet has been shown to prevent or reverse the breakdown and irregular repair of cartilage at the ends of bones in patients with osteoarthritis. The best diet to promote is one which is adequate in all nutrients and with a calorie level to achieve or maintain weight within a normal range.

Studies have shown that osteoarthritis develops earlier and more frequently in those who are greater than 10% overfat. Osteoarthritis occurs not only in the weightbearing joints but also in the finger joints. The reason obesity affects arthritis in the hands is not known. Unfortunately, no diet has been shown to prevent or reverse the breakdown and irregular repair of cartilage at the ends of bones in these patients.

Osteoarthritic patients should be encouraged to lose weight if they are overfat. Excess weight puts extra stress on the weight-bearing joints. Obese people tend to be less active, which might limit range of motion exercise and result in loss of joint function and contractures.

Is There a Special Diet for Prevention of Gout Attacks?

To treat gout, use of diet in addition to drug therapy is appropriate if the patient can continue to eat foods he enjoys and the protein intake is not restricted to less than needs. Control of obesity with use of a non-ketotic diet, avoidance of alcohol, and avoidance of strenuous exercise can work together with drug therapy to control gout.



Gout is the only form of arthritis that we know of today where diet may be preventive or therapeutic. Those who are susceptible to gout have a defect in the metabolism of uric acid which results in deposits of uric acid salts in joints such as the big toe. Hyperuricemia occurs either from overproduction and/or failure of the kidney to excrete adequate amounts of urate. Foods which are high in purines could theoretically precipitate a gout attack because the end product of purine metabolism is uric acid. In clinical settings, however, this probably rarely occurs. Rich sources of purines include organ meats, meat extracts, meat, poultry, seafood, beans, and peas. See Table 26-1 for the purine content of food.

Treatment of gout is usually through medications. If dietary management is also desired, the diet does not permit foods which are high in purines and the protein content of the diet should not be excessive. Protein needs in otherwise healthy adults can be met by providing 0.8 gm protein/kg of ideal body weight. (Example: A 150 lb. man should eat approximately 65-70 gm protein/day which would be contained in 1 egg + 2 cups milk + 6 oz. meat, poultry, or fish.) Use of alcohol should be avoided. Patients who follow this diet

Table 26-1

Purine Content of Food

Foods highest in purines (150-825 mg/100 gm)

Sweetbreads Anchovies Sardines Kioneys Meat extracts

Gravi**e**s

Liver

Foods high in purines (50-150 mg/100 gm)

Meat Poultry Seafood Meat soups Broth Beans, dried Peas, dried Lentils, dried Spinach Oatmeal

Wheat germ

eans, dried Bran

Adapted from Massachusetts General Hospital Diet Manual: Little, Brown, and Company. Boston, page 138, 1976.

may lower their serum uric acid levels by 0.5 to 1.5 mg/dl. Appendix B, Dietary Treatment for Gout, may be used as a handout for gout patients.

Other factors which can reduce serum uric acid include:

- Control of obesity
- Avoidance of diets which produce ketosis (ketosis can exacerbate hyperuricemia)
- Avoidance of strenuous exercise (exercise can result in an acute rise in serum uric acid levels)¹

Some health professionals have suggested that the diet for gout is outdated now that drug therapy is available. Persons who overproduce uric acid may be prescribed Allopurinol, a xanthine oxidase inhibitor. Allopurinol blocks the conversion of purine to uric acid in the liver. Those who do not excrete enough uric acid may take probenecid (Benemid) and sulfinpyrazone (Anturane) which inhibit tubular reabsorption of uric acid. To prevent renal stone formation, urine volume should be greater than 2 liters/day.

What Diets Have Been Promoted for Arthritis?

The list of diets for arthritis is quite lengthy since nearly every week a new plan is published in newspapers, magazines, and books. According to the Arthritis Foundation, the following diets have NOT been shown to be effective in scientifically controlled studies:

- Avoidance of citrus fruits (or acid fruits and vegetables, such as tomatoes)
- Allowance of only a single type of food per meal (such as a meat limited to protein with no carbohydrate or fat)
- Alteration of the acid-base balance of the diet
- Avoidance of "nightshades" (potatoes, tomatoes, peppers, eggn! int)
- Avoidance of dairy products
- Avoidance o: foods to which the person is "hyperser.sitive" (such as nuts or tomatoes)
- Avoidance of meat, processed foods, spices, additives, preservatives, alcohol, and dairy products
- Supplementation with alfalfa tablets

A list of publications (both recommended and not recommended) for education of arthritis patients can be found in the section on Resources for Patients. The list may be reproduced as handouts for patients and their families.



Why Do Many People Claim that Diet Helps Their Arthritis?

Since arthritis symptoms tend to "come and go," diet often is wrongly implicated as a curative (or causal) factor in arthritis. If a person stops eating tomatoes and other acid foods at the same time a remission starts, he/she could easily believe that the improvement is a result of diet.

Arthritis can go into remission due to drug therapy, physical therapy, bedrest, or for unknown reasons. The length of time which remissions continue can vary from one day to several years or a lifetime. There is no way to predict how long a remission will last.

The "placebo effect" is a powerful phenomenon which can occur when a special diet is followed or special food or supplement is consumed. If a person firmly believes that a diet, food, or supplement relieves arthritis, there is a good chance that it will appear to do so. If the diet, food, or supplement is subjected to a double-blind study, the results have always confirmed that it was "belief" which caused the improvement. Usually the "placebo effect" results in only temporary relief of symptoms.

Should Someone Who Has Arthritis Take Vitamin/Mineral Supplements?

Vitamin/mineral supplements are not helpful for those with arthritis who are well nourished.

Vitamin and mineral supplements are not helpful for those with arthritis who are well-nourished. Megadoses of certain nutrients can be toxic. A general purpose multi-vitamin/mineral supplement can be beneficial during periods of anorexia or in case of malnutrition. Use of calcium supplementation for osteopenia of rheumatoid arthritis is discussed at the beginning of the section on Arthritis.

Zinc deficiency in humans has been associated with immunological defects. Studies have shown that some people who have rheumatoid arthritis or osteoarthritis have inadequate zinc intakes.² Blood levels of zinc have

been shown to be lower than normal in some who have rheumatoid arthritis. However, there is currently not enough research to know if zinc supplementation would be of benefit or if foods rich in zinc should be encouraged for patients with arthritis. Toxicity of zinc from indiscriminate mineral supplementation is known to occur.

Constipation

Can Diet Prevent Constipation?

Dietary fiber can relieve constipation by increasing the water content of the feces and producing stools of larger diameter which are soft but well-formed. Fluid intake must be increased if the higher fiber diet is to be effective in relieving constipation. Patients should be encouraged to drink additional fluid with each meal and between meals. Failure to drink adequate amounts of fluid can aggravate constipation.

Dietary fiber can relieve constipation by increasing the water content of the feces and producing stools of larger diameter which are soft but well-formed. Bran is the most effective fiber for increasing stool size. Whole grain breads and cereals are rich in bran. Other sources of fiber are fruits, vegetables, nuts, seeds, and legumes. (See Appendix C for approximate fiber content of commonly used food portions.) Prunes and prune juice contain a laxative ingredient which stimulates intestinal motility.

Commercial fiber supplements are available; however, they are not equally effective in increasing stool size. Purified cellulose has minimal effect as a stool softener while bran is quite effective. Food form has an effect on the water-holding property of bran. Coarsely ground bran holds more water than finely ground bran.

If the individual is unable to eat adequate amounts of whole grain breads and cereals, up to 2 tablespoons per day of Miller's Bran may be added to foods such as cereal, mashed potatoes, soup, applesauce, or meatloaf. A level teaspoon of bran contains about two grams of dietary fiber. A high-fiber diet may initially cause flatulence. For this reason the fiber content of the diet should be increased gradually. If Miller's Bran is used, 2 teaspoons per day would be a good starting place. Each



week 2 additional teaspoons could be added — up to 6 teaspoons (which is two tablespoons) within 3 weeks.

It is not possible to recommend the amount of fiber needed to prevent constipation because there are limited data on the composition and physiological effects of fiber, and the amount differs between persons. Desirable fiber intakes suggested in the literature range from 25 to 50 grams/day. Many Americans eat less than 20 grams of fiber per day.

High-fiber diets have been shown to decrease the absorption of nitrogen, fat, and certain minerals such as calcium and magnesium. Bran has been shown to affect mineral absorption more than the fibers found in fruits and vegetables. Clinical significance of these findings is unknown. The effect of high-fiber diets on vitamin absorption is also unknown. In these patients, it would be prudent to advise taking a one-a-day type multiple-vitamin and mineral supplement equal to 100% of the U.S. RDA.

It is probably wise to avoid use of purified fiber supplements if possible, since knowledge about the physiological effects of dietary fiber is limited. Bran has been shown to reduce the required dosage of long-term anticoagulants, presumably by affecting the amount of endogenous vitamin K synthesized by intestinal flora, and the effect on clotting time should be checked.

Assuring adequate fluid intake is as critical in treating constipation as is fiber intake. Patients should be encouraged to drink a minimum of six to eight glasses of liquid daily with meals and between meals. Failure to drink an adequate amount of fluid can aggravate constipation and can contribute to dehydration, especially if dietary intake of fiber is high.

Long-term use of laxatives in the treatment of constipation should be discouraged. Laxative abuse can aggravate constipation by training the bowel to rely on exogenous stimulation. Laxative use also interferes with the absorption and utilization of essential trace nutrients.

If a patient habitually uses laxatives, the use should be tapered rather than abruptly stopped. People who have become dependent upon laxatives may be resistant to discontinuance of their use. Repeated explanations of the rationale of the high-fiber diet, with encouragement from the physician, may be needed.

In addition to a high-fiber diet, patients should be encouraged not to delay a bowel movement after the need arises. Many people will postpone a bowel movement for hours, waiting until a more convenient time. The longer the feces are in the colon, the less moisture they will contain. The urge to have a bowel movement will eventually subside as a result of stretch-receptor fatigue or stool shrinkage. This can start a vicious cycle, especially if laxatives are used. Appendix D outlines the diet to prevent and treat constipation.

Epilepsy

Will Diet Aid in the Treatment of Epilepsy?

Drugs used in the treatment of epilepsy tend to decrease the absorption of several nutrients. Long-term use of anticonvulsants may lead to the development of rickets and osteomalacia since the metabolism of vitamin D is impaired. For these medications, one multivitamin-with-mineral tablet daily meeting 100% of the RDA is recommended to prevent nutritional complications. If anticonvulsant therapy is not effective in controlling seizures, a ketogenic diet may be helpful. Such a diet requires many hours of teaching by a registered dietitian.

