Childhood schizophrenia is a rare but serious disorder with complex symptoms that affect children and their families. Childhood schizophrenia was once the term applied for all childhood psychoses, including autism and mood disorders, but more recently researchers have distinguished childhood schizophrenia from other disorders. There are differing views on the continuity of childhood schizophrenia into adult schizophrenia and on similarities between childhood and adult schizophrenia. A number of symptoms that some very young children with schizophrenia may exhibit have been identified by age, including lethargy (newborns); perseveration (3-12 months); and hypotonia and phobias (2 years). While many of these symptoms may continue through the elementary years, thinking, language, balance, and motor functions also may become disordered. The time of onset of the symptoms of schizophrenia seems to be an important variable in the course and outcome of the illness. The diagnostic criteria of childhood schizophrenia are the same as those for adult schizophrenia. Evidence has suggested that both biological and environmental factors play a role in the development of schizophrenia, although direct causes are still in question. Multimodal treatment programs, based on the child's individual cognitive level, have been suggested in working with children and adolescents. Much more methodologically sound research is needed in the areas of treatment, symptomatology, diagnosis, and etiology of childhood schizophrenia. (Contains 19 references.) (NB)
Overview of Childhood Schizophrenia

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Abstract

Childhood schizophrenia was used to classify all childhood psychoses at one time. However, researchers have now distinguished childhood schizophrenia from other disorders such as autism and mood disorders. This paper overviews some of the current research on the development of symptomatology, the etiology, and the treatment of childhood schizophrenia and stresses the need for more research in these areas in order to better serve this population.
Overview of Childhood Schizophrenia

Schizophrenia raises many questions in the mental health arena (Tsiantis, Macri & Maratos, 1986). The term "schizophrenia" has never been clearly defined (Tsiantis et al, 1986) and the etiology is still unknown (McClellan & Werry, 1992). Classification and diagnosis are other controversial issues in schizophrenia (Tsiantis et al, 1986). Yet, childhood schizophrenia poses even more complex issues (Tsiantis et al, 1986). The purpose of this paper is to overview the current research on the symptomatology, etiology, diagnosis, and treatment of childhood schizophrenia.

Research in the area of childhood or early adolescent schizophrenia has been very limited for several reasons (McClellan & Werry, 1992). Childhood schizophrenia became an umbrella term for all childhood psychoses in the early 1960s (McClellan & Werry, 1992; Tsiantis et al, 1986) and as a result the research done at this time actually explored autism and other psychotic disorders as well as schizophrenia (McClellan & Werry, 1992). McClellan and Werry (1992) also note that many of these studies have had methodological problems. Retrospective data collection is used frequently which may distort actual phenomena (Ambelas, 1992). Small sample sizes are likely (Watkins, Asarnow & Tanguay, 1988) due to the rarity of childhood schizophrenia and the difficulty in finding subjects (Burd & Kerbeshian, 1987; McClellan & Werry, 1992; Tanguay & Cantor, 1986).

Schizophrenia in children is unusual. A prevalence study in North Dakota revealed that .19 per 10,000 children age 2-12 years and .35 per 10,000 boys in the same age range were
diagnosed with schizophrenia (Burd & Kerbeshian, 1987). This prevalent rate, however, may not reflect the rate in the entire United States as the similarity of North Dakota's population to that of the U.S. is questionable. The authors suggest that the criteria of the DSM-III when applied to children may not be sensitive enough to diagnose schizophrenia (Burd & Kerbeshian, 1987). Much more work is needed in the area of childhood schizophrenia, despite the fact that childhood schizophrenia rarely occurs (Tanguay & Cantor, 1986). Tanguay and Cantor (1986) point out that this illness can lead to severe disruptions in the lives of children and their parents and therefore continuing the research in this area is extremely important.

