BIPOLAR DISORDER IN CHILDREN: A REVIEW OF THE LITERATURE

BY

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The undersigned certifies that she or he has read and recommends to the Faculty of Graduate Studies and Research for acceptance, a final project entitled BIPOLAR DISORDER IN CHILDREN: A REVIEW OF THE LITERATURE submitted by KRISTY BAYNTON in partial fulfillment of the requirements for the degree of Master of Counselling.

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ABSTRACT

Pediatric bipolar disorder (BPD) is a relatively neglected area of developmental psychiatry. Although there is recognition of the development of BPD in children, the lack of research hinders the present understanding of this disorder. Currently, the diagnosis of childhood BPD is based on the adult criteria in the Diagnostic and Statistic Manual, 4th edition (DSM-IV). However, research to date provides evidence for a unique presentation of pediatric BPD. Consequently, application of adult criteria may lead to difficulties in effectively and accurately diagnosing children. This project will review the current literature on pediatric BPD, including (a) historical background, (b) etiology, (c) diagnostic criteria, (d) differential and comorbid diagnoses, (e) treatment approaches, and (f) potential implications. The clinical implications will also be discussed.
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# TABLE OF CONTENTS

SUPERVISOR SIGNATURE PAGE 2
SECOND READER SIGNATURE PAGE 3
ABSTRACT 4
ACKNOWLEDGMENTS 5
TABLE OF CONTENTS 6

CHAPTER 1:
Introduction 7
Historical Background 8

CHAPTER 2:
Procedures 16

CHAPTER 3:
Etiology 17
Presentation of Pediatric BPD 24
Diagnostic Criteria 31
Differential and Comorbid Diagnoses 41
Treatment Approaches 48

CHAPTER 4:
Potential Implications 65
Conclusion 69

REFERENCES 71
CHAPTER 1

INTRODUCTION

BPD in children has been a neglected and controversial area in psychiatry (Faedda, Baldessarini, Glovinsky, & Austin, 2004; Faedda et al., 1995). Although interest in the idea of early-onset BPD has increased, the literature is concerning because it primarily consists of case reports, studies with small numbers, or studies with poorly defined populations (Geller & Luby, 1997). Pediatric BPD continues to be poorly studied and there is a lack of understanding of this increasingly prevalent disorder. There is a lack of epidemiological studies on the course and outcome of BPD during the pediatric years (Jairam, Srinath, Girimaji, Seshadri, 2004; Scheffer & Niskala Apps, 2004). Additionally, authors have noted that there are difficulties in diagnosing a child with BPD (Wolf & Wagner, 2003). The lack of research on pediatric BPD and diagnostic criteria that is specific to an adult population may be related to the underdiagnosis of BPD.

BPD in children affects their psychosocial development, including impaired functioning in school and relationships with family and peers (Wolf & Wagner, 2003). Children with BPD are also at an increased risk for addictions to drugs and alcohol (Kidd, 2005). Currently, the literature in this area consists primarily of case studies, retrospective chart reviews, and open-label studies. The research that exists suggests that BPD in children may present itself differently than BPD in adults (Wolf & Wagner, 2003). The assessment criteria for BPD in children have not been examined thoroughly, but it has been suggested that the current criteria is poor because of its basis on symptoms from an adult population. The lack of epidemiological studies, concerns about the criteria
for assessment, and impact of BPD on a child’s functioning support the need for further studies.

The lack of a comprehensive understanding of BPD in children highlights the need for research in this area. This project utilized a literature review to provide a comprehensive understanding of the current knowledge on BPD in children. A thorough critique of the research is provided in order to gain insight into the research that is needed on BPD. Specifically, this project examined the question, “What is the current understanding of BPD in children?” This project addresses the following: (a) historical background of pediatric BPD; (b) etiology; (c) characteristics and course of BPD; (d) diagnostic criteria, related challenges, and differential diagnoses; (e) treatment options; and (f) potential implications. Furthermore, this project will address some of the limitations of previous research.

HISTORICAL BACKGROUND

The historical background of the concepts of mania and depression can provide individuals with insight into the current understanding of BPD in children. Additionally, the progress throughout the decades in the conceptualization of pediatric BPD can help to frame the present perspectives and concerns. This section will review the historical background of childhood BPD and briefly discuss the conceptualization and understanding of the disorder to date.

The foundation of the concept of BPD began in Ancient Greece (Glovinsky, 2002). Alcmaeon of Crotona and additional Greek physicians described three types of madness and depression and two types of melancholia and mania. The cause of
melancholia was viewed as an interaction between bile and the brain. Both melancholia
and mania were described as chronic diseases without fever.

Melancholia and mania covered a larger spectrum of disorders than the twentieth-
century definitions (Glovinsky, 2002). Symptoms of melancholia included dejected
states, delusions, subdued behavior, insomnia, discouragement, and fear. Manic
symptomatology included excited states, delusions, wild behaviour, grandiosity, and
related affects. Behaviours that were symptoms of one state were often observed in the
other state. For instance, dejection was often observed in mania and grandiosity was
sometimes prominent in melancholia (Jackson, 1986, as cited in Glovinsky, 2002). In the
second century CE, Aretaeus of Cappadocia proposed a connection between melancholia
and mania and suggested that melancholy was the beginning and part of mania (Aretaeus,
1856, as cited in Glovinsky, 2002). Roccatiata coined the term cyclothymia to identify a
mental disease where phases of depression alternated with phases of mania (Roccatiata,
1986, as cited in Glovinsky, 2002). Glovinsky (2002) reported that controversies have
existed from ancient times until present with an emphasis on melancholia and mania,
their relationship, and the differing presentations in children and adults. Although
melancholia and mania were recognized in ancient times, they were conceptualized as
disorders of reason, rather than affective disorders. The relationship between melancholia
and mania was discussed in several publications during the 17th and 18th centuries.

The relationship between melancholia and mania from antiquity through medieval
times was summarized by Jackson (1986, as cited in Glovinsky, 2002) as follows: (a)
appear together among diseases of the head; (b) grouped together as chronic diseases or
forms of madness; (c) described as occurring without a fever; (d) presented in either
adjacent chapters or sections or in the same chapter; (e) mania referred to excited psychotic states; and (f) descriptions barely changed clinically.

In the 19th century, manic depression began to be viewed as a distinct entity (Glovinsky, 2002). A published book discussed the circular relationship between melancholia and mania that was manifested on a regular basis (Falret, 1854, as cited in Glovinsky, 2002). In this discussion, rational intervals of short duration were identified as part of the disease. Following this book, a lecturer proposed the term “folie a double forme” to describe a disorder with a phase of depression followed by an immediate, sudden or gradual, switch to mania (Pichot, 1995, as cited in Glovinsky, 2002).

Kraepelin is an influential figure in the area of psychiatry (Glovinsky, 2002). Distinctions between unipolar forms of the disorder were eliminated and all types of affective disorders were formed into the concept of unified illness. Kraepelin identified manic depression insanity as having a periodic or episodic course, more benign psychosis, and a family history of manic depressive illness (Goodwin, 1990, as cited in Glovinsky, 2002). There was opposition to Kraepelin’s work, but it remained significant until the distinction between unipolar and BPD was accepted 70 years later.

Psychoanalysis emphasized psychodynamic factors in relation to the development of psychopathology (Glovinsky, 2002). Stages of vulnerability were proposed as being related to the development of manic depression. Children lack higher-level cognitive structures and it was believed that manic depression was not a potential disorder in children.

The presence of mania and clinical depression in children and adolescents was observed between the mid-19th and early 20th centuries (Faedda et al., 1995). Group
studies of children with mania began to appear in the early 1920s (Glovinsky, 2002). In 1931, cases were reviewed for manic depression and it was noted that symptomatology began before 12 years of age for some individuals (Barrett, 1931, as cited in Glovinsky, 2002). From the 1930s until recently, BPD was considered rare before puberty. In the 1930s through to the 1960s, clinical theorists proposed that melancholia and mania were unlikely in children. At this time, major mood disorders were associated with postpubertal or late adolescent superego and psychotic disorders of children were related to schizophrenia. Influential child psychiatrists disregarded the possibility of BPD in children and this disregard may have contributed to the lack of attention given to cases and alternative diagnoses. Case studies provided objective support for the suggested rarity of mania or melancholia in children. However, this fact may reflect the lack of understanding and corresponding lack of diagnostic criteria for BPD in children.

Kraepelin proposed the concept of mixed mania in the late 19th century, but it was not utilized in the psychiatric classifications until 1987 (Marneros, 2001, as cited in Dunner, 2005). The concept of mixed mania meets the criteria for both episodes of mania and major depression, except for duration, for nearly every day for at least one week. Individuals with mixed mania often have classic manic symptoms, such as racing thoughts and delusions of grandiosity, along with a depressed mood, sadness, and anxiety (Swann, 1995, as cited in Dunner, 2005).

Kraepelin’s (1921, as cited in Carlson, 2005) monograph on manic-depressive insanity in the early 20th century increased interest in the prospect of childhood BPD. Kraepelin proposed that there is the potential for BPD to develop in children, but considered this disease rare in childhood (Kraepelin 1921, 1893, 1896, 1899, as cited in
Glovinsky, 2002). Furthermore, occurrences of the disorder were described in younger children in Germany during the same time period (Ziehen, 1999, as cited in Glovinsky, 2002). The description of the disorder in these children reported a sudden onset, quick climax, and rarely lasting a few days and commonly lasting for several weeks or months. Additionally, a period of depression followed by mania was common and most cases could potentially be cured.

Surveys completed with hundreds of state hospitals with individuals with manic-depression suggested that manic depression that reflects the adult characteristics was rare, occurred primarily post-puberty, and was mostly depressive (Barrett, 1931; Strecker, 1921, as cited in Carlson, 2005). In the 1950s, The Nervous Child published several articles with case descriptions of children and questioned the possibility of manic depression in preadolescents (Carlson, 2005). Childhood BPD was still viewed as a rare disease, but the possibility that a childhood depiction of BPD may be demonstrated by childhood behavioural psychopathology existed.

Despite the general resistance of accepting the idea of manic depression in children, Campbell (1952, as cited in Glovinsky, 2002) completed a study with manic-depressive patients. He suggested that children were being misdiagnosed and highlighted the following findings: (a) frequency and importance of manic-depression in children, strong familial tendency, and overemphasis on conventional and dynamic factors in psychiatric illnesses in children; (b) cyclothymic personality and manic-depressive psychosis in children are too commonly defined as psychoneurosis, schizophrenia or “problem children”; (c) the earlier process of cyclothymia peaks clinically, the more pronounced manic-depression is generally; and (d) cyclothymic disease manifested at a
young age is more likely to have a strong manic-depressive disposition, a chronic course, or many recurrent episodes.