The majority of patients with epilepsy respond to therapy with anticonvulsant drugs. Before anticonvulsant drugs were available it was observed that starvation with ketosis had a favorable effect on epileptic seizures. If a patient does not respond to medications, a ketogenic diet' may be helpful in controlling petit mal seizures. One great disadvantage of this diet is its extremely high fat and very low carbohydrate and protein content making it unpalatable and potentially dangerous, especially for young children. The diet must be supplemented with an aqueous solution of multiple vitamins, calcium, and iron.

The development of medium-chain triglycerides (MCT) and subsequent use in the ketogenic diet allows for a lower fat and a higher protein and carbohydrate intake.² Since MCT's are rapidly absorbed, transported to the liver, and metabolized to ketones, a state of ketosis develops quickly. Ketogenic diets are complicated to plan and teach to a mother who will be preparing the food for her child. Nutritional supplementation is critical for calcium, iron, B-vitamins, and vitamins C, E, and D since the MCT diet does not meet RDAs for

children or adults. There is no guarantee that the diet will alter or prevent seizures. Dietary effectiveness can be determined within 9-21 days. If there is no improvement within that time period, the diet should be discontinued.

Drugs used in the treatment of epilepsy tend to decrease the absorption of several nutrients. Phenytoin sodium (Dilantin) reduces the absorption of calcium, folic acid, B12, B6, vitamin D, and vitamin K. Phenobarbital increases the loss of folic acid, B12, B6, vitamin D, and vitamin K. A multi-vitamin/mineral supplement, equal to the RDA, is recommended for patients on anticonvulsant medications. Especially important is calcium supplementation in order to prevent osteoporosis.

If a decrease in serum folate develops with anticonvulsant therapy, and folic acid is given to correct the serum level, anticonvulsive agents may be less effective in some patients. More research is needed concerning this occurrence.⁵ For patients who use this drug, it would seem wise at the present time not to use the 5 mg therapeutic dose of folic acid, but rather the lower dose of less than 1 mg usually found in one multivitamin-with-mineral tablet daily.

Food Allergy

Does Diet Cause Food Allergy?

Controlled studies have shown that food can trigger sensitivity reactions in susceptible persons. However, 90% of all sensitivity reactions in one study of 132 children were caused by 4 foods: peanuts, nuts, eggs, and milk. The remaining 10% were caused by soy, shrimp, banana, tuna, chicken, and trout. Chocolate, strawberry, and tomato are commonly believed to be "allergic" foods. While there are probably people who do react to these foods, the incidence is certainly much less than is commonly believed. Wheat is another food commonly associated with food sensitivity reactions.

Incidence of food sensitivity in the general population is unknown. Approximately 1% of the population experiences immediate sensitivity reactions to food. Delayed hypersensitivity reactions are more difficult to

document since onset of symptoms may not occur for several days. Clinically significant hypersensitivity is far less common than generally perceived. 1-5

Prognosis of food sensitivity is variable. It is believed by many that food sensitivity in young children disappears with age. However, studies which document this opinion are not available. Results of one study suggested that children whose food sensitivity was diagnosed at an earlier age were more likely to "outgrow" their sensitivity than those who were diagnosed at a later age. The same study indicated that some allergenic foods became non-allergenic sooner than others. Foods to erated sooner in all age groups of sensitive individuals included milk, eggs, and soy. Peanut, fish, shrimp, and walnut continued to produce symptoms for a longer period of time. Several studies of infants and children with cow-milk sensitivity show that most children are able to tolerate cow-milk protein by 2 or 3 years of age. Some individuals sensitive to milk protein may outgrow it, only to redevelop sensitivity later.5

Food sensitivity has been implicated as a causative factor in asthma. One study tested use of a non-antigenic, elemental diet in patients with severe asthma. Researchers found that 9 of 21 subjects improved. Three of the nine subjects improved dramatically. Only one of the 17 control subjects improved. This study suggests that food sensitivities can be a factor in some patients with asthma and that such patients should be tried on an antigen-free diet for 2 to 3 weeks. However, more research is needed before clinical recommendations can be made. Trials of the elemental diet in severe asthmatics should be conducted only under close medical supervision in an in-patient setting.⁷

Several food additives have been shown to provoke asthma. These include metabisulfite (which is a preservative used to enhance the color of salad greens), tartrazine, PD&C Yellow No. 5, and monosodium glutamate (MSG).

Severe asthma develops in susceptible persons 11 to 14 hours after ingestion of chinese food which contains MSG. "Chinese-Restaurant Asthma" can be lifethreatening and difficult to recognize since it occurs many hours after ingestion. In contrast, "Chinese Restaurant Syndrome" is a relatively benign condition which has transient symptoms such as headache, nausea, warmth and tingling, light-headedness, heartburn, and gastric discomfort. The Glutamate Association points out that the number of individuals hypersensitive to



MSG is small. They suggest that reactions occur most frequently when a high concentration of MSG is eaten on an empty stomach.8

A second condition in which food sensitivity may be a factor is irritable bowel syndrome (IBS). One study, which used double-blind food provocation tests, confirmed food sensitivity in 3 of 19 subjects who had IBS, atopic disease, and positive skin tests to common inhalent allergens. The researchers concluded that the possibility of food sensitivity should be considered in IBS when atopic disease is present, since exclusion of the offending food results in marked improvement.

Does Diet Prevent Food Allergy?

Use of breast milk is effective in prevention of food sensitivity reactions in infants. Food sensitivity is common in infants and should be distinguished from lactose intolerance. It has been suggested that breast-feeding should be encouraged for all infants, especially those who have a positive family history of cows-milk ailergy.¹⁰

A lower incidence of cow-milk allergy has been found in children who did not receive cow's milk during the first 6 months of life." Breast-fed babies have also been shown to have a lower incidence of eczema than babies fed cow's milk." The rare breast-fed baby who develops cow-milk allergy likely had a mother who ingested excessively large quantities of cow's milk during lactation.

Breast-feeding does not totally eliminate the potential for development of food sensitivity reactions in allergic families. A baby can be sensitized to foods which are eaten in excessive quantities by the mother. Relief of symptoms occurs when the mother avoids those foods to which her baby is sensitive. 13 Symptoms include colic, vomiting, diarrhea, rhinorrhea, wheezy bronchitis, eczema, diaper dermatitis, and urticaria. Primary foods shown to provoke these symptoms include cow's milk and egg. A much smaller percentage of babies responded to citrus fruit, wheat, and chocolate in the mother's diet. 12

Actually, only trace amounts of a food to which a baby is sensitive reach him/her through breast milk, even though the mother may eat large quantities of the food. This is in contrast to formula-fed sensitive babies who

receive relatively large amounts of antigen (the amount of formula taken by an infant is comparable to a 70-kg man drinking 10 liters of milk/day).

Delayed introduction of solid foods into an infant's diet may also play a role in preventing food sensitivity.

The rate of maturation of the nervous system, intestinal rract, and kidneys should determine when solid foods are added to the diet. During the first three to four months of life an infanr's intestinal tract does not have defense mechanisms developed (such as IgA) for dealing with foreign proteins. Introduction of solid food during this period could increase the risk of developing food sensitivities.

The Committee on Nutririon of The American Academy of Pediatrics recommends that use of solid foods and juices should be delayed until four to six months of age, when the GI tracr is more mature. 15 When solid food is introduced, many suggest that ironfortified rice cereal should be given first. Additional foods should be added one at a time. Use of mixed dishes, such as fruit cobbler baby food or spaghetti, make it difficult to determine which food the infant is sensitive to if a reaction occurs. New foods should be introduced at a rate of approximately one per week. If an allergic response occurs, the offending food should be eliminated from the diet until the infant is older and is able to eat it without symptoms.

Objective diagnosis of food sensitivity and verification of symptoms is critical. Exclusion diets are the most successful form of treatment of food sensitivity. During the acute stage of a food sensitivity reaction in infants when diarrhea is present, all potentially allergenic proteins shou!! be excluded from the diet to avoid further sensitization.

Objective diagnosis of food sensitivity and verification of symptoms are critical. Failure to do this can lead to unnecessary avoidance of foods which could compromise the nutritional status of the patient and delay diagnosis



of potentially serious medical conditions.² Diagnosis of food sensitivity can be complex and may be complicated by the fact that psychological factor: riay cause physical findings similar to those of food sensitivity.¹⁶ Referral to a physician trained in the use of double-blind food challenge is recommended.

Exclusion diets are the most successful form of treatment of food sensitivity. The Care must be taken to provide a diet which is nutritionally complete. This is especially important to promote normal growth in children. Children have developed hypoproteinemia and malnutrition when given inappropriate milk-protein substitutes. Some children allergic to cow's milk are also allergic to soy-based formula. If this is the case, one of the infant formulas which does not contain soy must be used (see Table 26-2). Goat's milk may not be a suitable substitute for cow's milk since there appears to be a cross-reactivity between the two animal proteins.

A list of recommended publications, together with lists of foods containing eggs, milk, and wheat, for information for mothers with food-sensitive children can be found in the section, "Resources for the Patient - Food Sensitivity." The lists may be reproduced as handouts.

During the acute stage of food sensitivity reaction in infants when diarrhea is present, all potentially allergenic proteins should be excluded from the diet to avoid further sensitization. It is not known whether formulas

which contain protein hydrolysates decrease the incidence of multiple protein sensitivities if used during the acute diarrheal state. Formulas which contain mixtures of free amino acids are not recommended. Free amino acids increase formula osmolality (which could trigger additional diarrhea) and compete with other nutrients for transport mechanisms.¹⁷

An infant who is nutritionally compromised or who has diarrhea which is not responsive to oral feeding is a candidate for total parenteral nutrition (TPN). The Prompt initiation of TPN in the failure-to-thrive infant can be crucial to survival. Management of TPN should be done by health professionals trained in the special nutritional needs of infants.

When alternative infant formulas are used, an infant should be challenged periodically with a milk-based formula to determine if the protein sensitivity has been outgrown.¹⁷ If the challenge produces no symptoms, the infant should be placed on a conventional milk-based formula (which is less expensive than alternative formulas).