Early research attempted to identify objective criteria for diagnosing childhood schizophrenia (Tsaintis et al, 1986). Later Rutter and others (cited in Tsiantis et al, 1986) began questioning the relationship between intelligence and childhood psychosis; autism and schizophrenia; and childhood and adult schizophrenia. In 1973 Chapman and Chapman (cited in Schneider & Asarnow, 1987) found that although schizophrenia is associated with mild, general intellectual deficits, the mean IQ over several studies was within the normal range. Rutter (cited in Tsiantis et al, 1986) established that autism is clinically different than childhood schizophrenia and now this distinction has been fairly well documented (Asarnow, Tanguay, Bott & Freeman, 1987; Tsiantis et al, 1986). However, there have been differing views on the continuity of childhood schizophrenia into adult schizophrenia.

Now the question remains, "Is there a difference between childhood and adult
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schizophrenia?" Kolvin and Rutter (cited in Tsiantis et al. 1986) have argued that early and late childhood psychosis are fundamentally different in terms of age of onset, symptom patterns, family and social background, cerebral dysfunction and intelligence and therefore, early and late onset of psychotic symptoms deserve diagnostically distinct categories. On the other hand, other studies have revealed that children and adults with schizophrenia show the same limitation in their intellectual functioning suggesting that the disorder may be similar in both children and adults (Asarnow et al, 1987). Others have also demonstrated the similarity of symptoms in adults and children with schizophrenia (McClellan & Werry, 1992). Yet, this issue remains unanswered.

Symptoms

Evidence of the development of symptoms in childhood schizophrenia and the course of the disease has been overlooked (Watkins et al. 1988). Few researchers have systematically described the symptom development of childhood schizophrenia according to specific age groups (Cantor, 1982; Watkins et al, 1988) probably because of the rarity of this disease. It may be hard to identify children with schizophrenia as they are generally not diagnosed until later stages of childhood. Kolvin (cited in Watkins et al, 1988) noted that the onset of psychosis was most frequent either after the age of ten years or before 30 months.

Cantor (1982) is a psychiatrist who has worked with psychotic children doing therapy and research for a great many years. She has observed these children and has described a comprehensive list of symptoms that some children with schizophrenia may exhibit. Because
of her background in child psychiatry and her direct observation of these symptoms, the symptoms she has proposed will be described.

In the newborn, symptoms may be absent or the infant may appear lethargic and "floppy" (Cantor, 1982). The infant may sleep a lot, feed poorly, fail to grow and gain weight, and consequently may end up becoming sick or hospitalized. Hypotonia or poor muscle tone may appear in preschizophrenic infants so that they hang limply or become stiff when held or cuddled. On the other hand, the preschizophrenic infant may be fearful and irritable from the very beginning and unable to bear being touched or held. Other symptoms which may be present include sleep disturbances and remarkable visual attentiveness.

From three months to one year, the infant may become increasingly sensitive to sensory stimulation. Cantor (1982) gives the example of an infant only allowing the mother to touch him/her or an infant being unable to tolerate the rough textures of clothing or blankets. During this stage the infant may exhibit perseveration or the tendency to become absorbed in an activity. Cantor (1982) describes this as the most striking and alarming symptom of the preschizophrenic child. Similarly, the infant may begin to withdraw and only attend to selected aspects of the environment. These infants may become "picky" eaters and offer unusual resistance to trying new foods. The introduction of anything new becomes more and more difficult.

Hypotonia may appear again by the age two. Cantor (1982) has observed that a majority of children who develop symptoms of schizophrenia before the age of three or four
do not seem to have any motor impairments until the age of two years. Walking late, drooling excessively, eating slowly, and resisting foods that are difficult to chew may be signs of childhood schizophrenia. Children may begin to explore the environment but their tendency to perseverate begins to interfere with learning. Sleep disturbances and wandering during the night are often found in preschizophrenic children. The child may also experience early phobias and begin to babble. Furthermore, the bond between the child and the parents becomes increasingly strained (Cantor, 1982).