Anthony and Scott (1960, as cited in Carlson, 2005) reviewed the literature for the purpose of examining the existence of classical manic-depressive psychosis in preadolescents. Anthony and Scott (1960, as cited in Glovinsky, 2002) provided a comprehensive summary of the literature on manic-depressive psychosis in child. The literature between 1884 and 1954 was reviewed, then developed and outlined 10 criteria for manic depression in children. After completing this research, it was concluded that manic depression rarely occurs in children and only a few cases met their criteria for the disorder. This work is considered a pioneering attempt at systemization, but the inclusion of Kraepelin’s symptoms and criteria inconsistent with Diagnostic and Statistic Manual, 3rd Revision (DSM-III-R) criteria for adult mania led to the likelihood of exclusion of cases and underdiagnosis.

Neurologists raised the question of childhood mania (Carlson, 2005). Weinberg and Brumback (1976, as cited in Glovinsky, 2002) are responsible for the next major attempt at establishing diagnostic criteria for mania in children. In this attempt, the criteria utilized by Feighner et al. (1972, as cited in Glovinsky, 2002) for diagnosing adults was adapted and included euphoric or irritable mood, in addition to three or more of the following and reflecting a change from the child’s normal behaviour: (a) hyperactive, intrusive behaviour; (b) pressured speech; (c) flight of ideas; (d) grandiosity; (e) decreased amount of sleep or unusual sleeping pattern; (f) distractibility; and (g) one month duration of symptoms. Young children who were in a clinic for learning disabilities were also reported as displaying characteristics of mania (Carlson, 2005).
However, the observations were dismissed because the children were observed as being hyperactive children.

In the 1970s, clinicians began to present cases of manic behaviour in children (Glovinsky, 2002). At the same time, clinicians and researchers also began to report on using lithium in children. In 1978, a publication suggested that manic depression was rare because it was misidentified as schizophrenia (Carlson & Strober, 1978, as cited in Carlson, 2005). Case studies demonstrated youth with episodes of severe, psychotic mania or depression, reasonable intermorbidity functioning and the absence of negative symptomatology and deterioration. This publication raised awareness to the fact that affect-laden psychosis was more often BPD than schizophrenia and applicable to adolescents.

The revival of the concept of BPD in children could be credited to work by DeLong (1983/1990, as cited in Glovinsky, 2002). In this work, children and adolescents without a diagnosis, but demonstrating hyperaggressive behaviours and symptoms of manic depression were successfully treated with lithium. The success in treatment led to an increased interest in the area of BPD in the pediatric population.

Towards the end of the 1970s, a paper was published that identified a syndrome in children and adolescents below 16 years old (Davis, 1979, as cited in Glovinsky, 2002). This syndrome was identified by the following features: (a) affective storms, including a loss of control that is highly intense, disruptive, and transient; (b) significant family histories of affective disturbances; (c) mental, verbal, and physical hyperactivity; (d) high level of distractibility; and (e) rapid talk. This syndrome depicts the current conceptualization of pediatric BPD. Along with noting these primary features, it was also
highlighted that children differ from symptomatology of mania in adults. In children, hyperactivity and emotional upheaval is different than grandiosity exhibited in adults and children do not present with hallucinations or delusions. Additionally, children with manic-depression often experience interpersonal problems because of their erratic behaviour. This description also included a criterion of exclusion, the absence of psychotic thought disorder. Children with mania may present with behaviour that appears psychotic, but their thoughts are not bizarre. At the same time, researchers were beginning to find out that some adults with manic depression had also experienced symptoms during their childhood (Glovinsky, 2002).

Coll and Bland (1979, as cited in Glovinsky, 2002) were also studying the same phenomenon in Canada. It was proposed that children could develop manic depression, but it was underdiagnosed because of the difference in presentation. This disorder was now being referred to as BPD and required the one or both of the following:

1) euphoria
   a) denial of problems or illness
   b) inappropriate feelings of well-being, inappropriate cheerfulness, giddiness, and silliness
2) irritability and/or agitation (i.e. belligerence, destructiveness, and anti-social behaviour)
3) hyperactivity, “motor drive,” intrusiveness
4) pressured speech (potentially intelligible), garrulousness
5) flight of ideas
6) grandiosity (possibly delusional)
7) sleep disturbance (decreased sleep and unusual sleep pattern)

8) distractibility (short attention span)

By the early 1980s, people were becoming more accepting of the idea that children could present with characteristics of BPD (Glovinsky, 2002). It was observed that children presented with characteristics that were unique, whereas older children and adolescents may present with a more classic pattern of symptoms. Although more professionals were in agreement about the diagnosis of early-onset BPD, there was still uncertainty about the disorder. The use of the adult criteria contributed to this controversy. Developmental research highlighted the influence of developmental factors in the presentation of symptomatology.

The issue of BPD in children continues to be an area of controversy. Carlson (2005) reported that young children exhibiting premorbid histories, onset of clear episodes of euphoria, mania proceeded by severe, psychomotor-retarded depression and subsequent euthymia with a return to good functioning are still rare. However, there continue to be a significant number of children with symptoms of mania, often overlapping a number of diverse developmental and psychiatric conditions. Although there is a lack of studies on the prevalence of BPD in children, there is evidence supporting an increase in prevalence of prepubertal mania (Geller, Zimerman, Williams, DelBello, Bolhofner, Craney, et al., 2002).
CHAPTER 2

PROCEDURES

This project is a literature review designed to provide a comprehensive understanding of the current knowledge on BPD in children. This project will address the following: (a) historical background of pediatric BPD; (b) characteristics and course of BPD; (c) diagnostic criteria, related challenges, and differential diagnoses; and (d) treatment options. Electronic databases that were used for the literature review were “PsycINFO” and “MEDLINE.” Literature from 2000 to 2005 was reviewed and a key word search was utilized to access primary resources. Key terms utilized for this search included “bipolar disorder and children,” “mania and children,” “manic depression and children,” and “bipolar disorder and pediatric.” Primary resources were mainly chosen to summarize the current understanding of pediatric BPD. Secondary resources were utilized when the primary resources were unavailable or unattainable. The attainment of potential resources led to a review of the articles to ascertain whether or not they were primary resources. After categorizing the articles into primary or secondary resources, the articles were reviewed for relevancy to BPD in children. Research articles that fit within the scope of this project were chosen and reviewed.
The etiology of a mental health disorder is necessary in order to create appropriate and effective assessment tools. Additionally, a professional’s understanding of the etiology of the disorder is important in developing prevention and intervention strategies to help address childhood BPD. At the present time, the exact cause of pediatric BPD and research in this area is limited. Although there is research in the area of adult-onset BPD, the question of whether or not early-onset BPD represents the same disorder also raises the question of whether or not the etiology is the same. This section will discuss the current research on the etiology of pediatric BPD.

Biology

It has been suggested that there is continuity of the role of biology between early and later-onset BPD (Todd & Botteron, 2002). In adults with BPD, structural imaging studies have found increased frequencies of deep white matter hyperintensities, enlarged ventricles, decreased volume of temporal lobes and thalamus, and abnormalities in the size of the amygdale (Todd & Botteron, 2002). The role of structural abnormalities in the development of pediatric BPD is an area that needs to be further investigated.
The role of the basal ganglia in the development of BPD has been questioned, but not thoroughly researched in children. In a study by Sanches et al. (2005), anatomical abnormalities were investigated in order to evaluate whether or not they are present early in the course of the disorder. Specifically, the volume of striatal structures were evaluated in children and adolescents with BPD and compared to a healthy control group. Larger striatal volume was not observed in children with BPD. The sample had a heterogenous treatment history in relation to antipsychotic use. However, there were no children taking the medication during the magnetic resonance imaging and no significant differences among subgroups with varying treatment histories. The treatment status of the children is a methodological limitation of this study. The small sample size and children with comorbid disorders are additional limitations. Sanches et al. (2005) argued that their sample was representative of the population of children with BPD, since children with BPD are more likely than not to have a comorbid disorder. A final limitation is the inclusion of both children and adolescents with BPD-I and BPD-II in the sample. Given these limitations and the lack of additional studies on the role of the basal ganglia in children, further research in this area is warranted.
Biological markers would help to identify prodromal forms of BPD, but there have not been any markers identified for BPD (Chang, Steiner, Dienes, Adleman, & Ketter, 2003). The identification of markers of risk for BPD requires studies of a high-risk sample along with serial longitudinal assessments to observe the time to develop BPD. A biological marker to identify BPD could reflect a trait that predisposes someone to BPD or a biological change inflicted by the development of the disorder before clinical symptoms appear. The identification of a marker would allow for intervention and potentially prevention prior to the full development of the disorder.

**Genetics**

Although the cause of pediatric BPD is unknown, researchers have pointed out that there is substantial evidence to suggest a biological basis for the development of BPD in adulthood (Lofthouse & Fristad, 2004). There is evidence to support the idea that mood and behavioural dysregulation runs in families. Family history is more likely to be present in youth with BPD (Geller et al., 1994). The heritability of childhood BPD appears to be high (Faraone & Tsuang, 2003), but there are limited published studies on the heritability of early-onset BPD in twin (Todd & Botteron, 2002). However, this association does not necessarily mean that it is genetic and twin studies can help to elucidate genetic associations in relation to nongenetic associations. Additional twin studies would be beneficial in understanding the role of genetics in developing BPD.
Research in the area of heritability and early-onset BPD is limited (Todd & Botteron, 2002). Furthermore, researchers are beginning to acknowledge the existence of BPD in families, but there have not been any twin or adoption studies on pediatric BPD (Biederman et al., 2003). Twin and adoption studies provide evidence for researchers to elucidate the genetic and environmental relationship with respect to the development of a disorder. Family studies to date suggest that there is a genetic component to the development of BPD (Wozniak et al., 1995). In a study designed to investigate the presence and severity of childhood onset affective disorder in families, relatives of children with BPD had elevated rates and increased severity of affective disorders as assessed by an earlier age of onset and increased suicide attempts (Todd, Neuman, Geller, Fox, & Hickok, 1993). Todd et al.’s (1993) findings suggest that pediatric BPD may be a subtype of BPD with high familial loading.

Trauma

Researchers have suggested that trauma could be a cause of BPD in children (Biederman et al., 2003). Although research findings have supported an association between traumatic events and BPD in adults, there has been limited systematic research on the role of trauma in the development of pediatric BPD. Researchers have noted that early-onset BPD is often associated with extreme violence, severe behavioural dysregulation, and hypersexuality. Biederman et al. (2003) suggested that trauma could be a reaction to or risk factor for exposure to trauma. Wozniak et al. (1999) conducted a longitudinal study of boys with and without Attention Deficit Hyperactive Disorder (ADHD). Pediatric BPD was identified as an important antecedent of traumatic experiences. The relationship between trauma and pediatric BPD has important
implications. For instance, a traumatized child who presents with irritability and mood lability may have these symptoms attributed to the trauma. In contrast, longitudinal research has found that BPD is an antecedent risk factor for trauma and not a reaction to it. The recognition that BPD can precede trauma can affect clinical decisions, such as the treatment approach taken when symptoms are demonstrated. Additionally, improving recognition and assessment of children with BPD can also help to reduce their risk for trauma if they are treated in a timely and effective manner. A clear understanding of the relationship between trauma and BPD can affect prevention, assessment, and treatment of children with BPD.