Drug therapy for food sensitivity patients is controversial. Some recommend use of sodium cromoglycate if the offending food protein cannot be identified or if multiple protein sensitivity prevents an adequate protein intake. 19 Others have suggested that sodium cromoglycate be taken prophylactically before eating a meal which contains unidentified ingredients. 20 Concern has been

Table 26-2	Infant Formula Other Tha	Infant Formula Other Than Cow's Milk			
Source	Protein	Carbohydrate	<u>Fat</u>		
Breast Milk	Lactalbumin	Lactose	Saturated		
Prosc¹ee	Soy	Glucose polymers	Soy oil		
Isomil	Soy	Sucrose/glucose polymers	Corn and coconut oil		
Nutramizer	Protein Hydrolysate	Sucrose/glucose	Corn oil		
Pregestimil	Protein Hydrolysate	Glucose polymers	Long-chain fat Medium-chain fat		

Adapted from Lafshitz, F. and Carrera, E.. "Food Sensitivity and Intolerance Leading to Diarrhea," Clinical Nutrition, 3(1).5-13, 1984.



raised that use of sodium cromoglycate may mask immunologic or biochemical reactions which may have potential long-term side effects.² In addition, use of this drug becomes expensive when taken in the usual dose of 40 to 100 mg 3-4 times per day. Corticosteroids have been used successfully to clear gastro. ntestinal tract symptoms in severe cases.²¹

Gallstones

Does Diet Have a Role in Gallstone Formation?

More must be learned before the role of diet in gallstone formation is clear. A moderate increase in fiber intake may be beneficial; however, there is not enough evidence to recommend a decreased intake of sugar. Additional studies are needed to determine if reduction in meal frequency and prolonged overnight fasting are significant contributors to development of cholelithiasis.

The etiology of gallstone formation remains controversial as does its treatment by diet. Dietary fiber has been found to reduce the cholesterol content of bile, making it less lithogenic. Thus, an increase in dietary fiber has been suggested as both a therapeutic and a preventive measure for persons susceptible to gallstone formation. Cholesterol may crystallize, precipitate, and form stone nuclei if bile is deficient in solvent bile acids. Pomare has shown that bran can increase chenodeoxycholate bile while reducing formation of deoxycholate. This results in a less saturated bile. In vitro, bran has been shown to promote absorption of deoxycholate in the colon. Similar results have been shown by a more recent investigator.

Trowell suggests that excess calorie intake and use of fiber-depleted foods (especially sugar) may be linked to gallstone formation.³ Evidence cited to support this hypothesis is mainly observational and anecdotal and includes the following:

- A clinical association has been shown between gallstones and obesity.
- Some gallstone patients have been documented to have a high energy intake.
- Some obese patients have been shown to have increased cholesterol secretion.

- High energy intakes may be common when fiberdepleted foods are eaten.
- Hamsters have been shown to form gallstones when fed a fiber-depleted carbohydrate (highsugar) diet.
- Refined carbohydrate intake has been shown to decrease bile acid synthesis and reduce the bile acid pool; patients with gallstones have been observed to have a small bile acid pool.
- Deoxycholate suppresses chenodeoxycholate synthesis.

Capron suggests that the overnight fast is significantly longer in younger women (ages 20-35) with gallstones than in women without gallstones. During fasting there appears to be a decreased rate of bile acid secretion from the gallbladder while secretion of cholesterol into the bile continues at the same rate. Reduction of meal frequency and prolonged overnight fasting may thereby increase the chance of gallstone formation in younger women. This relationship was not found in older women. Additional studies are needed to determine if reduction in meal frequency and prolonged overnight fasting are significant contributors to development of cholelithiasis. Suggesting that patients eat 3-4 meals daily, including an evening snack, and advising patients not to fast seem to be prudent advice at the present time.

Hemorrhoids

Does Diet Play a Role in the Prevention of Hemorrhoids?

Currently, there is not enough evidence to conclude that a high-fiber diet will prevent hemorrhoids. A moderate increase in fiber intake to correct constipation would not be harmful and might have a beneficial effect.

There is some evidence that a low-fiber diet may be a factor in the development of hemorrhoids. Trowell suggests that constipation (due to a low-fiber diet) results in straining at stool which raises intra-abdominal pressure and increases the risk of hemorrhoids. Hemorrhoids, in turn, can make constipation worse because patients may be reluctant to defecate.

Evidence which links a low-fiber diet to hemorrhoids is based upon epidemiology. Westernization of the diet



in Africa, India, Pakistan, and the Middle East has been associated with a decrease in fiber intake and an increase in constipation and hemorrhoids.² It should be noted that causal relationships are not appropriate conclusions when evidence is based on epidemiology. No controlled experimental studies have demonstrated that a low-fiber diet causes hemorrhoids.

Results of one double-blind study found that treatment of hemorrhoids with a high-fiber, vegetable bulk agent (Vi-Siblin) resulted in significantly less bleeding and pain at defecation.³ This study lends support to the fiber hypothesis; however, there is not enough evidence for a cause/effect relationship to be established. A moderate increase in fiber intake would not be harmful and, if beneficial in certain patients, should be encouraged. No studies on use of high-fiber foods in the treatment of hemorrhoids have been done.

Treatment of existing hemorrhoids by use of a high-fiber diet has been questioned.³ Other forms of management, such as anal dilatation, rubber-band ligation, and sclerotherapy, have been shown to be effective and have few complications. Some physicians use a high-fiber diet for patients on the waiting list for hemorrhoidectomy or for those who 'efuse invasive anal therapy.

Kidney Stones

Can Diet Prevent the Formation of Kidney Stones?

Diet does not have an effect on the formation of kidney stones in healthy people. Those who are susceptible to kidney stones, however, may be able to prevent stone formation by diet modification.

Diet plays a secondary role in stone formation by influencing conditions in the urinary tract. There is no universal diet which is recommended for therapy of renal stones. Diet must be individualized according to the type of stone and the underlying etiology of the problem. Regardless of the type of stone, fluid intake should be increased to reduce urine concentration of stone-forming substances. Three to four liters of fluid per day are recommended. This should yield a urine volume of at least 2500 cc per 24 hours.

Stone formation is a multi-factorial process. In addition to the effects of fluid and dietary intake, me, abolic conditions such as primary hyperoxaluria and primary hyperparathyroidism, urinary bacterial infections, occupation, immobilization, socioeconomic status, and climate may play a role.¹

Hypercalciuria occurs in many calcium stone formers although not all patients who form calcium stones have this disorder. Calcium restriction is contraindicated in those with hypercalciuria who absorb calcium normally.

Hypercalciuria occurs in many calcium stone formers although not all patients who form calcium stones have this disorder. Some persons with hypercalciuria never develop stones. Four mg/kg body weight/day is considered the upper limit of calcium excretion in normal patients. The following four conditions have been identified which may be responsible for hypercalciuria.

- 1. Hyperparathyroidism is responsible for a relatively small percentage of those who have hypercalciuria. Treatment is usually surgical and does not include dietary therapy.
- 2. Increased absorption of calcium from the gastrointestinal tract can result in hypercalciuria. Diet may aid in prevention of stone formation in these patients. A low-calcium diet (less than 600 mg calcium/day) combined with an increased fluid intake (3-4 liters per day) has been reported to reduce calcium excretion.^{2,3} Not all patients respond to a low-calcium diet with significantly reduced calcium excretion.

A low-calcium diet (less than 600 mg/day) can be achieved by limiting dairy products. Two cups of milk or the combination of ½ cup cottage cheese, ½ cup custard, ½ cup ice cream, and 6 oz. milk would provide less than 600 mg calcium. Appendix E is a suggestion for a low-calcium diet, together with a list of the calcium content of some foods. It may be reproduced as a handout to patients for whom it may be wise to limit dietary calcium to less than 600 mg/day for a short period of time. The diet will be more palatable if the patient is given a list of calcium



values and taught how to include a limited number of dairy products in the diet rather than suggesting avoidance of all of them. Dairy products provide a variety of nutrients such as riboflavin, vitamin D, magnesium, and phosphorus. Total exclusion of dairy foods from the diet is not suggested. The low-calcium diet should be used only in known cases of increased calcium absorption.

- 3. Impaired tubular reabsorption of calcium (calcium leak) results in hypercalciuria. Treatment does not include diet therapy.
- 4. Idiopathic hypercalciuria is responsible for the majority of calcium-containing renal stones. It occurs more often in males than females, usually in the third or fourth decade of life. Calcium oxalate stones are the predominate type of stone found.

Six risk factors have been identified in the formation of idiopathic stones. These include:

- decreased urine volume
- alkaline pH of urine
- increased excretion of calcium
- increased excretion of oxalate
- increased excretion of uric acid
- decreased concentration of crystallization inhibitors¹

Usually more than one risk factor is present before stone formation occurs. Dietary components which may influence urinary risk factors are calcium, oxalate, protein, purines, refined carbohydrate, and sodium.

Patients with idiopathic hypercalciuria are often advised to decrease their intake of calcium. However, studies have shown that calcium intake is not, except in rare cases, the cause of idiopathic stone formation. In addition, restriction of calcium intake during childhood and early adulthood will contribute to osteoporosis in later life. Only those patients who have increased absorption of intestinal calcium might benefit from a low-calcium diet (less than 600 mg/day).

Calcium restriction is contraindicated in those with hypercalciuria who absorb calcium normally. Prolonged low-calcium intake at any age may lead to loss of bone mineral or osteoporosis. A low-calcium diet may increase oxalate absorption and excretion and may result in a lower magnesium and phosphorus intake. A high oxalate,

low magnesium, and low phosphorus intake may increase the risk of stone formation. More research is needed in this area before dietary recommendations are substantiated.

Treatment of hypercalciuria with thiazide diuretics has been effective in lowering urinary excretion of calcium and oxalate. During thiazide therapy, a No-Added-Salt (NAS) Diet is necessary to prevent a high sodium intake from counteracting the effects of the medication. In addition, the patient should be encouraged to supplement the diet with foods rich in potassium to avoid hypokalemia. Patients who do not obtain enough potassium through diet may need supplemental potassium. Appendix F offers suggestions for a No-Added-Salt and high-potassium diet, together with a list of foods suitable for increasing potassium intake.

A diet high in animal protein has been shown to increase the urinary excretion of calcium, oxalate, a...d uric acid. Excessive excretion of uric acid raises the undissociated uric acid and sodium hydrogen urate levels which act as nuclei for calcium oxalate crystallization.

It may be beneficial to limit protein intake to 0.8 gm/kg of ideal body weight if excessive excretion of uric acid is documented. This will provide adequate protein for the otherwise healthy adult while reducing the risk of a high protein intake. When uric acid levels are elevated, alloparinol is the drug of choice to lower uric acid levels. Treatment of uric acid stones is discussed later in this section.

Intake of refined carbohydrates may increase the risk of calcium oxalate stone formation in those who have an exaggerated insulin response to glucose load. Since insulin promotes calcium excretion, this subgroup of patients may have higher levels of calcium in the urine. Theoretically, a low simple sugar diet might benefit these patients by reducing calcium excretion. However, no studies have documented the value of this restriction, thus it would be premature to recommend such a diet.