By the age of two or three, some of the most severe symptoms include lethargy, poor eye contact, and constant preoccupation with a limited number of objects (Cantor, 1982). These children may be unpredictable, show a disturbance in affect, have poor judgement, and exhibit continued hypersensitivity as well as problems in peer relationships and play behavior (Cantor, 1982). Anxiety may begin in addition to phobias and a majority of these children will be delayed in their language development (Cantor, 1982; Perlmutter, Greenhill, Chambers & Kestenbaum, 1989).

Mahler (cited in Cantor & Kestenbaum, 1986) states that these children have not passed through the developmental stage of separation/individuation. These children have not been able to view themselves as separate individuals. Furthermore, they may come to experience the world as a hostile and scary place (Cantor & Kestenbaum, 1986). Depression seems to be quite common for schizophrenic children, perhaps as a reaction to the unsafe world (Asarnow & Ben-Meir, 1988). In addition, these children become focused entirely on themselves and
their experiences leaving them unable to show empathy for others (Tsiantis et al., 1986).

Many of the same symptoms persist throughout the nursery and elementary school years (Cantor, 1982). In addition, the thinking becomes disordered as it is recognized through the child's ability to speak (Cantor, 1982; McClellan & Werry, 1992). Disordered language may also appear as evidenced by the repetition of phrases and songs, misarticulations and mispronunciations (Perlmutter et al., 1987; Tanguay & Cantor, 1986) and involuntary vocalizations (Caplan, Tanguay & Szekely, 1987).

Disordered balance and motor function are common symptoms of schizophrenia (Cantor, 1982). Sensory and motor disturbances have been found by others to be associated with schizophrenia (Tanguay & Canton, 1986; Cantor & Kestenbaum, 1986). An abnormal gait (Cantor, 1982) and poor gross and fine motor skills may also be evidence of the severely affected schizophrenic child (Cantor & Kestenbaum, 1986).

Psychotic symptoms such as hallucinations and delusions are necessary for the diagnosis of schizophrenia (APA, 1987). Hallucinations and systematic delusions have been found consistently before the age of 18 (McClellan & Werry, 1992) but these symptoms are much less common in the child under nine years of age (Cantor, 1982; Tsiantis et al., 1986). In fact, in a group of children between the ages of 0 and nine years, none of these children were found to have hallucinations or delusions (Tsiantis et al., 1986).

Cantor (1982) suggests that these psychotic experiences for young children are extremely rare but they can and do occur as she has witnessed them in her clinical experience.
Researchers have also attempted to differentiate between the symptoms of autism and schizophrenia in an experimental study comparing the symptoms of children with schizophrenia and children with the onset of autism in infancy which later became schizophrenia (Watkins et al, 1988). A problem with this study was the overrepresentative and perhaps misrepresentative sample of autistic children. Non-retarded children were selected, but Rutter (cited in Watkins et al, 1988) points out that most autistic children are retarded. Therefore the generalizability of these results is questionable. For example, if these results were used as the basis for diagnosis between autism and schizophrenia, caution should be used as this sample is not representative of the more common autistic population.

The results revealed that children who develop autism in infancy and then later develop schizophrenia continue to exhibit symptoms of autism until the onset of schizophrenia (Watkins et al, 1988). The primary symptoms then became incoherence, loosening of associations, flat or inappropriate affect and other symptoms of thought disorders (Watkins et al, 1988). The results of the children diagnosed with only schizophrenia indicated that they were less disturbed during infancy but that they also had a significant number of developmental delays in infancy (Watkins et al, 1988). These children had motor developmental problems and poor coordination (Watkins et al, 1988). These symptoms are similar to those reported by Cantor (1982). The differentiation of symptoms between these two groups eventually faded into the same symptoms by the age of six when schizophrenia was diagnosed in the autistic group (Watkins et al, 1988). These results seem to suggest that there is a differentiation between
very early onset psychosis which may be more like autism and later onset psychosis which would be childhood schizophrenia.