Exposure to adverse life effects is common in pediatric BPD and may have a negative impact on prognosis (Marchand, Wirth, & Simon, 2005). Marchand et al. (2005) reviewed retrospective charts of youth with BPD to determine the frequency and effects of exposure to trauma in pediatric BPD. They found that exposure to adverse life events was associated with symptom severity and treatment outcome. Specifically, children who had experienced adverse life events were more likely to have a delayed diagnosis, psychiatric hospitalization, residential treatment, and a decreased response to treatment. In most cases, this association was noted whether or not the child met the diagnostic criteria for post-traumatic stress disorder (PTSD). This study suggests that children with BPD may have a greater risk of experiencing adverse life events and consequently may have a poorer prognosis. Additionally, girls were more likely to have experience trauma than boys and appear to be at an increased risk for exposure to adverse life events. These findings need to be confirmed through rigorous studies.
Leverich et al. (2002) examined the relationship between physical or sexual abuse as a child or adolescent and the development of psychopathology. Their study found that individuals who reported a history of physical or sexual abuse as a child or adolescent had a history of early onset of BPD, increased number of comorbid disorders, faster cycles, higher rate of suicide attempts, and more psychosocial stressors occurring before the first and most recent affective episode. The retrospective associations between abuse and a more severe course of BPD were validated prospectively.

Cognitive Structures

Children at a high risk for developing a mood disorder appear to have biases in attention and memory similar to biases exhibited by adults with BPD or unipolar depression (Gotlib, Traill, Montoya, Joormann, & Chang, 2005). Gotlib et al. (2005) examined information processing in children who did not have a history of mood disorders, but whose parents had BPD. These findings suggest that children who are at an increased risk for developing an affective disorder are characterized by potentially pathogenic cognitive structures that can be activated by a sad mood. In a negative mood state, these high risk children demonstrated characteristics of a cognitive vulnerability for affective disorders. The mechanisms of cognitive vulnerability for BPD are enhanced by these findings.

Risk Factors

Cyclotaxia refers to individuals at risk for developing BPD (Findling, Gracious, McNamara, & Calabrese, 2000, as cited in Findling, 2005). Specifically, this term refers to youth who have a parent with BPD and meet the diagnostic criteria for symptoms of
cyclothymia or Bipolar Disorder Not-Otherwise Specified (BPD-NOS). Individuals who develop cyclothymia have a very high risk of developing BPD (Kidd, 2005).

A salient risk factor for the development of BPD is family history of mood disorder (Coyle et al., 2003). Children of parents with BPD are at an increased risk for mental health disorders, including affective disorders during childhood (Lapalme, Hodgins, & LaRoche, 1997). Children of parents with BPD were also at a significantly higher risk for BPD than children of parents without a mental disorder. The associated relationship between developing a mental health disorder and having a parent with BPD was consistent, but moderate in strength. This finding suggests that the effects of having a parent with BPD may be more strongly related to mental disorders in adulthood. Additionally, parental illness is not a sufficient factor in solely predicting with high confidence the outcome for a child (Coyle et al., 2003).

Factors that may predispose children to develop BPD include prior history of depression and suicide, family history of BPD and disruptive disorders, psychosocial stressors, and treatment with psychopharmacologic agents (Robb, 1999). Cumulative psychosocial stressors and physical and sexual abuse are environmental risk factors for the development of pediatric BPD. Perinatal insult is a risk factor in the development of psychiatric disease, but has not been thoroughly investigated in a pediatric population (Rush, 2003). A focus on investigating the risk factors that may predispose a child to develop BPD would add to the current knowledge of pediatric BPD.

Interaction Between Genetics and Environment

Presently, BPD is viewed as a disorder that is developed through an interaction between genetics and environmental factors (Rush, 2003). Although the role of genetics
is still unclear, it has been suggested that early-onset BPD may have a heavier degree of genetic loading in combination with a significantly lower threshold for being triggered by environmental factors.

**PRESENTATION OF PEDIATRIC BPD**

BPD has typically been viewed as an adult disorder, but researchers have found that it is not age specific (Barton, 2001). Although there is now a recognition that BPD can occur in children, there is a lack of understanding of early-onset BPD. At the present time, the diagnosis of BPD in children is reliant upon the same criteria for diagnosing adults (Wolf & Wagner, 2003). However, children may present with different symptoms than adults. There is a lack of knowledge on characteristics of BPD specific to a childhood population (Barton, 2001). Symptoms of childhood BPD are frequently unrecognized or misdiagnosed. Pediatric BPD is commonly mixed with both the presence of depression and mania occurring simultaneously (Biederman et al., 2004). Given the discrepancy between presentation in children and adults, utilizing the diagnostic criteria for adults on a pediatric population has significant implications. Furthermore, characteristics of pediatric BPD are also characteristics of alternative childhood psychiatric disorders. The lack of knowledge about pediatric BPD affects the ability of professionals to effectively recognize and diagnose children. This section will discuss the present understanding of the presentation of early-onset BPD.

*Characteristics of Pediatric BPD*

Researchers have found that children present with BPD differently than adults. However, defining the characteristic features of pediatric BPD is difficult for numerous reasons. These reasons include the following: (a) common psychiatric disorders in
children may present similarly, (b) it may be the most prevalent idiopathic psychotic disorder, (c) strong association with aggression and suicide, (d) over the past century, trends were reported of younger onset of major mood disorders and earlier suicide, and (e) often a more severe and longer course of BPD illness in children than adults. Recent interest in childhood BPD has led to attempts to define the clinical characteristics and course of pediatric BPD.

Faedda et al. (2004) conducted a study on the characteristics of BPD in children. In this study, 82 children who had been diagnosed with BPD were participants. Prominent features in this sample include: (a) very early onset of psychopathology, including mood-instability, hyperactivity and aggression; (b) mood lability, irritability, and dysphoria, rather than euphoria; (c) onset with dysphoric or mixed states, rather than euphoric mania; (d) sleep disturbances, rather than decreased need for sleep; and (e) highly recurrent, labile or fluctuating affective and behavioral symptoms following a subchronic, rather than an episodic course. These findings provide support for the unique presentation of BPD in children. Additionally, researchers found high rates of misdiagnosis, psychosis, and suicidality. These findings may support the idea that psychosis and suicidality are common in children with BPD. Although this study indicated characteristics of children who had been diagnosed with BPD, longitudinal studies are needed to examine whether or not these children develop BPD as represented in adults.

Mania in Children

The current DSM-IV criteria for diagnosing mania was developed for adults, but are applied to the diagnosis of children (Kowatch, Fristad, Birmaher, Wagner, Findling,
Quantitative comparisons from assessments of symptoms of mania in children, adolescents, and adults provide evidence to support the idea that mania in children does not present in the characteristic way it presents in adults (Faedda et al., 1995). Psychomotor hyperactivity is a common manifestation of mania for people of all ages. However, hyperactivity is the most common behavioral symptom of mania in children and appears to be more severe than in adult-onset BPD (Shastry, 2005). Symptoms of mania include pressured speech, flight of ideas, euphoric elation, and grandiosity. These symptoms tend to rise with maturation and are considered more prevalent in adults. Mania in children rarely presents itself by euphoric mood (Biederman, Mick, Faraone, & Wozniak, 2004). The duration of manic episodes in children ranges from brief, tantrum-like affective storms lasting minutes or hours to episodes lasting days or months (Faedda et al., 1995). The characteristics of mania in children differ than the manic symptomatology in adults. However, pediatric BPD does not always present with mania.

In a four-year prospective longitudinal study, children with BPD-I (manic or mixed) with at least one cardinal symptom (i.e. elation and/or grandiosity) were examined to provide validation of the existence and long-episode duration of mania in children (Geller, Tillman, Craney, & Bolhofner, 2004). The findings validated the existence of childhood mania, demonstrated persistent mania and had not been diagnosed with solely ADHD at follow-up. Although there is a high rate of comorbidity of ADHD in pediatric BPD, there is still uncertainty about why this comorbidity occurs.

Geller et al. (2004) found a long duration of episodes of mania and mixed mania in children. Geller et al. (2004) suggested that these children are likely representative of
children with BPD who present clinically because of the selection process for subjects. However, this study did not address whether or not children with shorter episodes receive treatment.

Childhood mania with mixed symptoms or rapid cycling moods is common in a pediatric population (Geller & Luby, 1997). In older children and adolescents, mania is likely to present as a syndrome similar to adults with a clearer episodic course, euphoric-irritable mood, grandiosity, racing thoughts, and flights of ideas. Pediatric mania appears to be presented in an atypical manner including irritability, mania mixed with symptoms of major depression, and a chronic course. Psychosis can also be a symptom of childhood mania (Faedda et al., 1995). Given the differences in the presentation of BPD in children and adults, the application of diagnostic criteria created for adults to children may affect the efficacy of assessment.

The variation in estimates of manic episodes may reflect the problems in diagnosing children and limits related to the diagnostic criteria established for adults. Studies on manic episodes in pediatric BPD are needed and an emphasis on episode length, rates of cycling of mood, and treatment response would be beneficial.

In a study by Craney and Geller (2003), less than half of the participants in the prepubertal and adolescent group had received antimanic medication (defined as lithium, neuroleptic, or anticonvulsant) at a two-year follow-up. These findings suggest that community practitioners may have difficulty recognizing mania in children. Further studies on mania in children would be beneficial and could be useful in educating community practitioners.
Psychosis

Psychosis is defined by the presence of hallucinations or delusions (Biederman, Petty, Faraone, & Seidman, 2004). Few studies have examined childhood-onset psychosis. However, studies that examined childhood psychosis have documented hallucinations and/or delusions are most common in mood, schizoaffective, and schizophrenic disorders (Volkmar, 1996, McKenna et al., 1994, as cited in Biederman et al., 2004). In a study by Biederman et al. (2004), there was a higher rate of BPD in children with psychosis. Youth with BPD and psychosis may be misdiagnosed as having schizophrenia (Weller & Weller, 1986). The literature on psychosis and BPD suggests that psychosis may be a correlate of BPD.

Psychosis can be a symptom of mania and mood congruent psychotic symptoms may also be present in a child with mania. The presence of psychotic symptoms in a child means a clinician needs to ascertain whether or not psychosis is mood congruent and secondary to another psychiatric disorder (Kowatch et al., 2005). In the past, there appears to have been a failure to differentiate pediatric BPD from schizophrenia, schizoaffective disorder, and other psychotic disorders during the manic phase of BPD (Faedda et al., 1995). Additionally, psychotic symptoms in children are frequently overlooked (Pavuluri, Herbener, & Sweeney, 2004). The potential for unique characteristics for BPD in children and lack of corresponding assessment criteria provides support for further research in this area.

Irritability

The symptom of irritability is included as a criterion in the DSM-IV for mania in adults (Pruett & Luby, 2004). In children, irritability is a more inconsistent and
nonspecific marker in several childhood mental disorders, such as disruptive behaviour disorders. Geller et al. (2002) suggested that the inclusion of irritability as a central indicator of childhood mania contributes to the inaccuracy of diagnosing childhood BPD. Furthermore, irritable mood is a characteristic of depression that is specific to children and adolescents (Benazzi & Akiskal, 2005).