Sodium intake may be related to calcium stone formation. A direct relationship has been found between dietary sodium intake (80 to 200 mEq/day) and urinary calcium excretion in those who form calcium oxalate stones. It has been suggested that idiopathic hypercalciuria may be provoked by a high sodium intake and that sodium restriction may be beneficial, however, this hypothesis has not been tested.



Seven to eight percent of all renal stone patients in the United States, and 10-25% of patients with gout have uric acid stones. Restriction of excessive protein intake can help lower uric acid levels. Restriction of purine intake by limiting foods rich in purines is possible.

Seven to eight percent of all renal stone patients in the United States and 10-25% of patients with gout have uric acid stones. Men are more frequently affected than women. Abnormally acidic urine is an important risk factor in the development of stones. Dehydration aggravates the problem by reducing urine volume, thus favoring urine acidity. For prevention of stone recurrence, an oral alkali (sodium bicarbonate) to raise the urine pH to greater than 6.5 is beneficial. Increased fluid intake to yield a urine volume of at least 3 liters/day is also necessary.^{2,4}

Restriction of excessive protein intake can help lower uric acid levels (see section on Prevention of Gout Attacks.) Recent research suggests that certain dietary purines have a greater effect on urinary uric acid excretion than others.² Dietary instructions regarding purine intake are in Appendix B.

Restriction of purine intake by limiting foods rich in purines is possible (see Table 26-1 for the purine content of foods). Some health professionals prefer to use allopurinol therapy instead of dietary purine restriction since medical therapy may be more reliable and the diet may be unpalatable for some patients.

Oxalate stones are less common. Prevention for patients with hyperoxaluria includes limiting oxalate intake, increasing fluid intake, and avoidance of excessive amounts of fats and vitamin C.

Those patients who have certain intestinal diseases (Crohn's disease, celiac sprue) or have had intestinal bypass surgery or bowel resection have an increased risk for development of hyperoxaluria and stones. Oxalate intake is not a factor in normal subjects since less than 10% of oxalate excreted is derived from the diet.

The following guidelines are helpful for control of stone formation in those who have acquired hyperoxaluria:

 Limit dietary oxalate intake to 40-50 mg/day. Refer to The Low Oxalate Diet Book for oxalate tables and an explanation of the diet. This booklet is an excellent patient education tool and may be obtained from the General Clinical Research Center, University Hospital, 225 Dickinson St., San Diego, CA 92103.

It is not recommended that a list of "foods to avoid" be given since oxalate is found in many foods and such a diet would be difficult to follow. A more reasonable approach is to provide oxalate tables (such as those found in *The Low Oxalate Diet Book*) so that favorite foods can be included in the diet if possible.

- Avoid vitamin C supplements or megadoses. Vitamin C is metabolized to oxalate and can contribute to excessive oxalate excretion.
- Increase fluid intake to keep the urine dilute.
- Avoid foods high in fat. Those who have small bowel disease may not absorb fat as efficiently as normal. Unabsorbed fat forms calcium complexes. This prevents oxalate from precipitating with calcium and being excreted in the feces, thus allowing more oxalate to be absorbed.
- Consider use of calcium supplements if oxalate excretion remains elevated. One gm calcium/day may be suggested for patients who are not hypercalcemic. Calcium supplements increase fecal loss of oxalate and decrease its absorption. When used for this purpose, calcium is also poorly absorbed.¹⁰

Cystine stones comprise only a small percentage of all renal stones found in the United States. Although diet may be prescribed, it does not correct the disorder. Maintenance of a dilute urine (3-4 liters/day) and use of sodium bicarbonate or other alkaline sub-tance to achieve a urine pH of greater than 7.5 can aid in management.

Migraine Headaches

Can Diet Trigger Migraine Headaches?

Some foods have been identified as migraine triggers in susceptible persons. Patients may benefit from eliminating foods which contain



tyramine and phenylethylamine, monosodium glutamate, and alcohol. Patients with migraines should be cautioned not to take supplemental vitamin A and to avoid hypoglycemia.

Certain foods and substances have been identified which have the potential for acting as migraine triggers in susceptible persons. Non-food migraine triggers have also been identified and include stress, use of oral contraceptives, fatigue, menstruation, and glaring light.

A defect in the degradative pathways of monoamine oxidase (MAO) substrates (tyramine and phenylethylamine) may trigger migraine in some people. This results in an excessive release of norepinephrine, elevation of blood pressure, and headache. Patients with migraines may benefit from avoiding foods rich in tyramine such as aged cheeses (cheddar, brick, mozarella, mycelia, boursault, romano, stilton, and gruyere), fermented sausages, sour cream, broad beans, pickled herring, caplin, ale, beer, and chianti wine. Similar foods which do not contain tyramine and therefore do not need to be avoided include cream cheese, cottage cheese, processed cheese, yogurt, fruits, vegetables, and fresh meats. Chocolate contains phenylethylamine. Other amines, such as viz octopamine (found in citrus fruit), and certain phenolated compounds may be responsible for migraine. Further research is needed to clarify the role of these substances.

Many migraine patients have been advised to restrict monosodium glutamate (MSG) because it has been shown to cause headache in approximately 38% of those individuals with "Chinese-Restaurant Syndrome" (see the section on Food Allergy). MSG produces a generalized vasomotor response which can cause headache. It is not clear whether MSG can trigger migraines. Many Chinese foods, as well as soy sauce, contain MSG.

Alcoholic beverages have been associated with migraines.² Non-specific vasodilator effects of alcohol and its chemical components may be involved. Some practitioners advise migraine patients to drink diluted vodka slowly if they desire an alcoholic beverage.³ Diluted vodka contains fewer congeners than other alcoholic beverages. Wine contains histamine, a vasodilator, and two MAO substrates, tyramine and phenylethylamine, thus, some wines lead to migraine in certain patients. While not all migraine patients have symptoms as a result of use of alcohol, it might be halpful to eliminate alcohol from the diet temporarily to determine if it is a triggering factor.

Vitamin A toxicity can trigger migraines. Six patients taking megadoses of vitamin A (25,000 IU/day) were reported to develop migraine headaches several days to several weeks following initiation of supplementation. Headaches subsided after supplementation was discontinued, and plasma vitamin A levels returned to normal.

Some migraines are triggered by hypoglycemia, which affects the tone of cranial blood vessels. Fasting can produce relative hypoglycemia and trigger migraines. Several researchers have successfully used the diet for hypoglycemia to treat hypoglycemia-induced migraines. This diet includes six meals per day each of which is low in simple sugar and contains protein. To avoid overdiagnosis of reactive hypoglycemia, it should be remembered that a significant number of normal people have low postprandial blood glucose levels. This condition is described as a "transitional low blood glucose state of no clinical significance."

Some evidence suggests that sodium and fluid retention is associated with migraine. However, treatment with diuretics does not prevent their regular occurrence. More research is needed before a conclusion can be made. To late, evidence which implicates sodium has been anecdotal.

One case report documents that sodium nitrite, found in cured meats (bacon, hot dogs, salami), induced migraine in a 58-year old man. However, these results have not been reported in other individuals, and elimination or restriction of cured meats in the diets of all migraine sufferers is not warranted.

Identification of foods which trigger migraines is difficult. The placebo response often occurs with changes in therapy and the possibility of spontaneous cure complicates diagnosis. Symptoins appear over a broad range of time, from one hour to seven days, and last an average of two to three days. Use of skin tests or IgE antibodies has not been reliable in predicting causative foods.¹

Specific foods which have been suggested as potential migraine-associated all gens include cow's milk, eggs, chocolate, citrus fruits, wheat, tea, coffee, pork, beef, grapes, nuts, legumes, corn, cane sugar, yeast, pineapple, coconut, and cola drinks. It is not recommended that migraine patients be given a list of foods to avoid which includes every food implicated as a migraine trigger.



Such a list could lead to anxiety and become a psychological stressor, resulting in more migraines. 1

The food/symptom diary is a helpful tool to identify food intake patterns related to migraines. The patient records food and beverage consumption and times of headache onset. In addition, the physician should interview the patient to determine if any foods are suspected of being a migraine trigger. It is also wise to find out it the patient has been taking any vitamin supplements (particularly vitamin A) or if there have been any recent periods of fasting (hypoglycemia).

Once foods have been identified which are potential migraine triggers, a double-blind test can be performed to confirm the findings. A powdered form of the suspected food can be loaded into capsules and compared with the ingestion of capsulized lactose for 3 weeks. Both the patient and physician should be blind to the contents of the capsules so that bias does not alter the results of the challenge.

Patients who do not describe a clear case of food as being a trigger of migraines can follow an elimination diet to rule out diet as a factor. The elimination diet is unpalatable and is not nutritionally complete; therefore, it should be used for only short periods of time and only under qualified supervision. Foods allowed include distilled water, lettuce, cauliflower, carrots, boiled potatoes, baked potatoes, cottage cheese, chicken, corn oil, olive oil, and distilled white vinegar. If headaches continue while the diet is being followed, diet is ruled out as a migraine factor. If headaches cease, foods should be returned to the diet one at a time on a weekly basis. Frequency and severity of headaches should be recorded. Obviously this process is time-consuming and requires a motivated patient. Once the offending foods have been identified they should be avoided.

Recently, advertisements in newspapers have suggested that headaches caused by food allergy can be diagnosed by use of blood analysis. This test, the Cytotoxic Test, supposedly identifies a food allergen each time white blood cells "burst open and die" after being mixed with a food substance. Actually, the test reflects the osmotic pressure of the food and has nothing to do with a food's potential as a migraine trigger. Unfortunately this test has been publicized on a number of nationwide talk shows, and many lay people may be duped into believing its nonsense.

Multiple Sclerosis

At present there is not sufficient evidence to justify dietary manipulations either as a preventive measure or as part of management of MS.

Several studies have reported that diet may play a role in multiple sclerosis (MS). In 1970, Swank¹ reported evidence that a low-fat diet may retard the disease process and reduce incidence of attacks. The diet contains 10 grams of saturated animal fat and 40 to 50 grams of polyunsaturated oil daily. This diet is low in protein (as the fat intake is restricted), iron, B-complex vitamins, and trace minerals.

Milk² and wheat³ are reported to have an influence in the treatment of MS. Millar and associates⁴ suggested that mixtures of vegetable oils containing linoleic or oleic acid may be an advantageous treatment, the rationale being based on evidence that linoleic acid inhibits the responsiveness of lymphocytes. If it is assumed that lymphocytic responsiveness is directly related to the development of the lesion of MS, this observation could be promising. The summaries from these reports suggest that further evidence is needed to justify dietary changes either as a preventive measure or as part of management of MS.⁵ Your patients should have a well-balanced diet containing a variety of foods and calories to maintain a normal weight.