**Onset of Schizophrenia**

The time of onset of the symptoms of schizophrenia seems to be an important variable in the outcome and course of the illness. Onset before the age of 13 years has been called very early onset and is associated with an insidious onset and chronic outcome (McClellan & Werry, 1992). Negative symptoms such as inattention, flat affect, apathy, lack of interest in social relationships are more evident in the very early onset schizophrenia which has been related to a more chronic progression of the illness (McClellan & Werry, 1992).

Onset between the ages of 13 and 18 years has been termed early onset (McClellan & Werry, 1992). The outcomes and course of the illness at this age seems to have the same possibility of outcomes as adult schizophrenia which could be anywhere from an acute onset with a better prognosis to a slow onset with poorer outcomes (McClellan & Werry, 1992). However, Cantor and Kestenbaum (1986) state that poor outcomes do not always have to be associated with childhood schizophrenia. Assessing and diagnosing children according to their level of functioning is important in the treatment of the schizophrenic child (McClellan & Werry, 1992).

**Diagnosis**

The diagnostic criteria of childhood schizophrenia is the same as the criteria for adult schizophrenia (Asarnow et al, 1987; Burd & Kerbeshian, 1987; McClellan & Werry, 1992).
Psychotic symptoms such as delusions, persistent hallucinations, incoherence or loosening of associations, catatonic behavior and flat or inappropriate affect; or alternatively the presence of either bizarre delusions or prominent hallucinations must be present for at least one week (McClellan & Werry, 1992). The child must show a deterioration in social, occupational and self-care functioning below the highest previously obtained level or through the failure to achieve expected levels (McClellan & Werry, 1992). The disturbance must be present for at least six months and any organic factors must be ruled out (McClellan & Werry, 1992). Furthermore, other disorders such as autism and mood disorders must be ruled out as well (McClellan & Werry, 1992).

Diagnosing schizophrenia in children, however, can be more difficult than diagnosing adults (McClellan & Werry, 1992; Perlmutter et al, 1989). For instance, children with a very early onset of schizophrenia typically have a lifelong history of developmental and personality abnormalities (McClellan & Werry, 1992). Difficulties become apparent when the deterioration occurs over a period of years because the illness does not become markedly obvious (McClellan & Werry, 1992). Also, children's hallucinations are not always schizophrenic, so it is important to review their clinical relevance (McClellan & Werry, 1992; Perlmutter et al, 1989). Perlmutter et al (1989) stress the importance of neuropsychiatric tests in diagnosis because mere conversation with children may not accurately reflect their internal thinking processes and neuropsychiatric tests help to distinguish formal thought disorders from central language disorders.
Another difficulty in diagnosis is differentiating childhood schizophrenia from other childhood psychoses or disorders (Caplan et al., 1987; McClellan and Werry, 1992). Diagnoses that should also be considered include pervasive developmental disorders or autism, schizotypal or schizoid personality disorders, mood disorders, schizoaffective disorders, dissociative disorders and organic disorders (McClellan & Werry, 1992). For instance, subacute sclerosing panencephalitis (SSPE) is an organic psychoses caused by an infectious agent (Caplan et al., 1987). The symptoms exhibited in this disorder include a progressive deterioration and mental changes that eventually end in dementia (Caplan et al., 1987). These symptoms can be easily confused with schizophrenia (Caplan et al., 1987).

The direct cause of schizophrenia is still unknown and as a result, diagnosis is made solely on the basis of clinical judgements (McClellan & Werry, 1992). Therefore, being aware of the potential problems in diagnosing childhood schizophrenia is essential (McClellan & Werry, 1992). Considering diagnoses as tentative and using follow-ups can help therapists in making the most accurate diagnosis (McClellan & Werry, 1992).