The most common mood disturbance in children is severe irritability and “affective storms” or prolonged and aggressive temper outbursts. The frequency of explosive outbursts may be distressing and the child can be emotionally labile (Wolf & Wagner, 2003). The irritability can be very severe, persistent, and violent (Biederman et al., 2004).

A study by Geller et al. (2002) evaluated the prevalence of symptoms classified by the DSM-IV in prepubertal and early adolescents. The findings indicate that elation, grandiosity, flight of ideas or racing thoughts, decreased need for sleep, and hypersexuality were symptoms that best discriminated children with BPD from children with ADHD and the control group. The researchers required the presence of elation or grandiosity for the diagnosis of BPD in an effort to increase specificity and control for overlapping symptoms of ADHD. The researchers found that irritability was a common symptom of mania, but was very nonspecific. Specifically, 72% of the children with ADHD presented with irritability in contrast to children with BPD who presented with rapid cycling, elation, depression, and irritability.

In children who met the criteria for mania, the predominant mood was irritability, rather than euphoria (Biederman et al., 2004). Irritability is a common symptom in child psychopathology and tantrums are common in children with ADHD. However, irritability
in a child with BPD tends to be very severe, persistent, and extremely disabling. Irritability is also often associated with a volatile temper and aggression. In the absence of euphoria, clinicians might attribute irritability to psychosocial factors or conduct disorder without consideration being given to mania. The irritability of pediatric BPD appears to be qualitatively distinct in intensity, frequency, and aggressive and out-of-control behavior.

Course of Pediatric BPD

In contrast to adult BPD, the natural course of BPD in children is atypical (Biederman et al., 2004). Geller and Luby (1997) conducted a 10-year review on the literature of pediatric BPD. In this review, it was noted that the course of pediatric BPD tends to be non-episodic, chronic, rapid-cycling, and mixed manic state. The course of mania in child is often less episodic than in adolescents or adults and sustained irritability, dysphoria, lability, and functional impairment are common (Kowarth et al., 2005). Geller et al. (2004) reported that children with pediatric BPD often present with long episodes, chronicity, and severity (i.e., ultradian rapid cycling, psychosis, and mixed mania).

Cycling

Cycles are defined as “mood switches occurring daily or every few days during an episode” (Tillman & Geller, 2003, p. 270). The moods of children with BPD do not cycle in the same manner as adults with BPD (Biederman et al., 2004). The onset and offset of an episode can be difficult to recognize in children because many children with BPD present with frequent daily mood swings that have been occurring for months to years. The first episode of mania may occur after depression or dysthymia or during treatment
with an antidepressant. Pediatric BPD is commonly mixed with both the presence of depression and mania occurring simultaneously (Biederman et al., 2004). Mixed or dysphoric moods characterized by frequent, brief, but intense mood lability and irritability are common in pediatric BPD, whereas euphoric mania is more common in adults.

A study conducted by Geller, Williams, Zimerman, Frazier, Beringer, and Warner (1998) found that there is a higher prevalence of rapid, ultra-rapid, and ultradian cycling in prepubertal cases of BPD than adult onset BPD. The cycling patterns of children with BPD are contrary to the discrete episodes with clear onsets and offsets that are commonly observed in older adolescents and adults. Geller et al. (1998) suggested that the cycling patterns of a prepubertal population may indicate: (a) a developmental pattern specific to prepuberty that will change to discrete episodes with age; (b) more severe illness associated with childhood onset, but analogous pathogenesis to adult BPD and similar to the course of well-known medical illnesses; (c) illness that is phenotypically similar to late adolescent or adult-onset BPD, but has a different pathogenetic mechanism and analogous to juvenile-onset in comparison to adult-onset diabetes; and (d) subtype where probands and pedigree members rapidly cycle in adulthood.

**DIAGNOSTIC CRITERIA**

The diagnosis of BPD in children is based on the adult criteria for BPD. Given the atypical presentation of BPD in children, diagnosing a child is a difficult task. The age of the onset of BPD in children widely varies. Clinicians who identify BPD in children must rely heavily case reports in the literature to determine appropriate treatment (Wozniak, 2005). There is a need for developmentally sensitive questions to appropriately assess the
presence of mania or BPD in children (Wozniak, 2005). Childhood BPD may have been neglected because of the lack of assessment tools that select childhood traits from mood disorders. This section will discuss the diagnostic criteria for childhood BPD.

Distinct Presentation

The atypical presentation and course of BPD raises the question of misdiagnosis when children are assessed utilizing the criteria from the DSM-IV that is based on adult BPD (Biederman et al., 2003). The diagnosis of BPD in children has numerous challenges in order to prevent under or over-diagnosis, to consider differential diagnoses and comorbidity, and to minimize substance abuse, suicidal acts, and other risky behaviours (Faedda et al., 2004; Weller, Calvert, & Weller, 2003). Diagnostic standards specifically for pediatric populations are not generally developed and field-tested. It is also important to note that behaviours that are symptomatic of BPD are also relatively common in youth and not specific diagnostically. Furthermore, the cognitive ability of a child affects the ability to communicate the subjective symptoms of the disorder, such as flight of ideas and delusional thinking (Scheffer & Niskala Apps, 2004). The assessment process can also be complicated by comorbid disorders. Despite these concerns and the implications for assessing child for BPD, there is a lack of understanding of pediatric BPD and the paucity of information affects the timely and accurate diagnosis of children.
Medical Conditions and Medication

In assessing children for BPD, professionals need to be aware of conditions that can mimic symptomatology of BPD. Medical disorders can mimic typical characteristics of BPD, such as hyperthyroidism and head injuries (Kowatch et al., 2005). Medical conditions that may have manic symptoms include neurological diseases, including brain tumours, central nervous system infections, and human immune-deficiency symptoms, multiple sclerosis, and temporal lobe seizures and endocrinopathies, such as hyperthyroidism and Cushing’s Syndrome (James & Javaloyes, 2001). A thorough physical examination and necessary biochemical examinations can aid in the appropriate diagnosis.

BPD also needs to be differentiated from pediatric psychiatric disorders, including ADHD, disorders of development, conduct, personality, early schizophrenia and other psychoses, neuropsychiatric disorders, anxiety, and substance use disorders. Current diagnostic criteria can be reliable and effective in diagnosing pediatric BPD, but there are significant limitations in extending adult criteria to pediatric psychopathology.

There are medications that are associated with an increase in symptoms of mania (Kowatch et al., 2005). Prescribed medications, such as antidepressants or steroids, and substance abuse can lead to manic symptoms (James & Javaloyes, 2001). Children who are taking stimulants may experience a rebound effect, agitation or increased hyperactivity when the stimulant wears off (Carlson, 2005). Professionals have used the rebound effect as an alternative for the development of a mood disorder. Carlson and Kelly (2003) examined children in a psychiatric hospital and noted that they were more irritable and difficult on afternoons that they received stimulants in comparison to
afternoons they did not receive stimulants. Another medication that can be increase symptoms of mania are medications used to treat depression. In adults, tricyclic antidepressants have been observed to induce mania, whereas Selective Serotonin Reuptake Inhibitors (SSRIs) appear to be more frequently responsible for inducing mania in children (Carlson, 2005). However, Carlson (2005) notes that this relationship may be a measure of their wider usage in the population. An assessment of a child for BPD needs to consider the child’s current medication and whether or not the medication could be responsible for inducing symptoms of mania.

Carlson and Mick (2003) examined the drug-induced behavioural disinhibition in children through the use of weekly recordings on behaviours, medications, and dosages. They found that it was difficult to distinguish behaviours that were a part of the child’s natural history and behaviours that were medication induced in children with significant emotional dysregulation. Limited power in this study prevented Carlson and Mick (2003) from identifying medication responsible for drug-induced behavior. However, it was suggested that stimulants were potentially protective and SSRIs were more likely to produce disinhibited behavior than tricyclics. Similarly, Craney and Geller (2003) found that there was no evidence that stimulants or antidepressants led to switching to a mania from a major depressive episode. Despite this finding, it has been recommended that parents and professionals be aware of the potential activation and agitation as a side effect of antidepressants and rebound as a side effect of stimulant medication (Carlson, 2005).
Assessment of Pediatric BPD

A primary challenge in diagnosing BPD in children is its distinct presentation from BPD in adults (Wozniak, 2005). At the present time, there are not any psychometric measures designed for the purpose of assessing a child for BPD (Findling, 2004). Research studies on the developmentally distinct presentation of BPD have primarily focused on the atypical presentation in children and the development of sensitive assessment tools (Wozniak, 2005).

At the present time, professionals are responsible for diagnosing a child through the use of the DSM-IV criteria. The diagnosis of pediatric BPD is primarily clinical (James & Javaloyes, 2001). Professionals require carefully charted history with family and school professionals as informants. Additionally, a physical examination is required with baseline biochemical and haematological tests. Semistructured interviews can also be used to reliably elicit symptoms that have been operationally defined. The Young Mania Rating Scale (YMRS) is an assessment instrument that has been used in children and can chart both progress and be useful in diagnosis (Fristad, Weller, & Weller, 1992).

The DSM-IV explicitly lists the criteria for diagnosing BPD in adults (Coyle et al., 2003). As previously noted, these criteria do not necessarily apply to children. For instance, a hallmark of adult BPD is a pattern of discrete episodes of mania. In children, discrete episodes are less likely and children are more likely to present with chronic, non-episodic, rapid-cycling, mixed episodes. The presentation can vary widely and symptoms can overlap with other child psychiatric disorders. The unique presentation and course of BPD and overlapping symptomatology can present a challenge for professionals.
Although the DSM-IV criteria are used for diagnosis, the methods used to gather information concerning the criteria vary among children, adolescents, and adults (Coyle et al., 2003). An accurate diagnosis relies on the child’s ability to understand questions and provide information on their current mood state and mood state during the previous few weeks. Children are not necessarily capable of providing such information and interviews with both the parent and child are needed until the child is at least 14 years old. Collateral information should be considered in the assessment of BPD in children (Biederman, 2004).

The DSM-IV classifies BPD into four distinct subtypes (Barton, 2001): (a) bipolar I disorder (BPD-I), (b) bipolar II disorder (BPD-II), (c) cyclothymia, and (d) BPD not otherwise specified (BPD-NOS). Barton (2001) argued that knowledge of the symptoms and subtypes of BPD will help professionals distinguish between early-onset BPD and adult onset BPD.

Tillman and Geller (2003) argued that there is a need to define episodes and cycles in order to distinguish the duration of an illness and cycling phenomenon in individuals who present with ultra rapid or ultradian cycling. Tillman and Geller (2003) proposed that episodes be defined as “the duration from onset to offset of a period of at least 2 weeks in length during which only one mood state persists” or “the duration from onset to offset of a period of ultra rapid or ultradian cycling for at least 2 weeks” (p. 270). Children who have experienced at least one manic episode are classified as having BPD-I (Dunner, 2005). Children who have had at least one distinct episode of both hypomania and major depressive disorder are defined as having BPD-II (Dunner, 2005; Kowatch & DelBello, 2005). BPD-II is more likely to occur in adolescents than children. Past
episodes of hypomania may not be recognized without carefully assessing historical information.