Psoriasis

What is the Role of Diet in Psoriasis?

At this time there are no recommendations for the dietary intervention in patients with psoriasis.

No clinical benefits have been demonstrated from use of a low-protein diet for psoriasis. Observations made during World Wars I and II are conflicting about the benefits of food deprivation for this illness. Interpretation of the data is difficult since a variety of factors changed during wartime, not just food intake.

Douglass suggests that citrus fruits, nuts, corn, milk, coffee, soda, tomatoes, and pineapples should be eliminated from the diet of patients with psoriasis. This recommendation is based on subjective observation of



six people. It would not be appropriate for physicians to suggest avoidance of these foods since evidence is limited and the nutritional contribution of many of these foods is beneficial.

Research is currently being done on the effects of arachidonic acid in psoriasis. The hypothesis has been made that a diet rich in linoleic acid (a precursor of arachidonic acid) might improve the condition of psoriasis patients.² Linoleic acid is found in ve. table seed oils, corn oil, safflower oil, and evening primrose oil. Another hypothesis suggests that supplementation of the diet with eicosapentaenoic acid (EPA), found in cold water fish, could be beneficial.² Further studies are needed before the value of these hypotheses will be known.

A vitamin A derivative, etretinate, is currently under study for use in psoriasis therapy. Early studies have shown that the drug appears to inhibit psoriasis only as long as therapy is continued. Etretinate is a potent teratogen and currently is not available for clinical use.

Sexual Potency

Do Certain Foods Increase Sexual Potency?

Throughout the centuries a number of foods and herbs have been promoted to increase sexual potency. None of these claims has been substantiated, however such ideas are popular and will concinue to exist in folklore. Until further research is done, this area should be considered fad in nature. Some "aphrodisiacs" are listed below.

Varicose Veins

Does Diet Play a Role in the Prevention of Varicose Veins?

Currently, there is not enough evidence to conclude that a high-fiber diet will prevent varicose veins. A moderate increase in fiber intake to correct constipation would not be harmful and might have a beneficial effect.

There is limited evidence that a low-fiber diet may be a factor in the development of varicose veins. Evidence which links a low-fiber diet to varicose veins is based on epidemiology. Westernization of the diet in Africa, India, Pakistan, and the Middle East has been associated with a decrease in fiber intake and an increase in constipation and varicose veins. It should again be noted that cause-effect relationships are not appropriate conclusions when evidence is based on epidemiology; therefore, advocating changes in diet is not appropriate.

Alcohol	While alcohol may stimulate interest in sexual activity, it can impair sexual performance. In men, alcohol promotes increased breakdown of testosterone by increasing production of the enzyme responsible for testosterone degradation.
Ginseng	Sometimes called jen shen, this root from China is also known as the "man plant." It is considered a fertility drug and aphrodisiac in Asian folklore. An ancient medical book from India, the Atharva Veda, states that "Ginseng causes an aroused man to exale fire-like heat."
Lean meat	At one time meat was thought to "stir animal passions."
01:	

Olives	Olives were said to be an aphrodisiac because their shape resembled that of testicles.
Oysters	Oysters were said to be an aphrodisiac because their shape resembled that of testicles.

Raw Eggs In many societies raw eggs symbolize fertility and new life.

Spirula The substance is said to have "inagical" properties, but this has not been

substantiated.

Vitamin E deficiency does interrupt normal reproduction in experimental animals,

but this has not been demonstrated in humans. Excess vitamin E will not increase

fertility.

Rhinocerous Horn This substance is also said to have "magical" properties, but this is not confirmed.



Table 26-3	Summary of Select	ed Ailments Where Dietary Treatment is Effo	ective in Health Promotion	
Ailment	Who Is at Risk?	Clinical Manifestations to Monitor	When to Monitor	Dietary Treatment
Acne	Adolescents Teenagers College Students	Papules and pustules that may appear on face and upper trunk. Poor appetite.	If there is a family history of acne. If dietary intake indicates excessive use of sweets, cola drinks, et Stressful period in life.	 Common offenders that may aggravate acne in some patients are chocolate, nuts, sweets, cola drinks, alcohol, and fatty foods. A decreased consumption of these foods may be recommended. Vitamin A supplements should not be used since toxicity may occur before beneficial results are obtained. Reduce intake of fried foods, pastries, cream sauces, gravies, etc. Encourage a variety of foods from the four food groups to mean patient's nutritional needs. Suspect foods should be eliminated one at a time.
Constipation	Elderly Bed ridden Laxative abusers	Stool consistency and frequency	During pregnancy. When fluid intake is decreased. If dietary intake is low in fiber. Increased use of laxatives.	To increase fiber content of daily diet to 30-60 gm/day, use the following foods: 1. Whole grain breads — 3 or more slices. 2. Whole grain cereals — 2 cups. Fruits — ½ cup or more, 2 to 3 times of fresh or frozen fruits, including pulp and peel (apples, peaches, etc.). 3. Vegetables — ½ cup or more, 2-3 times of fresh, frozen, or canned vegetables. 4. Water — increase intake to 6-8 glasses/day. 5. Avoid foods which contribute to constipation (varies with individual).



Summary

Diets for common ailments are often controversial, and more research is needed to prove the efficacy of nutrition for such conditions. Most of the information in lay journals, magazines, and newspapers concerning the use of various diets and claims for one specific nutrient in curing and preventing illness is based on ancedotal evidence and not scientific research.

Physicians should use patience in explaining why nutrition information in the popular media may be misleading. Patients tend to believe what they read since they do not have the knowledge to effectively evaluate what they read (see Module 20, Decoding Fad Diets, for

suggestions on how to evaluate popular nutrition claims).

Millions of dollars are spent yearly by people in the belief that some specific nutrient or diet will cure them of all their problems. Physicians should be aware of the possible dangers involved in using nutrients and certain non-nutrients (choline, lecithin, etc.) for the prevention or treatment of diseases. Until further scientific studies are reported, it is recommended that an adequate diet using the basic four food groups, with the caloric level planned according to the age and activity of the individual, be used to assure an intake of a variety of foods.

Evaluation

It would be meaningless at this point to ask you to regurgitate any or all of the information related to the 14 ailments examined in this unit of study. The evaluation for this module, therefore, is designed to help you both organize the information and to assess the information related to nutritional issues associated with ailments not included.

On the next page you will find a form entitled, "Ailment-Related Nutrition Information." This is a blank form which asks seven questions of importance whenever ailment-specific nutrition information is being examined. This form can be copied so that it may be used to summarize information related both to the illnesses examined in this module and to other diseases as you see fit. So that you may practice using this form, take the time to select several of the ailments covered in this module and answer for each ailment the seven questions on the form provided. When you have answered these questions, you may check the accuracy of your information simply by rereading the appropriate section on the specific illness you selected in this unit. Additionally, if at all possible, it would be worth the effort to discuss this information with colleagues.

You may find that you wish to complete similar forms on all the ailments within this module and on other ailments for which you find yourself dealing with nutrition-related issues.



Ailment-Related Nutrition Information

Anment-Related Nutrition information
Ailment: 1. This ailment can be best described (to the best of our current knowledge):
2. The following nutrition information is felt to be true regarding this ailment:
3. The following nutrition information is regarded to be false concerning this ailment:
4. The following nutrition recommendations can be made regarding the prevention of this ailment.
5. The following nutrition recommendations can be made regarding the treatment:
6. If further information is needed concerning this ailment and related nutrition information, the following are appropriate resources for me:
7. If further information is needed by patients concerning this ailment and nutrition-related information, the following are appropriate resources for the patient:



References

Acne

- 1. Michaelsson, G.: "Diet and Acne." Nutrition Reviews, 39(2):104-106, 1982.
- 2. Shaleta, A.K., Cunningham. W.J., et al.: "Isotretinoin Treatment of Acne and Related Disorders: An Update." Journal of the American Academy of Dermatologists 9(4):629-38, 1983.
- 3. Valentic, J.P., Elias, A.W., and Weinstein, G.D.: "Hypercalcemia Associated with Oral Isotretinoin in the Treatment of Severe Acne." Journal of the American Medical Association 250(14):1899-1900, 1983.
- 4. Baer, M.T., et al.: "Acne in Zinc Deficiency." Archives of Dermatology, 114:1093, 1978.

Alzheimer's Disease

- 1. Schneck, M.K., et al.: "An Overview of Current Concepts of Alzheimer's Disease." American Journal of Psychiatry, 139:170, 1982.
- 2. Thal, L.J.: "Current Concepts of the Pathogenesis of Senile Dementia of the Alzheimer Type." Geriatric Medicine Today, 3:86, 1984.
- 3. Lione, A.: "The Reduction of Aluminum Intake in Patients with Alzheimer's Disease," in Liss, L., (ed.), Clinical Implications of Aluminum Neurotoxicity, Pathotox Publications, 1983.
- 4. Levick, S.W.: "Dementia from Aluminum Pots" New England Journal of Medicine, 303:164, 1980.
- 5. Underwood, E.J.: Trace Elements in Human and Animal Nutrition, 4th edition. New York: Academic Press, 1977.
- 6. Flinn, G.A.: "The Loneliness of Millions: Alzheimer's Diseases." Health Spectrum, 1:4, 1983.
- 7. Boyd, W.D., et al.: "Clinical Effects of Choline in Alzheimer Senile Dementia." *Lancet*, 2:711, 1977.
- 8. Fervis, S.H., et al.: "Long-Term Choline Treatment of Memory Impaired Elderly Patients." Science, 205:1039-40, 1979.
- 9. Sullivan, E., et al.: "Psysostigmine and Lecithin in Alzheimer's Disease," in Corkin, S., et al. (eds.), Alzheimer's Diseases: A Report of Progress in Research. New York: Raven Press, 1983.

Arthritis

- 1. Khachadurian, A.K.: "Hyperuricemia and Gout: An Update." American Family Physician, 24(6):147-48, 1981.
- 2. Kawsari, B., et al.: "Assessment of the Diet of Patients with Rheumatoid Arthritis and Osteoarthritis." Journal of the American Dietetic Association, 82(6):547-59, 1983.

Constipation

ļ.

1. Stratton, J.W. and Mackeigan, J.M.: "Treating Constipation." American Family Physician, 25(6):139-42, 1982.

Epilepsy

- 1. Lasser, J.L. and Brash, M.K.: "An Improved Ketogenic Diet for Treatment of Epilepsy." Journal of the American Dietetic Association, 62:281, 1973.
- 2. Signore, J.M.: "Ketogenic Diet Containing Medium-Chain Triglycerides." Journal of the American Dietetic Association, 62:285, 1973.
- 3. Krause, M.V., and Mahan, L.K.: Food, Nutrition, and Diet Therapy, 6th edition. Philadelphia, PA: W.B. Saunders Co., 1979, p.659.