**Etiology**

Genetic factors have been studied for their role in the development of schizophrenia (Perlmutter et al., 1989; Tanguay & Cantor, 1986; Tsiantis et al., 1986). Yet Shields, Heslon, and Gottesman (cited in Tsiantis et al., 1986) note the uncertainty that still exists regarding the mode of transmission of schizophrenia. A direct biological gene or link has never been identified (Tsiantis et al., 1986).
The genetic studies are now beginning to lean toward a position of etiological diversity. Kety (cited in Tsiantis et al, 1986) has suggested that adoption studies give credibility to this position. However, Carlier (cited in Tsiantis et al, 1986) notes that many of these adoption studies have had crucial methodological flaws and direct genetic cause cannot be inferred from these studies. Therefore, the debate about the etiological cause continues.

Most researchers have recognized a link between genetics and schizophrenia by studying children of schizophrenic parents (Tanguay & Cantor, 1986). For example, a sample of high-risk children born to schizophrenic mothers had an increased incidence of neurosensory and neuromotor deficits (Tanguay & Cantor, 1986). This correlation may suggest that neurosensory and neuromotor deficits are linked to schizophrenia, but before this conclusion could be made, a comparison of normal children's neurosensory and neuromotor skills should be measured to see if there is a difference between the normal and schizophrenic children's abilities. Again direct cause cannot be established from these studies but correlations can be acknowledged.

Further research has explored the area of neuropsychology (Asarnow, Sherman & Strandburg, 1986). Normal children have been matched on age and intelligence with a group of schizophrenic children (Asarnow et al, 1986). A group of younger normal children were also compared to children with schizophrenia (Asarnow et al, 1986). The overall results indicated that the children with schizophrenia were experiencing an information processing impairment which was independent of the general level of intellectual skills (Asarnow et al,
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1986). This impairment has been linked to very different patterns of both general and selective central nervous system activations related to the control of information acquisition and information processing (Asarnow et al, 1986). Asarnow et al (1986) have also linked the neocortex, the ascending reticular activating system, and the corticofugal connections with the reticular formation and the limbic system to the mediating activities of the brain.

Actual areas of the brain have been eventually linked to schizophrenia in other research studies. Unusual responses are identified and then related to specific areas of the brain (Asarnow et al, 1986; Erwin, Edwards, Tanguay, Buchwald & Letai, 1986). However, after these areas of the brain have been identified, further studies comparing the differences between normal and schizophrenic individuals are needed to establish actual differences in the biological structures. In fact, in one study blood platelet size, number, and serotonin concentration levels were compared among autistic, schizophrenic and normal individuals because hyperserotoninemia had been reported to occur in individuals with schizophrenia (Giller, Yuwiler, Freeman & Ritvo, 1988). Yet, no differences were found.

Dopamine, a neurotransmitter in the brain, has also been linked to schizophrenia (McClellan & Werry, 1992; Perlmutter et al, 1989). The dopamine theory of schizophrenia suggests that there is an excess of dopamine in which there is either an elevated release or supersensitive postsynaptic receptors (Perlmutter et al, 1989). The use of neuroleptic medications has been used to support this theory because they block dopamine and decrease the overstimulation (McClellan & Werry, 1992; Perlmutter et al, 1989). However, to make a
conclusion about the direct relationship between dopamine and schizophrenia, direct measures should be used to make this statement. The use of neuroleptics is an indirect measure of the dopamine theory.

There has been a great deal of research in the area of adult schizophrenia but much less in the area of childhood schizophrenia (McClellan & Werry, 1992). Many of the assumptions of childhood schizophrenia are drawn from the research on adults (McClellan & Werry, 1992). Schizophrenia in children and adults includes the same symptoms and uses the same criteria for diagnosis and therefore it seems logical to assume that there are similar mechanisms and causes in both childhood and adult schizophrenia. However, Rutter and Kolvin (cited in Tsiantis et al, 1986) have provided evidence to suggest that schizophrenia at different ages of onset really is not the same and continuous illness. Consequently, the research on adults with schizophrenia may provide erroneous conclusions regarding childhood schizophrenia. Much more research is needed in this area.