Cyclothymia can be difficult to diagnose because hypomania and depressive symptoms are less severe than in BPD-I or BPD-II (Kowatch & DelBello, 2005). Cyclothymia is characterized by a pattern of symptoms that are similar to characteristics of BPD, but their severity is less (Rush, 2003). Children who present with cyclothymia may have high-mood lability, emotional over activity, impulsive-aggressive emotionally erratic behaviours, and hypersensitivity (Akiskal, 1995, as cited in Kowatch & Delbello, 2005). Prospective mood charting is a useful assessment tool for cyclothymia. A history of cyclothymia is often reported in individuals with BPD and it is often viewed as a precursor to BPD-I and BPD-II (Rush, 2003).

BPD-NOS is the most common subtype of BPD (Kowatch & DelBello, 2005). This diagnosis is warranted when an individual has characteristics of BPD, but the symptoms are not severe or of long duration to meet the criteria for a diagnosis of BPD-I, BPD-II, or cyclothymia. The diagnosis of BPD-NOS is also warranted when BPD is secondary to a medical condition, such as fetal alcohol syndrome. There are medications and medical conditions that exacerbate or mimic BPD. Professionals need to be aware of confounding variables and assess them prior to proceeding with treatment.

The criteria for manic disorders in children are the same as the criteria for both adolescents and adults. Although is has been argued that a chronic mood-labile state depicts childhood mania, it has also been suggested that the classic depiction of mania is necessary in order to diagnose a child with BPD (Faedda et al., 2004). Symptoms of BPD are different in children than adults (Dunner, 2005). Irritability, ultra rapid cycling, and
mixed mania are more common in younger age groups. Disruptive behavior disorder and ADHD are the two most common co-morbid disorders.

Children who present with manic symptoms and a high degree of impairment may still not meet the DSM-IV diagnostic criteria (Biederman, Wozniak, & Kiely, 2000). In these cases, the DSM-IV categorizes these children as having BPD-NOS, cyclothymia, or both (Ahn & Frazier, 2004). These youth may experience severe impairments and subclinical cases should be monitored for the development of psychopathology over time.

Settings

The difficulty that arises in diagnosing a child with BPD is that many children only present with symptoms at home (Carlson, 2005). It has been noted that informants frequently disagree about a child’s symptomatology in almost all of the child psychiatric disorders. Although reasons may vary for the difference in reports, it is possible that one informant has a higher threshold than another for tolerating or observing psychopathology. Additionally, children are different in varying settings. The question that arises is what this difference indicates to professionals.

Carlson and Youngstrom (2003) investigated the variance in informant reports. Children with symptoms of mania with parent-only endorsements were compared with children with manic symptoms with parent and teacher endorsements. It was found that children whose parents and teachers both reported manic symptoms were more likely to have psychologists and nursing staff independently observe manic symptoms on subsequent hospitalizations. On the other hand, if there had only been one person reporting the child’s symptoms, symptoms of mania in a hospital were significantly less frequent. Although Carlson and Youngstrom (2003) did not provide explanations for the
differences, Carlson (2005) offered several explanations, including: (a) children may act differently in public and express their frustration and fury in places and with people whom they are comfortable; (b) children may behave better at school because the structure is better than home; and (c) in some cases, parents, siblings, or both, have a psychiatric disorder and the inconsistency, stress, or lack of supervision exacerbates the chances of a child decompensating. The varying reports from informants provide more support for the recommendation to consider collateral sources of information in an assessment for pediatric BPD.

**Characteristics of BPD**

Characteristics of pediatric BPD have been noted over time. The presentation of mania in children is considered atypical (McClellan, 2005). Children frequently present with recurrent periods of irritability and agitation (Biederman et al., 2000). Children with BPD typically have explosive and emotionally reactive outbursts, but the DSM-IV childhood psychiatric diagnostic categories do not account for these behaviours (McClellan, 2005). The qualitative difference between children and adults raises the question of whether or not these children depict BPD or another illness. However, differences between mania in children and the classic depiction of adult-onset mania may be accounted for by developmental differences. Furthermore, the relationship between childhood BPD and adult BPD may be irrelevant. Children who demonstrate the atypical characteristics of BPD typically have mood and behavioural dysregulation problems that are not accounted for in diagnostic categories. These children are likely experiencing impairments in areas of their lives and are difficult to treat.
The lack of clear episodes of mania can make diagnosing a child with BPD a challenge (Findling et al., 2001). The use of the DSM-IV “rapid cycling” modifier does not apply well to children because of the different presentation of mania (Kowatch et al., 2005). Children with BPD are best conceptualized with severe mood dysregulation and multiple, intense, and prolonged daily mood swings. This mixed presentation frequently includes longer periods of irritability and shorter periods of euphoria. Comorbid diagnoses are common and can complicate the diagnosis of BPD.

*Early-onset versus Late-onset BPD*

Schurhoff et al. (2000) compared two groups of individuals with BPD, an early-onset group and late-onset group, to examine the clinical features, comorbidity, and familial risk. Although these findings suggest a difference in clinical expression and familial risk between the two groups, it is important to note that the early-onset group included both children and adolescents and may not be specific to pediatric BPD. In this study, the early-onset group was associated with a more severe form of BPD. This group was also associated with a higher risk of affective disorders among first-degree relatives. Individuals with early-onset BPD are more likely to be diagnosed with BPD-I, have more psychotic features, and higher proportion of mixed episodes. There was also a comorbidity with panic disorder and Schurhoff et al. (2000) suggested that it may be a marker of genetic heterogeneity in BPD. The high comorbidity may also help to identify a subtype of BPD.

**DIFFERENTIAL AND COMORBID DIAGNOSES**

Regardless of age, there are specific characteristics that distinguish BPD from other disorders (Barton, 2001). The characteristic presentation of BPD involves
experiencing high and low moods. Specifically, high moods are referred to as mania and low moods are referred to as depression. Despite the observed cycles in mood, diagnosing a child with BPD is a difficult process. Children with BPD are more likely than not to be diagnosed with an additional disorder (Kowatch & DelBello, 2005). The presence of another disorder can complicate the presentation of BPD and the assessment and treatment process. This section will discuss the differential and comorbid diagnoses relevant to pediatric BPD.

**BPD and ADHD**

The most common comorbid disorder for children with BPD is ADHD (Kowatch & DelBello, 2005). Children with early-onset BPD are often diagnosed with ADHD when the child may actually have both BPD and ADHD (Geller & Luby, 1997). Research studies have provided evidence for a high correlation of BPD and ADHD in children (Barton, 2001). Geller and Luby (1997) suggested that ADHD with hyperactivity may be the first indicator of early-onset BPD. There is a higher incidence of ADHD in children with early-onset BPD than those individuals with later-onset BPD.

ADHD and BPD share common symptomatology and it may be difficult for professionals to differentiate between the two disorders (Barton, 2001; Biederman, Mick, Faraone, & Wozniak, 2004). However, children with ADHD do not have elated mood, grandiosity, hypersexuality, decreased need for sleep, racing thoughts, or other symptoms of mania that are present in children with BPD and ADHD (Geller et al., 1998).

There is symptomatic overlap between ADHD and pediatric BPD (Biederman et al., 2003). Researchers have demonstrated that children with a diagnosis of mania or BPD have symptoms that overlap with ADHD (Geller et al., 2002). This relationship appears
to be related to the onset age of BPD (Wozniak, 2005). Consideration of attention-deficit hyperactive disorder (ADHD) is an important differential diagnosis in early BPD because of the overlap of symptoms (Faedda, Baldessarini, Glovinsky, & Austin 2004; Scheffer & Niskala Apps, 2004). Symptoms of ADHD that may overlap with the diagnostic criteria for mania include distractibility, physical hyperactivity, talkativeness and pressured speech (Wozniak, 2005). ADHD and BPD share similar features and misdiagnosis can occur, especially in children (Kim, & Miklowitz, 2002). Individuals diagnosed with BPD present with behaviours that would support a diagnosis of ADHD, but BPD is not common in people with ADHD. A clear understanding of how to differentiate between ADHD and BPD in children is needed.

The rate of children with BPD who have been diagnosed with ADHD is high. However, the age of onset modifies the risk of comorbidity. These disorders also have similar diagnostic criteria in the DSM-III-R. Out of seven criteria listed in the DSM-III-R for a manic episode, three of the criteria also identify ADHD. Two techniques have been devised to avoid counting the same symptoms toward the diagnosis of both disorders. These techniques correct the overlapping diagnostic criteria that are used to evaluate the association between ADHD and pediatric BPD.

The two methods for correcting the overlapping diagnostic criteria are the subtraction method and proportion method (Biederman et al., 2003). In the subtraction method, the overlapping symptoms are not considered in making the diagnosis. In the proportion method, overlapping symptoms are not considered, but the diagnostic threshold is lowered and the same proportion of symptoms from the reduced set are as necessary as the proportion required for the diagnosis. In a study assessing the influence
of comorbid symptoms on diagnosis, researchers utilized the subtraction and proportion method and found that comorbid diagnoses are not a methodological artifact caused by shared diagnostic criteria (Milberger et al., 1995, as cited in Biederman et al., 2003).

There is a high comorbidity of BPD and ADHD in children (Biederman et al., 2004), but rates of comorbidity may vary depending on the subtype of ADHD (Biederman et al., 2003). Masi et al.’s (2001) naturalistic study of children and adolescents with BPD indicated an association between childhood-onset BPD with comorbid ADHD. It was suggested that this finding provides evidence for the idea that ADHD is a developmental marker for an early-onset type of BPD.

Both ADHD and BPD are related to high morbidity and disability. The comorbidity may mean the clinical presentation is more complex. ADHD can also affect the impulsivity of a severe mixed state and comorbidity could be lethal. Further research is needed on the rates of children with BPD and the different subtypes of ADHD.

Given the similar characteristics in pediatric mania and ADHD, a screen for prepubertal BPD is needed to differentiate between the presence of the disorders (Tillman & Geller, 2005). Additionally, the high comorbidity of ADHD in children with BPD also adds to the need for a screening tool. In a study by Tillman and Geller (2005), the Conners’ Abbreviated Parent Questionnaire was administered, as well as additional instruments, to determine if it was an effective tool to differentiate between ADHD and mania. The Conners’ Abbreviated Parent Questionnaire appears to be a promising tool as a screen for prepubertal BPD. This instrument provided good psychometrics as a screen for outpatient prepubertal BPD.
**BPD and Disruptive Behaviour Disorders**

Symptoms of childhood BPD must also be differentiated from disruptive behaviour disorders, including oppositional defiant disorder and conduct disorder (Weckerly, 2002). Children with mania have a high rate of oppositional defiant disorder (ODD) (Wozniak, Biederman, Kiely et al., 1995, as cited in Wozniak, 2005). Conduct Disorder (CD) is another common disorder that is comorbid with BPD (Kowatch & DelBello, 2005). Symptoms of disruptive disorders that often overlap symptoms of mania include restlessness, agitation, distractibility, aggression, sleep disruption, and poor school performance. Aberrant behaviours that are consistent with a diagnosis of CD may be related to the behavioural disinhibition of BPD (Biederman, Mick, Faraone, & Wozniak, 2004). These behaviours may also be caused by the irritability and low frustration tolerance that frequently accompanies pediatric BPD. Since childhood BPD has serious implications, the presence of CD in addition to BPD can complicate the situation.