- 4. Govoni, L.E., and Hates, J.E.: Drugs and Nursing Implications, 3rd edition. New York: Appleton-Century-Crofts, 1978.
- 5. Reynolds, E.H.: "Folate and Epilepsy," in Bradford, H.F. and Marsden, C.D. (eds.). *Biochemistry and Neurology*, New York: Academic Press, 1976, p.247-252.

Food Allergy

- 1. Bock, S.A.: "The Natural History of Food Sensitivity." Journal of Allergy Clinical Immunology, 69,(2): 173-177, 1982.
- 2. Bock, S.A.: "Food Sensitivity: A Critical Review and Practical Approach." American Journal of Diseases of Children, 134: 973-83, 1980.
- 3. Perlman, F.: in N. Calsimpooles (ed.), *Immunological Aspects of Foods*. Westport, Conn. Avi Publ Co., Inc., 1977, pp. 279-316.
- 4. Golbert, T.M.: "Food Allergy." Journal of the Medical Society of New Jersey, 77:895-899, 1980.
- 5. Rapp, D.J.: "Milk Allergy-From Birth to Old Age." Allergy, 14:120, 1974.
- 6. Hoj, L., et al.: "A Double-Blind Controled Trial of Elemental Diet in Severe, Perennial Asthma." Allergy, 36:257-62, 1981.
- 7. Podell, R.N.: "Food Allergy and Asthma." Postgraduate Medicine, 73(5):287-89, 1983.
- 8. Glutamate, 5775 Peachtree-Dunwoody Road, Suite 500-D, Atlanta, Georgia 30342.
- 9. Bentley, S.J., et al.: "Food Hypersensitivity in Irritable Bowel Syndrome." *Lancet*, ii:295-97, 1983.
- McClenathan, D.T., and Walker, W.A.: "Food Allergy. Cow Milk and Other Common Culprits." Postgraduate Medicine, 72(5):233-39, 1982.
- 11. "Food Sensitivity." Dairy Council Digest, 54(2):7-11, 1983.
- 12. Glaser, J., and Johnstone, D.E.: "Prophylaxis of Allergic Diseases in the Newborn." Journal of the American Medical Association, 153(7):620-2, 1953.
- 13. Grulle, C.G., and Sanford, H.N.: "The Influence of Breast and Artificial Feeding on Infantile Eczema." *Journal of Pediatrics*, 8:223-25, 136.
- 14. Gerrard, J.W.: "Allergy in Breast Fed Babies in Ingredients in Breast Milk." Annals of Allergy, 42(2):69-72, 1979.
- 15. Stevenson, D.K., et al.: "Pulmonary Excretion of Carbon Monoxide in the Human Infant as an Index of Bilirubin Production IV. Effects of Breast-feeding and Caloric Intake in the First Postnatal Week." *Pediatrics*, 65:1170, 1980.
- 16. Pearson, D.J., and Keithe, J.B.: "Diet and Illness." Lancet, 8336:1259-61, 1983.
- 17. Lifshitz, F., and Carrera, E.: "Food Sensitivity and Intolerance Leading to Diarrhea." Clinical Nutrition, 3(1):5-13, 1984.
- 18. Sinatra, F.R., and Merrit, R.J.: "Iatrogenic Kwashiorkor in Infants." American Journal of Diseases of Children, 135:21-23, 1981.
- 19. Gerrard, J.A.: "Oral Cromoglycate: Its Value in the Treatment of Adverse Reactions to Food." Annals of Allergy, 42:135, 1979.
- 20. Lessof, M.H.: "Food Allergy Fact and Fiction." The Practitioner, 227:573-82, 1983.
- 21. Katz, A.J., et al.: "Eosinophilic Gastroenteritis in Childhood: Basic for Allergic Etiology." Journal of Allergy Clinical Immunology, 61:157, 1978.



Gallstones

- 1. Pomare, E.W., and Heaton, K.W.: "Alteration of Bile Salt Metabolism by Dietary Fiber." British Medical Journal, 4:262-4, 1973.
- 2. Kritchevsky, D.: "Metabolic Effects of Dietary Fiber." Western Journal of Medicine, 130:123-27, 1979.
- 3. Trowell, H.: "Definition of Dietary Fiber and Hypothesis that it is a Protective Factor in Certain Diseases." American Journal of Clinical Nutrition, 29 (April): 417-27, 1976.
- 4. Capron, J.P.: "Meal Frequency and Duration of Overnight Fast: A Role in Gallstone Formation?" British Medical Journal, 283:1435, 1981.

Hemorrhoids

- 1. Trowell, H.: "Definition of Dietary Fiber and Hypothesis that it is a Protective Factor in Certain Diseases." American Journal of Clinica. Nutrition, 29 (April):417-27, 1976.
- 2. Burkitt, D.P., Trowell, H.C. (eds.): Refined Carbohydrate Foods and Disease: The Implications of Dietary Fiber. London: Academic Press, 1975, pp. 335-42.
- 3. Mosegaard, F., Nielson, M.L., et al.: "High Fiber Diet Reduces Bleeding and Pain in Patients with Hemorrhoids." Diseases of the Colon and Rectum, 25:454-56, 1982.

Kidney Stones

- 1. Robertson, W.G., Peacock, M.: "Stone Disease of the Urinary Tract." The Practitioner, 225:961, 1981.
- 2. Van Reen, R.: "Nutrition and Urinary Stones." The Professional Nutritionist, 12(3):11-14, 1980.
- 3. Smith, L.H., et al.: "Nutrition and Urolithiasis." New England Journal of Medicine, 298:87, 1978.
- 4. "Diet and Urolithiasis." Dairy Council Digest, 54(5):25-30, 1983.
- 5. Heaney, R.P., et al.: "Calcium, Nutrition, and Bone Health in the Elderly." American Journal of Clinical Nutrition, 36 (suppl):986, 1982.
- 6. Smith, L.H.: "Medical Treatment of Idiopathic Calcium Urolithiasis." Kidney, 16:9-15, 1983.
- 7. Robertson, W.G., et al.: "The Effect of High Animal Protein Intake on the Risk of Calcium Stone Formation in the Urinary Tract." *Clinical Science*, 57:285, 1979.
- 8. Rao, P.N., et al.: "Are Stone Formers Maladapted to Refined Carbohydrate?" British Journal of Urology, 54:575, 1982.
- 9. Muldowney, F.P., et al.: "Importance of Dietary Sodium in the Hypercalciuria Syndrome." *Kidney International*, 22:292, 1982.
- 10. Ney, D.M., et al.: The Low Oxalate Diet Book. San Diego: General Clinical Research Center, University Hospital, 1981.

Migraine Headaches

- 1. Perkin, J.E., and Hartje, J.: "Diet and Migraine: A Review of the Literature." Journal of the American Dietetic Association, 83(4):459-63, 1983.
- 2. Medina, J.L., and Diamond, S.: "The Role of Diet in Migraine Headache." Headache, 18:31, 1978.
- 3. Brainard, J.B.: Control of Migraine. New York: W.W. Norton and Co., 1979.
- 4. Raskin, N.H., and Appenzeller, O.: "Headache," in Smith, L.D. (ed.), Major Problems in Internal Medicine Vol. 19. Philadelphia: W.B. Saunders Co., 1980.



- 5. Byer, J.A., and Dexter, J.D.: "Hypoglycemic Migraine." Missouri Medicine, 72:194, 1975.
- 6. Lance, J.W.: Mechanisms and Management of Headache, 3rd edition. Boston: Butterworth, 1978.
- 7. Henderson, W.R., and Raskin, N.H.: "Hot-Dog Headache: Individual Susceptibility to Nitrite." Lancet, 2:1162, 1972.
- 8. Rowe, A.H., and Rowe, A.: Food Allergy: Its Manifestation and Control and the Elimina.: on Diets: A Compendium. Springfield, IL: Charles C. Thomas, 1972.

Multiple Sclerosis

- 1. Swank, R.L.: "Multiple Sclerosis—Twenty Years on Low Fat Diet." Archives of Neurology, 23:460-474, 1970.
- 2. Agranoff, B.W., and Goldberg, D.: "Diet and the Geographical Distribution of Multiple Sclerosis." *Lancet*, 2:1061, 1974.
- 3. Jellinek, E.H.: "Multiple Sclerosis and Diet." Lancet, 2:1006, 1974.
- 4. Millar, J.H., et al.: "Double Blind Trial of Linoleate Supplementation of the Diet in Multiple Sclerosis." *British Medical Journal*, 1:765, 1973.
- Olson, W.H.: "Diet and Multiple Sclerosis." Postgraduate Medicine, 59:219, 1976.

Psoriasis

- 1. Douglass, J.M.: "Psoriasis and Diet." (Letter), Western Journal of Medicine, 133:450, 1980.
- 2. Voorhees, J.: "Leukotrienes and Other Lipocyganese Products in the Pathogenesis and Therapy of Psoriasis and other Dermatoses." Archives of Dermatology, 119(7):541-47, 1983.

Varicose Veins

1. Burkitt, D.P., Trowell, H.C. (eds.): Refined Carbohydrate Foods and Diseases: The Implications of Dietary Fiber. London: Academic Press, 1975, pp. 335-42.

Resources for Physicians

- 1. Anderson, J.A., and Sogn, D.D. (eds.): Adverse Reactions to Foods. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402 (Stock No. 017-044-00045-1).
- 2. ACSH News and Views. The American Council on Science and Health, 1995 Broadway, New York, New York 10023.
- Contemporary Nutrition. Production Manager, General Mills, Inc., P.O. Box 1112, Dept. 65, Minneapolis, MN 55440. A free newsletter concerning controversial nutrition issues.
- 4. Ferguson, A.C.: "Food Allergy." Progress in Food and Nutrition Science, 8 (1/2):77-107, 1984.
- 5. Goodhart, R.S., and Shils, M.E.: Modern Nutrition in Health and Disease, 6th Ed. Philadelphia: Lea and Febiger, 1980.
- 6. Lasswell, A.B., Roe, D.A. and Hochheiser, L.: Nutrition for Family and Primary Care Practitioners. Philadelphia: George F. Stickley Co., 1984.
- 7. Nutrition and the M.D. PM, Inc., 9931 Van Nuys Blvd., Van Nuys, CA 91405.
- 8. Nutrition News. National Dairy Council, 6300 North River Road, Rosemont, IL 60018-4233. A free newsletter that reviews timely nutrition topics.