Social factors have also been postulated in the etiological hypotheses of childhood schizophrenia. Previous research has focused on parent-child relationships and the development of schizophrenia (Tsiantis et al, 1986). Alanen (cited in Tsiantis et al, 1986) suggested that domineering, possessive and hostile mothers who were unable to understand the needs of their children were commonly found in homes of schizophrenic children. Others reported that these families communicate less effectively in conflict situations (Tsiantis et al, 1986). Several relationships have been established through these studies, but as in the genetic
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studies, no direct causes have been identified (Tsiantis et al, 1986).

Problems with the family environment studies stem from the retrospective nature of these studies (Tsiantis et al, 1986). Normal controls are needed for comparisons in order to establish true relationships. Another problem arises because these families are often identified only after the schizophrenic child is diagnosed or hospitalized and then it is difficult to determine if the observed family communication patterns were present before the child's illness or if they developed in response to the schizophrenic child (Tsiantis et al, 1986).

Evidence has suggested that both biological and environmental factors play a role in the development of schizophrenia. Most researchers have come to agree on a nature-nurture interactional model of schizophrenia (Tsiantis et al, 1986). Yet, direct causes are still in question. Researchers still cannot predict who will become schizophrenic. Possible links are being identified in the hopes that early intervention and prevention can be utilized (Asarnow & Ben-Meir, 1988; Cantor, 1982; Fish, 1986; Hellgren, Gillberg & Enerskog, 1987; Watkins et al, 1988).

Treatment of Childhood Schizophrenia

Again the research literature on the treatment of very early onset and early onset schizophrenia is lacking (McClellan & Werry, 1992). However, multimodal treatment programs have been suggested in working with children and adolescents (McClellan & Werry, 1992). Therapists have suggested that treatment should be selected and based on the child's individual cognitive level (Cantor & Kestenbaum, 1986). Issues to address in treatment should
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include the symptomatology both positive and negative; the psychological, social, and cultural needs of the child and family; and the appropriate phase of the illness such as the acute, recuperative, or residual phase (McClellan & Werry, 1992).

Neuroleptics have typically been used to decrease the positive symptoms of schizophrenia such as hallucinations, delusions and formal thought disorders (McClellan & Werry, 1992). In fact, neuroleptics have been the only specific treatment of documented efficacy in schizophrenia (McClellan & Werry, 1992). Yet, the use of neuroleptics with children and adolescents must be extrapolated from the research done with adults because no research has been done with this population (McClellan & Werry, 1992).

Controlled studies with the use of psychotropic medications have been nonexistent (Tanguay & Cantor, 1986). There have been no studies of the cognitive changes that occur in early onset schizophrenia with psychotropic medication (McClellan & Werry, 1992). Yet, psychotropic medication has often been the treatment of choice in adult schizophrenia and therefore, it is surprising that more research has not been done in this area with childhood schizophrenia. In fact, Campbell and Spencer (cited in McClellan & Werry, 1992) have found through clinical work that children seem to respond differently to antipsychotics than adults.

Nevertheless, psychotropic medication is assumed to be a fundamental component of treatment (McClellan & Werry, 1992). Choosing an appropriate medication that will affect the symptoms is important (McClellan & Werry, 1992; Perlmutter et al, 1989). Side effects are another issue to consider when medications are prescribed (McClellan & Werry, 1992).
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seems that discovering the effects of medication, both the therapeutic and side effects, is essential to the future use of psychotropic medications with children.

The psychosocial therapies are also lacking in outcome research with childhood schizophrenia (Cantor & Kestenbaum, 1986; McClellan & Werry, 1992). Cantor and Kestenbaum (1986) have suggested that the treatment adopted with schizophrenic children has more often reflected the theoretical orientation of the therapist rather than the needs of the child. Therapy should be directed at improving family interactions, social skills training, vocational and basic life skills training, problem-solving, and strategies (McClellan & Werry, 1992). Psychosocial therapies along with medication have been shown to decrease relapse rates in adult schizophrenics (McClellan & Werry, 1992).