It is important to note that features of overlapping behaviours may distinguish mania or suggest its presence overlapping ADHD and/or a disruptive disorder (Kowatch & DelBello, 2005). Additionally, ADHD and disruptive disorders do not present with primary features of mania, including psychosis, flight of ideas, euphoria, and grandiosity.

The association between conduct disorder and BPD in youth may be related to the criteria for diagnosis of CD and the characteristics of pediatric BPD. Specifically, children with BPD may have severe irritability, impulsivity, and aggression (Ahn & Frazier, 2004). Biederman, Faraone, Chu, and Wozniak (1999) completed a retrospective study comparing youth with mania, youth with conduct disorder, and youth with both
mania and conduct disorder. The profiles of symptoms for conduct disorder were similar for youth with or without mania. These findings led the researchers to suggest that CD and BPD were independent diagnoses. Additionally, children with both BPD and CD had higher levels of aggression and higher rates of ADHD and ODD. These youth were also more restless and more likely to have worse judgment than youth with mania alone. Biederman et al. (1999) suggested that the presentation and prognosis can become worse if CD is also present.

Aggression in children with BPD occurs in the context of an irritable mood and is highly reactive in nature, whereas children with conduct disorder do not present with aggression in the same context and tends to be planned and goal oriented (Wozniak & Biederman, 1996). Furthermore, BPD is often associated with the sudden onset of severe behavioural difficulties (James & Javaloyes, 2001). This sudden onset contrasts with the longer-standing behaviour difficulties demonstrated in CD. A family history of affective disorders, rather than disruptive behaviour, may help a professional in diagnosing a child with BPD.

Depression

Prediction of BPD in children who present with depression is a problem (Faedda et al., 1995). It has been suggested that psychomotor retardation, hypersomnina, hyperphagia, psychotic features, multiple relatives with affective illness, and new agitation or hypomanic during antidepressant treatment are predictive features. However, there is a lack of sufficient research to accurately estimate the proportion of pediatric patients with depression or dysthymia who are later diagnosed with BPD.
Psychosis

Psychotic symptomatology in children with BPD is higher than in adults (Geller et al., 2000). The presence of psychotic symptoms can be very pronounced in children (Ahn & Frazier, 2004). The pronounced psychotic symptoms and disorganization can lead to a significant proportion of children being misdiagnosed with schizophrenia. Psychotic symptoms commonly reported include hallucinations, ideas of reference, and delusions (Geller et al., 1998). Werry, McClellan, and Chard (1991) distinguished childhood BPD from schizophrenia by noting that children with schizophrenia often had more premorbid social withdrawal and anxiety and a more insidious onset of symptoms. Children with schizophrenia also had more mood-incongruent delusions and hallucinations and a longer course of illness.

Biederman et al. (2004) found that psychosis is more common among children who met the criteria for BPD than children with ADHD. Consequently, children with psychosis may be misdiagnosed as having schizophrenia. The literature on psychosis and BPD suggests that psychosis may be a correlate of BPD.

Anxiety

Children with BPD also have a high rate of anxiety disorders (Wozniak, 2005). In a sample of youth assessed for BPD over the last seven years, there was a high comorbidity of children with BPD and anxiety disorders (Biederman et al., 2004). Biederman et al. report that this finding is consistent with data that reveals comorbidity between anxiety disorders and BPD.
Masi et al. (2001) completed a naturalistic study of outpatient youth diagnosed with BPD. The findings suggest a high rate of comorbid anxiety disorders with only a few subjects in this sample without an anxiety disorder. Comorbid externalizing disorders have received more attention and research than internalizing disorders. However, Masi et al. (2001) suggested their findings indicate that anxiety disorders may be more represented in youth with BPD. Additionally, there was a high rate of anxiety disorders preceding onset of BPD. Limitations of this study include the retrospective design, poor reliability of children and parents’ recall, potential for misdiagnosis of comorbid disorders, and standard clinical assessment that could affect informant and clinician bias.

**TREATMENT APPROACHES**

Treatment approaches have not been adequately studied (Faedda et al., 1995; Geller & Luby, 1997; Wolf & Wagner, 2003). Given the concerns about diagnostic criteria for pediatric BPD, there are implications in evaluating the efficacy of treatment. Systematic evaluation of treatment options for BPD in children is lacking. Treatment options for BPD in children are often based on research on the efficacy of interventions for adults (Scheffer & Niskala Apps, 2004). This section will discuss the research on treatment approaches for children with BPD.

**Current Understanding**

Despite these limitations, researchers have suggested that the treatment of children with BPD is a multimodal approach (James & Javaloyes, 2001). Furthermore, an assessment for comorbid disorders and whether or not the existence is mood dependent is needed. Treatment of pediatric BPD needs to take into account the age and developmental context of the disorder. Specifically, the child’s age and development will
have implications for the presentation of the disorder. A treatment approach should involve both the parents and the child. However, James and Javaloyes (2001) cautioned about limitations of the child’s involvement and points out that a child’s mental health may pose concerns for the child’s competency.

The level of the child’s behavioural disturbance, risk of harm or suicide, need for medical supervision and level of family support are factors to consider when determining the treatment setting (James & Javaloyes, 2001). It is recommended that a manic child receive inpatient treatment until there are no safety concerns and the environment can be adapted to the child’s needs.

**Pharmacotherapy**

Pharmacotherapy is considered the first line of treatment for BPD (Weckerly, 2002). However, the area of pharmacotherapy in treating youth with BPD remains controversial because of the lack of long-term research (Costello et al., 2002; Ahn & Frazier, 2004). The lack of research negatively affects the ability to provide effective and timely interventions for BPD. Additionally, administering medication to children can be a concern because of their development. Psychotropic medications used to treat adults with BPD are used to treat children and good responses have been noted (Kowatch & DelBello, 2005). There needs to be long-term research that provides support for the safety and effectiveness of pharmacotherapy in treating children. Furthermore, pharmacological interventions used for behavioural disorders can exacerbate mania in adults (Costello et al., 2002). Research to evaluate the impact of pharmacological intervention on children is needed.
Research on treatment options for BPD in a pediatric population has not been systematically evaluated (Faedda et al., 1995; Scheffer & Niskala Apps, 2004). Despite this limitation, the treatment options that appear most promising include the antimanic and possibly mood-stabilizing anticonvulsants carbamazepine and valproate. These medications have been used for adults with BPD and the effectiveness and safety have been demonstrated in use for children with epilepsy. However, there have not been any randomized controlled trials for a pediatric population (Weller et al., 2003). Neuroleptics, atypical antipsychotics, sedatives, and various antihypertensive agents are also options for treating pediatric BPD, but they have not been systematically evaluated for this population (Faedda et al., 1995). The lack of research on treatment options for BPD in children is a concern. Research that focuses on the effectiveness and safety of treatment options is important.

Pharmacologic treatment of pediatric BPD can be divided into two stages (James & Javaloyes, 2001). In the first stage, focus is on the acute treatment of mania or depression. In the second stage, the focus is on prophylaxis. During acute illness, individuals can progress through stages of mania and then depression before recovery. It is suggested that multiple changes to medication and polypharmacy be avoided. Generally, it is recommended that children with BPD be treated for an extended time period to manage the frequent relapse associated with early-onset BPD. There is a need for randomized, double-blind, controlled trials in treating children with BPD to examine the safety and efficacy of the commonly used psychotropics and to compare them to one another (Weller, Calvert, & Weller, 2003).
Lithium

A significant portion of the literature on pharmacotherapy focuses on lithium. Lithium is the mood stabilizer used for children with BPD that has been best studied (Kowatch & DelBello, 2005). Although the effectiveness of lithium has been evaluated in juvenile BPD, it primarily focused on an adolescent population who presented with mania similar to adults (Faedda et al., 1995; Weller et al., 2003). Based on the current research that suggests a unique presentation of BPD in children, this research may not be applicable to a younger population. Additionally, this research is viewed as variable in quality and limited in quantity.

There are not any controlled long-term studies of lithium in pediatric BPD. However, DeLong and Aldershof (1987) completed a retrospective study examining the long-term efficacy of lithium on treating childhood behavior disorders. The participants typically had been treated extensively with psychiatric and pharmacological interventions that were ineffective or unsatisfactory. Lithium was found to be most effective for children with BPD. Additionally, the beneficial effects of lithium were maintained for more than a decade. However, the data does not indicate the outcome of behaviourally disordered children treated with lithium when they are adults. A significant limitation of this study is the question of the participants’ diagnoses. Specifically, the diagnostic categories may represent excessive splitting. It was suggested that children who benefited from lithium may represent a form of childhood BPD.

DeLong and Aldershof (1987) recommended a trial of lithium treatment in refractory childhood behavioural disorders if clinical features are present, including: (a) cyclic affective extremes, especially hateful hostile anger and manic overexcitement; (b)
family history of affective disorder, especially BPD; (c) aggression; (d) prominent neurovegetative disorders, especially hyperphagia, hyperdipsia, salt-craving, and encopresis. Symptoms of pediatric BPD are likely more extreme than adult BPD and neurovegetative symptoms are more prominent. A prospective study is needed to understand BPD and pharmacological interventions. It will be important to compare a treated group of children with an untreated control group and to follow these groups to understand the long-term implications of pharmacological treatment.

Conventional mood stabilizers, such as lithium, valproic acid, or carbamazepine, have demonstrated low effect sizes in children with BPD. Recent studies have examined whether or not a combination of various agents will lead to better outcomes. Traditional mood stabilizers and atypical antipsychotic medication have become increasingly used to treat BPD in children (Kowatch & DelBello, 2005). However, there are few controlled trials of the use of this medication to treat pediatric BPD.

Common side effects of lithium in children include hypothyroidism, nausea, polyuria, polydipsia, tremor, acne, and weight gain (Kowatch & DelBello, 2005). These side effects were noted in both children and adolescents and may not be specific to a child population. During the course of treatment, lithium levels and renal and thyroid function should be monitored at baseline and six months, similar to the administration of lithium in adults (Kowatch & DelBello, 2005).
Atypical Neuroleptics

Atypical neuroleptics have become increasingly more common in treating children with BPD (Biederman, McDonnell, Wozniak, Spencer, Aleardi, Falzone, et al., 2005). This class of agents typically has a combined dopaminergic and serotonergic effects on youth. The mood stabilizing properties appear to work together and there is a decreased risk of tardive dyskinesia than conventional, first-generation neuroleptics. There have been recent studies examining the effectiveness and tolerability of olanzapine, risperidone, and quetiapine in managing children with BPD. An eight-week, open-label, prospective study of risperidone monotherapy for youth with BPD found that risperidone was associated with significant short-term improvement in symptoms of pediatric BPD (Biederman et al., 2005b). However, placebo-controlled, double-blind studies are needed to confirm these results.