- 9. Nutrition Reviews. The Nutrition Foundation, Office of Education and Public Affairs, 888 Seventeenth St., N.W. Washington, DC. A monthly publication containing reviews of current nutrition research.
- 10. Olson, R.E. (ed.): Present Knowledge in Nutrition, 5th Ed. Washington, DC: The Nutrition Foundation, Inc., 1984.
- 11. Schneider, H.A., Anderson, C.E., Coursin, D.B.: Nutritional Support of Medical Practice, 2nd Ed. Philadelphia: Harper and Row, 1983.
- 12. Tufts University Diet and Nutrition Newsletter. P.O. Box 2465, Boulder, CO 80322.

Resources for Patients

Alzheimer's Disease

Mace, N.L., and Rabins, P.V.: The 36 Hour Day: A Family Guide to Caring for Persons with Alzheimer's Disease. Baltimore: Johns Hopkins University Press, 1982.

Powell, L.S., and Courtice, K.: Alzheimer's Disease: A Guide for Families. Reading, MA: Addison-Wesley, 1983.

Reisberg, B.: A Guide to Alzheimer's Disease for Family. New York: The Free Press, 1983.

Alzheimer's Disease and Related Disorders Association, Inc., 360 North Michigan Avenue, Chicago, IL 60601. This is the national organization that has other publications available to family members.

Kelly, W.E. (ed.): Alzheimer's Disease and Related Disorders. Charles C. Thomas, 2600 South First St., Springfield, IL 62717, 1984.

Pajk, M.: "Alzheimer's Disease: Inpatient Care." American Journal of Nursing, 216, 1984.

A Guide to Books on Arthritis

Diet Books Recommended

The following books provide reliable information about arthritis according to the Arthritis Foundation.

The Arthritis Book—A Guide for Patients and Their Families, by Ephramin P. Engleman, M.D., and Milton Silverman, Ph.D. Painter Hopkins Publishers, 1979. Dr. Engleman is one of the world's best known arthritis experts. This book is an informative and readable guidebook on the major forms of arthritis, the problems they cause, and how sufferers and their families can deal with them.

Arthritis: A Comprehensive Guide, by Dr. James F. Fries. Addison-Wesley Publishing Company, 1979. The author has included practical and easy-to-read advice on managing the diseases and solving problems. The book clears up many common misunderstandings.



The Arthritis Helpbook—What You Can Do For Your Arthritis, by Kate Lorig, R.N., Ph.D., and James F. Fries, M.D. Addison-Wesley Publishing Co., 1980. This book is a companion book to Arthritis: A Comprehensive Guide. Topics covered include exercise, drugs, self-help aids, and diet/nutrition.

Food and Arthritis, by Gaynor Maddox. Taplinger Publishing Company, 1979. Information contained in this book is reliable and still applicable even though the book is older. Rheumatoid arthritis, osteoarthritis, and gout are discussed, and good eating habits for each type of arthritis are suggested. The book includes sample menus.

Recommended Pamphlet for Patients

Arthritis, Diet and Nutrition: Facts to Consider. Available from local arthritis foundation or The Arthritis Foundation, 3400 Peachtree Road, N.E., Atlanta, Georgia 30326.

Diet Books-Not Recommended

These books promote diet therapy for which there is no valid scientific evidence. Each diet proposed is different from the others, and each claims to have "the answer" for relieving symptoms and curing the disease. If any of these diets were scientifically proven they would be prescribed and arthritis would be a disease of the past. It is curious that each year brings a new diet breakthrough with the same empty promises.

Dr. Atkins' Nutrition Breakthrough. How to Treat Your Medical Conditions Without Drugs, Robert C. Atkins, M.D.

Childers' Diet to Stop Arthritis-The Nightshades and Ill Health, Norman F. Childers.

There is a Cure for Arthritis, Paavo O. Aerola, M.D.

Arthritis and Common Sense and Good Health and Common Sense, Dan Dale Alexander.

Victory for Arthrais, Rasmus Alsaker, M.D.

An 80-Year Old Doctor's Secrets of Positive Health, William Brady, M.D.

A Doctor's Proven New Home Cure for Arthritis, Girarid W. Campbell, D.O.

You Can Stay Well and Let's Get Well, Adelle Davis, A.B., M.S.

The Arthritic's Cookbook, Colin H. Donz, M.D. and Jane Banks.

Arthritis and Folk Medicine, D.C. Jarvis, M.D.

Arthritis, Food and Arteries in Joint Disease, Victor Manley, D.O.

Arthritis Discovery and Arthritis is Not Forever, Robert Liefmann, M.D.

Natural Relief for Arthritis, Carol Keough.

Arthritis Can be Cured-A Layman's Guide, Bernard Aschner, M.D.

Arthritis, Nutrition, and Natural Therapy, Carlson Wade.

Bees Don't Get Arthritis, Fred Malone.

Pain-Free Arthritis, Dvera Berson with Sander Roy.



Food Sensitivity

Recommended Books, Cookbooks, and Booklets About Food Sensitivity All About Food Allergy, by Faye M. Dong. George F. Stickley Co., 1984.

Coping with Food Allergy, by Claude A. Frazier. Quadrange/The New York Times Book Co., 1974.

Baking for People with Food Allergies. U.S. Department of Agriculture, Home and Garden Bulletin No. 147. Request order form from Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402.

Allergy Recipes. Request order form from the American Dietetic Association, 430 North Michigan Avenue, Chicago, Illinois 60611, 1969. Contains recipes for wheat, milk-, and or egg-free foods, 64 pages.

Wheat-, Milk-, and Egg-Free Recipes. Consumer Services Department, Quaker Oats Co., Merchandise Mart, Chicago, Illinois 60654, 34 pages (free).

Good Recipes to Brighten that Allergy Diet. Best Foods, CPC International, Inc., Dept. AB, Englewood Cliffs, New Jersey 07632 (free). Contains 2- receipes for food which contain no wheat, egg, and/or milk.

125 Great Recipes for Allergy Diets. Request order form from Good Housekeeping Bulletin Service, 959 Eighth Avenue, New York, New York 10019.

Rudoff, C.: The Allergy Gourmet. Menlo Park, CA, Prologue Publications, 1983.

Foods Which Contain Wheat

Read the ingredient list to determine if the food contains wheat flour (white, whole wheat, or graham).

Ale Farina

Baked goods Gravy thickened with flour

Beer Ice cream cones
Bran Malted milk
Breaded foods Pastas, noodles

Breads Pretzels

Cereals containing wheat Sauces thickened with flour

Chili-con-carne Semolina

Chowders Soups containing flour or noodles

Crackers Wheat germ

uencio Wile

Desserts



Foods Which Contain Milk

Read the ingredient list to determine if the food contains milk in some form: caseinate, sodium caseinate, casein, curds, lactalbumin, lactoglobulin, lactose, and whey.

Baked goods containing milk Bread and pastry containing milk

Butter and margarine

Cheese

Cream pies

Cream sauces

Cream soups

Ice cream, custard, pudding, sherbert

Non-dairy products such as Pream, Coffee-mate, Cremora, Coffee-Rich Yogurt

Note: The above list is not appropriate for lactose-restricted diets—only for those who are sensitive to milk protein. See module 13 in the *Nutrition in Primary Care Series*, "Dietary Management in Gastrointestinal Diseases," for information about the lactose-restricted diet.

Foods Which Contain Egg

Read the ingredient list to determine if the food contains egg protein: albumin, ovoglobulin, livetin, ovomucin, ovomucoid, powdered or dried egg, egg yolk, vitellin, or ovovitellin.

Baked goods, most commercial

Baking powder, some contain egg white

Beverages: ovaltine, root beer

Bisquick, pancake flour

Bread foods

Candy: chocolate creams, fondants, marshmallows, nougats

Cold cuts

Custards, puddings

Egg noodles

Ground meat dishes

Hollandaise sauce

Ice cream, sherbet

Mayonnaise, tarter sauce

Pasta

Pretzels

Salad dressings, some

Sausages

Souffles

Wine, coffee, bouillon that has been clarified with egg white

"Egg Replacer," an egg-free powder made by Ener-G-Foods may be used in baking



Appendix A

Dietary Treatment for Acne

Certain foods may aggravate acne in some individuals. Chocolate, nuts, sweets, cola drinks, alcohol, and fatty foods may be common offenders. Suspected foods should be eliminated one at a time. Total avoidance of such items is probably not indicated, but a decreased consumption may be helpful. A variety of foods selected from the four major food groups is recommended to meet the nutrient needs of the individual.

Vitamin A supplements should not be used, since toxicity may occur before beneficial results are achieved. Foods considered high in fat content are:

Bacon Butter

Chocolate Cold cuts

Cream

Cream cheese

Cream sauces Fat of meat

Fish canned in oil

Fried foods

Gravies Margarine Mayonnaise

Nuts

Oils used in cooking

Pastries

Peanut butter

Salad dressings

Sausage

Skin of chicken

Weiners

Whole milk

Whole milk cheeses



Appendix B

Dietary Treatment for Gout

A moderate restriction in purines may be useful in the treatment of gout. Restricting foods which have a high purine content is recommended. In addition, a variety of foods should be recommended to meet the nutrient needs of the individual. Excessive protein intake should be avoided.

*Foods

Amount To Include Daily

Milk

2-3 cups

Chese

1 or 2 ounces

Eggs

1 or 2

Lean meat, fish, or poultry

4 to 6 ounces

Vegetables

4 servings, 1/2 cup each including potato, green leafy, or yellow

vegetables, plus other vegetables.

Fruit

2 to 3 servings, 1/2 cup each as desire 1, including 1/2 cup citrus fruit

Breads and Cereals

4 to 6 servings as desired

Fat

2 to 5 servings, depending on calorie allowance.

Water

6-8 glasses daily

**Foods Highest in Purines to be Omitted

Anchovies

Liver

Bouillon

Mackeral

Broth Gravies Meat extracts

Gravies Sardines
Kidney Sweet breads

*A lapted from Krause, M.V., and L.K. Mahan, Food Nutrition and Diet Therapy, W.B. Saunders Co., Philadelphia, PA 1979, p. 550. **lbid, page 551.