Some childhood psychiatrists have recommended certain treatment approaches in working with children with schizophrenia through their own clinical experiences (Cantor, 1982; Cantor & Kestenbaum, 1986). Therapy with preschool children should focus on helping children to develop a separate identity which can be done through gross motor therapy (Cantor & Kestenbaum, 1986). The gross motor therapy should continue and increase in complexity (Cantor & Kestenbaum, 1986). Then children should be aided in focusing their attention on something external for a few minutes (Cantor & Kestenbaum, 1986). The child is encouraged to become less autistic-like and to actively explore and master the environment through a nurturing and caring environment provided by the therapist (Cantor & Kestenbaum, 1986).

Children with a later onset of schizophrenia may be better able to communicate, seek
out information, and relate to others (Cantor & Kestenbaum, 1986). The focus of therapy should include limit setting and should be reality oriented at all times (Cantor & Kestenbaum, 1986). The emphasis of therapy is to strengthen the ego, supply cognitive structure, clarify areas of conflict and help to find more adaptive defense mechanisms (Cantor & Kestenbaum, 1986). The therapist can help change the distortions experienced by the child and therefore serve as a bridge to reality or an interpreter of the real world (Cantor & Kestenbaum, 1986).

The pace of therapy and the child's reality testing will vary for each child (Cantor & Kestenbaum, 1986). Therapy may become frustrating for the therapist as these children are likely to experience setbacks because of their hypersensitivity to stress (Cantor & Kestenbaum, 1986). Nevertheless, the therapist should persist. Countertransference issues may arise as well because the schizophrenic child is unable to inhibit, repress, or transform psychological material (Cantor & Kestenbaum, 1986). Extreme understanding of the therapist is also required as these children may have outbursts of rage, engage in obsessional rituals or experience periods of regression (Cantor & Kestenbaum, 1986). These clinical insights and descriptions provide useful treatment guidelines for the new therapist. However, much more research is needed in order to determine the effectiveness of certain therapies in working with children with schizophrenia.

**Conclusion**

Childhood schizophrenia is a rare but serious disorder with complex symptoms that affect children and their families (Cantor, 1982; Tanguay & Cantor, 1986). Early histories of
schizophrenic children have been found to be far from benign (Watkins et al, 1988). Yet, the research in this area is severely lacking.

Most of the findings in childhood schizophrenia have been extrapolated from studies on adult schizophrenia (McClellan & Werry, 1992). The diagnostic criteria and the symptoms of childhood schizophrenia have been reported to be the same as those in adult schizophrenia (McClellan & Werry, 1992; Tsiantis et al, 1986). However, some research has suggested that the early onset of schizophrenia is distinctively different than adult schizophrenia (Tsiantis et al, 1986). Research in this area is essential in order to determine differential diagnoses and symptoms in childhood schizophrenia.

The etiology of schizophrenia also remains unknown (McClellan & Werry, 1992). Biological and environmental factors have been explored and although direct causes have not been identified, factors linked to schizophrenia have been acknowledged. Most researchers have come to agree on a biopsychosocial cause of schizophrenia suggesting that there are many issues that play a part in the development of schizophrenia (Perlmutter et al, 1989; Tsiantis et al, 1986).

Moreover, research in the area of treatment of childhood schizophrenia is virtually nonexistent. There are no studies of children and the effectiveness of psychotropic medication (McClellan & Werry, 1992). Yet, psychotropic medication remains one of the most widely used and accepted treatments in schizophrenia. Research on the effectiveness of the psychosocial therapies is also lacking (Cantor & Kestenbaum, 1986; McClellan & Werry,
Several childhood psychiatrists have made recommendations on the basis of their clinical experiences for appropriate treatment approaches (Cantor, 1982; Cantor & Kestenbaum, 1986).

Overall the literature on childhood schizophrenia is scarce. Generalizations can be made from the research done on adults, but in order to provide the best, most effective, and most comprehensive treatment for children with schizophrenia, much more methodologically sound research is needed in the areas of treatment, symptomatology, diagnosis, and etiology.
References