Aripiprazole

Aripiprazole is a promising medication for treating acute mania in adults (Keck et al., 2003), but data on its use in children is limited (Biederman et al., 2005). Recently, a large-scale, randomized clinical trial documented the safety and efficacy of aripiprazole in treating adults with BPD (Biederman et al., 2005). Researchers found a favourable adverse-effective profile for this compound. Aripiprazole could potentially be a promising treatment option for children with BPD because of its pharmacological profile in combination with the favourable adverse-effect profile.
Biederman et al. (2005) conducted a systematic chart review of outpatient youth with a diagnosis of BPD and bipolar spectrum disorder treated with aripiprazole with or without additional treatment options. Although these findings may be affected by observation and assessment bias because of the chosen methodology, the findings provided support for the use of aripiprazole in treating children with BPD (Biederman et al., 2005). Specifically, treatment with aripiprazole was associated with a robust response and well tolerated. There were no serious adverse effects experienced by the youth. Improvement in symptoms did not appear to be related to tranquilization or sedation. The most common side effects appeared to be insomnia and nausea. Weight gain did not appear to be a concern as reported by parents or clinicians, but Biederman et al. (2005) noted that they did not adequately quantify this variable. The response rate (70%) was consistent with open-label studies of compounds in this class of medication. Additionally, the youth in this study had previously been aggressively medicated with multiple compounds with limited success. Although additional research is needed to provide support for the efficacy and tolerability of aripiprazole, initial results appear promising. Initial experience with aripiprazole indicates that dosing strategies for youths may differ from those for adults (Findling et al., 2003). Randomized, controlled trials with this agent in pediatric BPD may also be warranted.
In addition to the potential assessment bias, there is potential for confounding results. The majority of the subjects took aripiprazole in combination with other medications (Biederman et al., 2005). Consequently, the findings of this study do not provide evidence for whether or not aripiprazole is responsible for improvements or if it was a combination of aripiprazole and additional psychotropic medications. Moreover, the dosage of aripiprazole during the study remained relatively low. It is uncertain whether or not aggressive treatment regimens with aripiprazole would prevent the need for additional antimanic treatments or the safety of a higher dosage. Objective measures of weight, electrocardiogram, and prolactin were not administered to evaluate the adverse-effect of the medication. The results of this study highlight the need for prospective controlled clinical controls of youth with BPD being treated with aripiprazole.

*Sodium Divalproex*

Sodium divalproex is a commonly used psychotropic for treating children with BPD (Kowatch & DelBello, 2005). However, there have not been any placebo-controlled studies published on the use of divalproex in children. Wagner et al. (2002) conducted the largest open-label study on sodium divalproex treatment for youth with BPD. In this study, improvement was observed to be fairly rapid and adverse side effects were generally mild or moderate in severity. Common side effects included headache, nausea, vomiting, diarrhea, and somnolence. The results of this study provide support for the safety and efficacy of sodium divalproex in children. However, the results are limited because of the small sample size at the end of the study, variability in the duration of open-label treatment, and lack of placebo control. Additional side effects commonly
reported in the literature include weight gain, sedation, and tremor (Kowatch & DelBello, 2005). Despite these promising results, further research is warranted with a focus on double-blind, placebo-controlled trials.

**Carbamazepine**

Carbamazepine is a medication that has been frequently administered for children and adolescents with seizures (Kowatch & DelBello, 2005). It is also a medication that has been prescribed to treat affective disorders, particularly BPD, when treatment with lithium bicarbonate is not successful (Evans, Clay, & Gualtieri, 1987). There have been several case reports and series on the success of carbamazepine in both a monotherapeutic approach and as an adjunctive treatment for children with BPD (Kowatch & DelBello, 2005). Carbamazepine appears to be effective in treating affective symptoms, elements of emotional instability, and anger or aggressive outbursts (Evans et al., 2005). However, caution must be taken in administering carbamazepine to a child because of concerns for a child’s health. During treatment, it is imperative that a child’s blood levels are frequently monitored (Kowatch & DelBello, 2005). Side effects that may occur in a child include aplastic anemia, severe dermatologic reactions (i.e., Stevens-Johnson syndrome), hyponatremia, nausea, and sedation. Evans et al. (1987) also reported the possibility of behavioural side effects, including irritability, developmental regression, agitation, obsessive thinking, auditory hallucinations, delirium, psychosis, combativeness, “thinking problems”, insomnia, aggressiveness, hyperactivity, and paranoia. Given that these adverse effects can mimic symptoms of pediatric BPD, it is important that medication be decreased or discontinued if a child’s symptoms do not
improve. Further studies are warranted to determine whether or not the adverse effects of this medication eliminate them as a treatment option for children with BPD.

**Novel Antiepileptic Medication**

The development of antiepileptic medications for treating epilepsy might also be a development for treating pediatric BPD, since these medications may have mood-stabilizing properties (Kowatch & DelBello, 2005). The research on the efficacy and tolerability of antiepileptic medication for treating childhood BPD is limited. However, it has been suggested that these agents may be beneficial as adjuncts for treating manic and hypomanic episodes. Oxcarbazepine is an analogue of carbamazepine and appears to be a promising agent in treating adult BPD (Kowatch & DelBello, 2005). There is no data on the use of this agent in pediatric BPD. Lamotrigine has been reported as an adjunctive treatment for children with BPD (Kowatch & DelBello, 2005). However, the use of lamotrigine has been limited because of the potentially lethal cutaneous reactions (i.e. Stevens-Johnson syndrome) and toxic epidermal necrolysis.

**Atypical Antipsychotics**

Researchers have found atypical antipsychotic medication to be effective in treating adult BPD (Kowatch & DelBello, 2005). These agents have antipsychotic activity and mood-stabilizing properties. Case studies and open-label reports on the efficacy of atypical antipsychotics in treating pediatric BPD have reported favourable effects (Kowatch & DelBello, 2005). Medications that have been proven to be successful in treating pediatric BPD in these studies include clozapine, risperidone, olanzapine, and quetiapine (Dunner, 2005; Kowatch & DelBello, 2005). The findings of these studies also provide evidence that these agents might be more effective in treating children with BPD.
than traditional mood-stabilizing psychotropics. At the present time, there are ongoing controlled trials for treating children and adolescents with mania with atypical antipsychotic agents, including risperidone, olanzapine, and quetiapine. These controlled trials will likely add to the current understanding of the efficacy of treating children with BPD with atypical antipsychotics.

Atypical antipsychotics appear to have beneficial effects in treating pediatric BPD, but there are side effects associated with their use (Kowatch & DelBello, 2005). Clozapine and olanzapine have been associated with weight gain in adolescents. This side effect may also be a concern in treating children with these agents and further studies should examine whether or not it is an adverse effect. Additionally, consequences associated with being overweight can occur, such as Type II diabetes. Researchers have found that atypical antipsychotics are effective in treating BPD in children, but there are side effects that professionals and families need to be aware of.

Antidepressants

There is a lack of information on treating depression in children with BPD (Kowatch & DelBello, 2005). The role of antidepressants in treating depression in pediatric BPD is unclear. Antidepressants are typically not the first line of treatment for depression in BPD or for the whole disorder (Kidd, 2005). Antidepressants can be effective in treating depression in BPD, but they can also cause switching to mania and trigger rapid cycling (Kowatch & DelBello, 2005).

Kowatch and DelBello (2005) reported that a retrospective study found SSRIs to be effective in treating depression without interfering with the effects of mood stabilizers on mania. This report also found mood stabilizing effects with the use of antidepressants.
A prospective chart review on a large clinical sample of children with a DSM-III-R diagnosis found that SSRIs were selectively effective in the treatment of pediatric BPD (Biederman, Mick, Spencer, Wilens, & Faraone, 2000). It was also found that SSRIs were associated with destabilizing effects on symptoms of mania in children with depressive symptoms in the absence of mania. Furthermore, the antimanic effects of mood stabilizers to treat the active symptoms of mania were not inhibited by concomitant use of SSRIs. Biederman et al. (2000) highlight the common presence of youth with depression in clinical practice and the need for an assessment that includes a lifetime history of BPD when making clinical decisions in depressed children. The finding that SSRIs can be selectively efficacious and destabilizing in youth with BPD has significant implications for clinical practice. Children typically present with mixed states and this presentation can complicate the practice of pharmacotherapy for these children.

Concomitant Pharmacotherapy

In a study by Duffy et al. (2005), the rates and correlates of concomitant pharmacotherapy were assessed in children and adolescents treated by psychiatrists in varying clinical settings. Researchers found that concomitant pharmacotherapy was highest in children with BPD and schizophrenia. After adjusting for variables, BPD was a strong predictor of concomitant pharmacotherapy. Limitations of this study include the small sample size, data older than five years, and reliance on psychiatrist-reported data. There were no independent assessments of the subjects to confirm the validity of the diagnostic information. Additionally, there was no information on compliance with prescribed medication or access to information about prior treatment history, response to treatment, and the process leading to concomitant pharmacotherapy.
Kowatch, Sethuraman, Hume, Kromelis, and Weinberg (2003) studied the effectiveness of combination pharmacotherapy in children and adolescents with BPD in a six-month period of prospective, semi-naturalistic treatment. Kowatch et al. (2003) found that over half of the subjects needed a stimulant, atypical antipsychotic agent, or an antidepressant agent in addition to one or two mood stabilizers during the extension phase of treatment. Furthermore, the response rate to combination therapy was very good. Kowatch et al. (2003) reported that 80% of the subjects responded to combination therapy with two mood stabilizers after not responding to a single mood stabilizer during the acute phase of treatment. Finally, results showed that a significant percentage of subjects needed treatment for comorbid ADHD before their overall functioning (i.e., mood and attention) improved. A small sample size, variable length of treatment during the acute phase, lack of a double-blind study, and lack of strict criteria for the addition of psychotropic agents limit the study’s findings. Additionally, subjects were outpatients and results may not generalize to inpatient children, whom are more likely to be experiencing more severe and chronic symptoms of BPD. The use of pharmacotherapy for children with BPD is often complicated by comorbid diagnoses, such as ADHD. It is necessary for further investigation using controlled studies of combination therapy with children with BPD.

Treatment for Comorbid Diagnoses

Comorbid diagnoses have implications for the prognosis of children with BPD and children frequently require targeted interventions aside from ones specific to treating BPD (Ahn & Frazier, 2004). In treating children with BPD, it is important to stabilize their moods, then comorbid disorders are treated (Kowatch & DelBello, 2005). A
significant number of prepubertal youth with BPD present with mixed mania or hypomania and respond best to traditional mood stabilizers or atypical antipsychotics. Atypical antipsychotic agents are becoming the first line of treatment for children with mania or hypomania because of their effectiveness and ease of use. Children with classic euphoria and an absence of psychotic symptoms may benefit from a trial of lithium. However, children may be unable to continue taking lithium because of the associated side effects, such as weight gain, exacerbation of acne, and hypothyroidism. Psychotic symptoms along with the presence of mania may best be treated by atypical antipsychotics.