Appendix C

A	approximate Fiber in Commonly Used	Food Portions
	Breads & Cereals	Fruits
Group I*	white flour plain white bread, crackers, French bread	strained clear fruit juices grapefruit, fresh
Less than 1.5 g DF**	pastas white rice Cheerios Rice Krispies Special K plain cooked refined cereals	
Group II	brown rice most cold cereals*** pancakes	applesauce avocado cantaloupe
1.6-1.99 g DF	oatmeal cornmeal	plums, canned
Group III	wheatflakes	most canned, cooked, and fresh fruits*** banana
2.0-2.99 g DF		peach
Group IV	whole wheat and rye flour, bread, etc. cereals with dried fruits	raisins oranges, fresh figs, fresh
3.0-3.99 g DF	or nuts granola grapenuts	dates prunes pears blackberries
Group V	shredded wheat ryebread ryekrisp	raspberries, fresh and canned
4.0-5.99 g DF	whole wheat bread	
Group VI	bran, bran cereals, bran muffins, etc.	figs, dried persimmon, fresh currants, dried
Greater than 6.0 g		apples, fresh blackberries, fresh

^{*}Foods which are generally not restricted for Minimal Residue Diets, but for which actual figures for dietary fiber or residue were unobtainable are. cereal beverages, offee, tea, bouillon, broth, margarine, butter, vegetable oil, shortening, whipped cream, eggs, tender meats, poultry, fish, strained vegetable juices, and seasonings.

dietary fiber = 5 x crude fiber for breads, cereals, and grains

dietary fiber =3.5 x crude fiber for legumes, nuts, seeds, and vegetables

dietary fiber =4 x crude fiber for fruits



^{**}Dietary Fiber. Based on value for dietary fiber when available, or estimated from crude fiber as follows.

Appendix C (cont)

Approximate Fiber in Commonly Used Food Portions			
Vegetables	Legumes, Nuts, Seeds	Miscellaneous	
asparagus beets peppers, cooked	most texturized vegetable protein entrees split peas, cooked lima beans	jam jelly marmalade	
asparagus, cooked cucumber, peeled lettuce tomato, canned			
most canned or cooked vegetables without peelings or seeds*** cucumber, unpeeled, lettuce, turnips (fresh), tomatoes (fresh)	smooth peanut butter cashews		
white potato with skin beet greens wax beans winter squash pumpkin, canned	pinto beans brown beans	popcorn	
sweet potatoes broccoli corn, green peas parsnips	kidney beans white beans lentils soybeans, most nuts***		
artichoke winter squash	chick peas baked beans filberts sunflower seeds		

Dictary fiber includes all the indigestible substances in food, crude fibet is the residue remaining after treatment with boiling sulfuric acid, sodium hydroxide, water, alcohol, and ether. While some sources state that crude fiber is approximately 50% of dietary fibet (1), comparison of foods for which both figures are available yielded the above estimates of 20-30%. Pottion sizes and data from which the above chart was calculated are listed in the following table and in "Nutrient Composition of Vegetable Protein Foods." Most vegetable portions equal ½ cup; all legume portions are ½ cup; portions of nuts and seeds are 60 g.

From Heath, M.K. (ed.) Seventh Day Adventist Dietetic Association Diet Manual--Including a Vegetarian Meal Plan, 6th edition. Loma Linda, CA. Seventh Day Adventist Dietetic Association, 1982, pp. 148-149. Used with permission from the Seventh Day Adventist Dietetic Association.



33

^{***}Unless specified elsewhere.

Appendix D

Dietary Treatment for Constipation

The dietary treatment for prevention and treatment of constipation include (1) eating a variety of foods from the four basic food groups, and (2) increasing intake of foods high in fiber. The following recommendations will meet an individual's nutrient needs:

Foods to Use Daily	<u>Amount</u>
Whole Grain Breads	3 or more slices of whole wheat, raisin, cracked wheat, rye, corn, or pumpernickel bread or muffins. Ry-Krisp Crackers.
Whole Grain Cereals	1/2 cup to 1 cup of high-fiber cereals such as 100% Bran, Bran Flakes, Bran Buds, All Bran, Shredded Wheat, Raisin Bran, Grape-Nuts, Oatmeal, Mother's Oats, grits.
Fruits	1/2 cup two to three times daily of fresh, frozen, or canned fruit including pulp and peel; the following fruits are highest in fiber: raspberries, strawberries, blackberries, rhubarb, prunes, plums, pear with skin, oranges, nectarines, figs, currants, apple with skin, banana, melon.
Vegetables	1/2 cup two to three times daily of fresh, frozen, or canned (raw or cooked); the following vegetables are highest in fiber: broccoli and other greens, Brussels sprouts, corn, peas, potatoes with skin, sweet potatoes, yams, popcorn, tomatoes, pumpkin, winter squash, carrots, lettuce, mushrooms.
Meat, eggs, poultry, fish, nuts, legumes	4 to 6 oz meat or meat substitute; legumes high in fiber include beans (navy, white, pinto, baked, garbanza, brown, kidney) and peas; nuts are also high in fiber as are chick peas and soybeans.
Milk	3 to 4 cups of milk, buttermilk, yogurt, or cheese as substitute; dairy products are low in fiber.
Water	6 to 8 cups water.

Low-Calcium Diet (600 mg Ca)

This diet is restrictive in calcium and should not be used for an extensive period of time. The patient is advised to include foods from the four basic food groups daily (meat group, milk group, fruit and vegetable group, and grain group). Fluid intake should be at least 6 to 8 cups daily. The following foods are highest in calcium, and to limit intake to 600 milligrams daily, the following table should be used. This will allow the patient to plan these foods into the diet without exceeding 600 mg. daily.

Dairy Products		Vegetables	
Milk, average Cheese	280 mg/8 oz	Greens (kale, broccoli, collard, mustard,	
American, Cheddar	200 mg/oz	turnip, dandelion),	
Cottage	100 mg/½ cup	average	100 mg/1/2 cup
Swiss .	260 mg/oz	Beans, canned in sauce	138 mg/cup
Velveeta	120 mg/3/4 oz	Soybeans	65 mg/½ cup
Spread	80 mg/Tbsp.	Miscellaneous	
Ice Cream, ice milk	100 mg/½ cup	Cornbre: 1	95 mg/2" square
Soft serve ice cream	140 mg/½ cup	Tortilla	60 mg each
Pudding, Custard	130 mg/½ cup	Waffles	60 mg/4" x 4"
Yogurt		Tofu	150 mg/4 oz piece
Plain	300 mg/cup	Molasses, blackstrap	137 mg/Tbsp
Fruited	240 mg/cup	Pizza	150 mg/5" wedge
Half-and-Half	15 mg/Tbsp	Macaroni and Cheese	350 mg/cup
Cream, light	30 mg/oz	Almonds	160 mg/½ cup
Seafood		Pecans, walnuts, peanuts	
Clams, canned	5.5 mg/½ cup	Milk chocolate	65 mg/oz
Clam chowder	240 mg/cup		
Mackeral, canned	260 mg/½ cup		
Oysters	70 mg/4 medium		
Oyster Stew	300 mg/cup		
Salmon	220 mg/½ cup		
Sardines	50 mg/1" x 3" fish		

A suggested meal plan could be as follows:

Breakfast	Lunch	Dinner
1/2 cup orange juice 3/4 cup Rice Krispies 1 slice toast 1 tsp margarine 1/2 cup milk Coffee Non-dairy creamer	3 oz hamburger/bun Lettuce Catsup Mustard ½ cup coleslaw ½ cup fruit cobbler ½ cup milk	3 oz roast pork Baked potato Margarine, as desired ½ cup green beans 1 slice bread ½ cup fresh fruit 8 oz milk Coffee Non-dairy creamer



35

Dietary Treatment for Hypercalciuria

No-Added-Salt and High-Potassium Diet

When thiazide diuretics are used in the treatment of hypercalciuria, a No-Added-Salt and high-potassium diet is recommended.

A No-Added-Salt diet (4,000 mg of sodium) and high-potestium diet (5000 to 6000 mg) may be achieved by:

- 1. Use of a variety of foods selected from four food groups prepared with a small amount of salt (1 teaspoon maximum per day).
- 2. No salt added to the food at the table.
- 3. Avoid foods high in sodium such as ham, bacon, salt pork, chipped beef, corned beef, smoked meats and fish, luncheon meats, frankfurters, sausage, canned meats, meat extracts, salted broth, sauerkraut, canned tomatoes and canned tomato juice, salted nuts, salted potato chips, salted pretzels, snack crackers, TV dinners, commercially packaged entrees or starches or vegetables, and seasoning salts.
- 4. Increase intake of fruits, fruit juices, and vegetables to provide a high intake of potassium as in the following examples. Each of the following supplies approximately 500 mg of potassium:

Foods Suitable for Increasing Potassium Intake

100ds Juliable for increasing 1 of assium inta-
6
1 medium
1 cup
. 10
1 cup
1½ cups
1 cup
1 ¹ / ₄ cups
1 medium or 10 fries or ² / ₃ cup mashed
1 large or ½ cup
6 oz
¹/₂ cup
¹/₂ cup
3/4 cup
1/3 cup
4 whole
10
2 medium
2 cups
3∕4 cup
1 cup
3∕4 cup
1½ cups
3" square
½ cup



Index

Acne	Hemorroids 11-12
dietary effects 2	Hypercalciuria 12-13;36
vitamin and minerals, therapeutic use 2	Kidney stones 20(t)
Alzheimer's disease 3	dietary modifications 12-14
Calcium-restricted diet 35	formation 12
Constipation 19t*	oxalate stones 14
diet 6-7	renal stones 14
fluid intake 7	Migraine headache 20t
laxatives 7	allergens 15-16
dietary treatment 34	foods implicated 15
Egg, foods which contain 29t	vitamins and minerals 15
Epilepsy	Milk, foods containing 29t
dietary control 7	Multiple sclerosis 16
drug therapy 7-8	Osteoarthritis 4
Fiber, content in foods 32-33	Psoriasis 16
Food allergies	Sexual potency 17
asthma 8-9	Varicose veins 17
drug therapy 10-11	Vitamin A
incidence of 8	acne 2
prevention 9	migraine headache 15
treat ment 10	Wheat, foods which contain 28t
Gallstones 11	Zinc 3
Gout 4;31	
Infant	*a page number followed by a "t" ind. ates a table; an
food allergies 9	"f" refers to a figure.
formula 10t	



37

Some Abbreviations Used in the Nutrition in Primary Care Series

ATP adenosine triphosphate

c cup

cc cubic centimeter
CNS central nervous system

FDA Food and Drug Administration

gm gram

IBW ideal body weight IU International Units

kcal kilocalorie kg kilogram lb pound lg large

MCV mean corpuscular volume MDR minimum daily requirement

med medium

µg microgram

mEq milliequivalent

mg milligram

MJ megajoule

ml milliliter

oz ounce

RDA Recommended Dietary Allowances

RE retinol equivalents

sl slice sm small Tbsp Tablespoon

TPN total parenteral nutrition

tsp teaspoon

USDA United States Department of Agriculture