*Electroconvulsant Therapy (ECT)*

ECT is a treatment option that has been recommended for treating BPD in resistant cases (Kidd, 2005). It is also a form of therapy that has been recommended in life-threatening mania and depression (James & Javaloyes, 2001). ECT is rarely used in treating children with BPD (Kafantaris, 1995). However, there is a lack of research on the safety and efficacy of ECT in pediatric cases. Additionally, Kidd (2005) reported that ECT is not allowed to be used on children under 16 years of age in some locations.

*Nutrition*

Alternative treatments cannot be recommended for treating pediatric BPD because there is only preliminary data available (James & Javaloyes, 2001). Researchers have proven that vitamins B3, B6, B12, and folic acid are beneficial in treating mental health disorders when prescribed at levels beyond the common mixed formulation of B vitamins (Kidd, 2005). Studies investigating the efficacy and safety of alternative treatments for children with BPD are needed.
Psychotherapy

Although pharmacotherapy is the primary treatment for pediatric BPD, individual and family psychotherapy are also necessary forms of treatment (James & Javaloyes, 2001). In-depth individual psychotherapy is not recommended in the treatment of pediatric BPD. However, individual supportive work is a critical component of treatment.

In treating BPD, it is recommended that individuals avoid emotional stress (Kidd, 2005). Additionally, stabilizing the home environment and daily routines, avoiding aggressive styles of living, excessive goal seeking, and personal confrontations are encouraged. Psychotherapy can be useful in helping individuals to achieve these recommendations.

Despite the use of psychosocial interventions on children with BPD, there have not been any large-scale scientific studies on it (Ahn & Frazier, 2004). Currently, there are no studies published on the feasibility and efficacy of psychosocial interventions for children with BPD (Pruett & Luby, 2004). Geller et al.’s (2002) retrospective study reported that family therapy did not predict recovery or relapse. In this study, youth who received individual and/or group therapy were less likely to recover. Geller et al. (2002) noted that youth receiving multiple interventions tended to be more ill and had more significant psychopathologies and more likely to demonstrate psychosis.

Pavuluri, Graczyk, Henry, Carbray, Heidenreich, and Miklowitz (2004) described a child and family focused cognitive behavioural therapy (CFF-CBT) approach to pediatric BPD. CFF-CBT was described as a developmentally sensitive psychosocial intervention for pediatric BPD and was intended to be used in conjunction with medication. This intervention integrates principles of CBT with family focused therapy.
and bases it on the biological theory of excessive reactivity. Environmental stressors that are associated with pediatric BPD are also targeted as part of treatment. CFF-CBT has a strong foundation, both theoretically and conceptually. Initial results indicate that this approach is promising in treating children with BPD. Additionally, preliminary results provide support for the feasibility of this intervention.

Multi-family psychoeducation groups (MFPG) are groups to help families of children with mood disorders in coping with their disorders. MFPG were developed and tested for treating adults, but limited studies were available on their use in children with BPD (Fristad, Goldberg-Arnold, & Gavazzi, 2002). Fristad et al. (2002) completed a pilot study on MFPG for families of children with mood disorders. Preliminary data supports the beneficial use of MFPG for treating children with BPD and major depressive disorder/dysthmia disorder (MDD/DD). Clinical concerns about facilitating groups for families of children with BPD or MDD/DD were discussed and concluded that it is both feasible and potentially beneficial. Given that this research was based on a combined group of families with children with BPD and MDD/DD, further research to examine the effectiveness of MFPG for families of children with BPD would be beneficial.

*Therapeutic Parenting and Education*

Regular therapy sessions with a licensed social worker, psychologist, or psychiatrist are often beneficial when treating children with BPD (Kidd, 2005). A focus on parenting strategies and education can help parents to have a better understanding of their child’s disorder and ways to provide appropriately respond to their child’s needs and any related difficulties. Furthermore, children with BPD may have special needs
educationally and these needs should be addressed through collaboration between the education professionals, child, and parents.
CHAPTER 4

POTENTIAL IMPLICATIONS

This project is designed to add to the existing knowledge on BPD in children. Although adult-onset BPD has long been recognized as having detrimental outcomes for adults, there have been controversial positions on the existence and presentation of child-onset BPD. A thorough literature review could be beneficial for professionals, since it will provide a comprehensive review and limitations on the present research. Research findings inform clinical decisions and the lack of research affects clinical practice. The current understanding of early-onset BPD is limited and further research may contribute to more accurate assessments of children, appropriate interventions, and improved outcomes. Pediatric BPD is associated with high rates of morbidity and impaired functioning. The controversy over the existence of pediatric BPD means that professionals may be unwilling to assess or diagnose a child for BPD until further research is completed. The lack of research also affects the assessment process in terms of the availability of reliable and valid assessment tools. Furthermore, a professional’s understanding of and ability to differentiate between psychiatric disorders and identify the existence of comorbid disorders will also affect the diagnosis of BPD. Psychologists and psychiatrists who assess children for BPD need to rely on the limited available research, but the diagnosis process heavily relies on a clinician’s judgment.

A comprehensive summary of the current research could potentially impact children and families who are struggling with mental health issues and receiving conflicting information from professionals. Parents who have a child demonstrating characteristics of BPD are likely to receive varying evaluations from professionals. An
eight-year-old girl who displays a severely irritable mood, hyperactivity and talkativeness, and violent behaviours towards peers and authority figures could potentially be diagnosed with pediatric BPD from one professional, yet receive diagnoses of ADHD and ODD with an intense temperament from another professional. These diagnoses are made based on the professional’s understanding of the literature and diagnostic criteria. The differing diagnoses are both applied to explain the child’s behavior and moods, but significant differences exist despite similarities in symptomatology.

Tillman, Geller, Frazier, Bering, Zimerman, Klages, et al. (2005) critiqued professionals’ abilities to distinguish between ADHD and childhood BPD. Tillman et al. (2005) argued that psychiatrists receive specialized training that enables them to distinguish ADHD from alternative psychiatric disorders. They also argued that practitioners who do not receive this specialized training may not be aware of the possibility that similar characteristics may exist in additional psychiatric disorders. It is recommended that practitioners refer children diagnosed with ADHD to a psychiatrist if children respond poorly to monotherapy with ADHD medication, course of the disorder worsens, or non-ADHD symptoms develop. However, Carlson and Kelly (2003) noted that intolerance to a stimulant does not necessarily indicate BPD in a child, but another strategy should be tried. Carlson (2005) indicated that emotionally labile children are difficult because of mood fluctuations. It was recommended that a good baseline of the child’s behavior be established. Changes from the baseline behavior do not necessarily indicate BPD, but will indicate a need to change the treatment protocol.
The question of whether or not pediatric BPD exists and research that supports the idea of a unique presentation raises implications for professionals. Specifically, the similarity of characteristics that define ADHD and pediatric mania needs to be differentiated. In addressing concerns about symptomatology, the presence of irritability in numerous child psychiatric disorders affects the ability to recognize and diagnose child-onset BPD. These concerns would best be addressed by the development of an assessment tool to accurately assess prepubertal mania. This assessment tool would need to highlight the expression of characteristics in age and developmentally appropriate ways. For instance, children are unable to max out a credit card on an impulsive shopping trip during a euphoric moment. Additionally, professionals need to be able to distinguish between a normal child happiness and expansiveness from a pathologically impaired elated mood and grandiosity. The developmental context of characteristics needs to be thoroughly considered. Standard diagnostic criteria for pediatric BPD that are developmentally appropriate and display high interrater reliability and validity is necessary (Geller & Luby, 1997).

The difficulties in accurately diagnosing a child with BPD also leads to concerns regarding the most appropriate and effective treatment for childhood psychiatric disorders. Professionals frequently rely on diagnoses to help understand the child’s problems and guide the clinical decision-making process. The lack of a diagnosis or incorrect diagnosis can have significant implications. Professionals often rely on current research to create and implement relevant interventions. This research is based on the assessments of the child and any diagnoses given. A child who is treated for BPD will most likely receive a differing treatment plan than a child who is treated for CD.
Additionally, the effective administration of psychotropic medication is reliant upon accurate diagnoses. This project also highlights the lack of large scale studies on individual and family psychotherapeutic approaches in treating childhood BPD. The lack of systematic research on the safety and efficacy of psychotropic medication for children with BPD is also an important implication. There is not a clear understanding of the psychotropic medication that is most effective and safe in treating manic, depressive, or mixed states. There also needs to be further research to evaluate the efficacy and safety of concomitant pharmacotherapy in comparison to treating a child with a single psychotropic medication. Furthermore, there is only preliminary data available on alternative treatments for pediatric BPD and further investigation is needed.

The project could also provide support to educate families of the expectations of BPD in children and provide a foundation for helping and advocating for children. For instance, this knowledge could help families to understand the characteristics and needs of a child with BPD and ways to support these needs. This information can assist parents in understanding their role in creating a stabilized environment for a child with BPD. Furthermore, the government and mental health agencies may also benefit from understanding BPD and what interventions would be most effective. The identification of children with BPD can be improved with a better understanding of the unique presentation of BPD. Accurate and efficient diagnoses can also have a significant impact on the child’s treatment and allow for increased success of the child. This knowledge can also be useful in obtaining money to support assessments and interventions for children who present with BPD.
This project can also enhance a counsellor’s knowledge on pediatric BPD and improve the effectiveness of counselling. A comprehensive overview of the symptomatology of childhood BPD can add to a counsellor’s ability to identify characteristics of BPD and refer a child to the necessary professional. This recognition can also provide for a more thorough and effective treatment plan. A counsellor’s understanding of the types of individual and family therapy that can be beneficial for treating a child with BPD can help in creating a treatment plan to meet the child’s needs and address areas of concern. For instance, an understanding of the type of home environment and daily routines that are both helpful and necessary in stabilizing a child with BPD can assist a counsellor in educating the family and developing treatment tasks.

Despite the need for research in pediatric BPD, it is a difficult disorder to research. Large and frequent samples are difficult when researching a younger population and legal and ethical issues are involved (Barton, 2001). Research studies on treatment in young children are difficult to conduct and frequently met with resistance. Additionally, legal and ethical issues affect the administration of medication to children for the purpose of research. Consequently, BPD is frequently studied in an adult population. As previously discussed, children do not have similar symptomatology as adults and the use of research in an adult population may not be representative of a younger population of individuals.

CONCLUSION

Pediatric BPD is controversial in the area of psychiatry. Research to date is suggestive of a unique presentation of symptomatology in children in comparison to adults. However, there is a lack of long-term research to provide evidence that children
with these unique characteristics have the same disorder as adults. Researchers have suggested that pediatric BPD may be a subtype of adult-onset BPD, whereas others have questioned if the disorder requires its own classification due to the differences. Despite this question, the diagnosis of BPD in childhood is made based on a professional’s understanding of the current literature and the application of the DSM-IV criteria that is specific to the symptomatology for adult-onset BPD. The use of criteria that may not accurately reflect the characteristics of pediatric BPD can affect accurate and timely diagnoses. The detriments of pediatric BPD are concerning, particularly when determining an appropriate treatment protocol can be a difficult process.
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