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Psychosocial Interventions for Children With Early-Onset Bipolar Spectrum Disorder

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Once considered virtually nonexistent, bipolar disorder in children has recently received a great deal of attention from mental health professionals and the general public. This paper provides a current review of literature pertaining to the psychosocial treatment of children with early-onset bipolar spectrum disorder (EOBPSD). Commencing with evidence of the emerging interest in this topic, we then focus on terminology, the rationale for studying EOBPSD in children, current research and clinical progress, possible explanations for the recent increase in recognition, and essential issues that form the foundation of effective psychosocial treatment. Next we explore areas of research with direct implications for psychosocial treatment. These include biological and psychosocial risk factors associated with bipolar disorder; and the psychosocial treatment of adult-onset bipolar disorder, childhood-onset unipolar disorder, and anger management in children. Following this, we discuss treatments being developed and tested for children with EOBPSD. Finally, we conclude with recommendations for future studies needed to move the field forward.

KEY WORDS: psychosocial treatment; children; bipolar disorder; manic depression.

Bipolar disorder in children was until recently a neglected area in developmental psychopathology. Over the last decade, bipolar disorder in children has received increasing attention from both the scientific community and the general public.

A recent literature review by the authors, using PsychINFO and Medline and relying on various key word combinations (i.e., children, pediatric, prepubertal, early-onset, child, bipolar disorder, manic depression, manic–depressive, and mania), revealed 174 articles and book chapters. A substantial recent increase in research was observed, with 26 items published before 1980, 36 during the 1980s, 66 during the 1990s, and 46 during the first 2 years of the new millennium. A similar pattern of growth has also occurred in the public sector. A search of Amazon.com revealed 18 books on the topic of bipolar disorder in children with 15 published since the year 2000. Websites devoted to the topic also have flourished (e.g., the Child and Adolescent Bipolar Foundation, established 1999, and the Juvenile Bipolar Research Foundation website, established 2002). As a further measure of general public interest in this topic, *Time* magazine recently featured the cover story "Young and Bipolar" (Kluger & Song, 2002).

CLINICAL VIGNETTE

John (not his real name), an 8-year-old Caucasian male, and his 35-year-old Caucasian mother, Mrs D., were recruited into our treatment study for children aged 8–11 with mood disorders after viewing a local television news story about the study. Mrs D. reported a paternal family history of bipolar disorder, drug and alcohol abuse, antisocial personality disorder, anxiety disorders, and learning disabilities. She recalled John was "very impatient and irritable" as an infant," and developed an extreme case of the "terrible two's"

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with multiple, long-lasting daily rages during which he was frequently physically and verbally aggressive. Along with "daily power struggles," verbal threats and severe distractibility, impulsivity, and hyperactivity, these rages continued until he started kindergarten. At this time, Mrs D. recalled John began having daily "mood swings" involving discrete hour-long episodes of rage accompanied by increased energy, distractibility, impulsivity above and beyond his usual level, rapid speech, racing thoughts, and involvement in many activities in and out of the home. John also began experiencing "lows" for more days than not, feeling he was no good, hopelessness, and, from the age of 7, wishing that he "was dead."

These mood swings continued to increase in frequency and severity and were often accompanied by a reduced need for sleep. They were exacerbated by the prescription of Adderall® and Dexedrine®, but later reduced by the introduction of Lithium. Still, within the last 2 weeks, Mrs D. observed John experiencing "daily 5–10 minute rages" during which he would "become more physically and verbally aggressive towards peers and try to break things." She further described John as "ready to jump out of his skin, he had constant energy . . . very distractible, very imaginative, [his] thoughts go from one thing to another . . . he changes topics and constantly tells me about new projects . . . [and becomes] very talkative, very fast, he has to get it out and interrupts constantly." Most recently, he "told teachers at school that he drank beer, smoked dope and had sex in his room with condoms," all of which his mother denied. John was given a bipolar disorder-not otherwise specified (BP-NOS) diagnosis, comorbid with attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD), separation anxiety disorder, specific phobia of the dark, and encopresis.

As this clinical vignette demonstrates, EOBPSD may present quite differently from the "classic" manic–depressive illness described in adulthood, which involves discrete and alternating episodes of mania and depression interspersed with symptom-free periods of recovery (Goodwin & Jamison, 1990). Children with EOBPSD often present with rapid cycling (i.e., $\geq$4 mood episodes per year) to continuous cycling (i.e., abrupt mood shifts $\geq$1 per day), "mixed" depressive and manic symptoms often in the form of intense irritable mood swings and an insidious and chronic course from early childhood (Biederman et al., 2000; Geller & Luby, 1997). In contrast, adults with "classic" manic–depressive illness typically present with less rapid cycling (i.e., <4 mood episodes per year), more interepisode recovery, discrete episodes of depression and mania, and an acute onset of symptoms (Goodwin & Jamison, 1990). Finally, children with EOBPSD, unlike adults with "classic" manic depression, usually have high rates of comorbidity, such as ADHD, oppositional defiant disorder (ODD), CD, and various anxiety disorders (Biederman, Faraone, Chu, & Wozniak, 1999; Findling et al., 2001; Geller, Zimmerman, et al., 2000; Lewinsohn, Seeley, & Klein, 2003; West et al., 1995; Wozniak et al., 1995).

This atypical presentation in childhood is at the core of the controversy that presently surrounds EOBPSD (Faraone, Glatt, & Tsuang, 2003). However, it is also important to recognize that there may be more similarities than differences between EOBPSD and the nonclassic adult "softer" bipolar phenotypes, in terms of irritable or mixed mood, comorbidity, and rapid cycling (see a review by Weckerly, 2002).

BACKGROUND

What Term to Use?

Various terms have been used to describe bipolar disorder in children, including pediatric, juvenile, early-onset, childhood, and prepubescent bipolar disorder. Rather than adding to this plethora of terms, we suggest the use of a term rooted in what we currently know, or more precisely do not know about bipolar disorder in children.

During much of the nineteenth and twentieth centuries, a biological approach to the explanation and treatment of bipolar disorder has predominated. In that context, it has made sense to refer to bipolar disorders that occur in childhood as *pediatric bipolar disorder*, as pediatrics is the "branch of medicine dealing with the development, care, and diseases of children" (Merriam-Webster Collegiate Dictionary, 2004). However, as with adult-onset bipolar disorder, more recent research suggests a medical approach alone is necessary but not sufficient. Although the term *prepubescent bipolar disorder* is frequently used, studies using this term have not consistently utilized an objective measure of pubertal status; rather, age has sometimes been used to make this determination. The term *juvenile bipolar disorder* has the unfortunate association with the legal term "juvenile delinquency" and the negative connotation of being defined as "physiologically immature or undeveloped" (Merriam-Webster Collegiate Dictionary, 2004).

The singular term *bipolar disorder* is also misleading as there appears to be a group of disorders (Bipolar I [BP-I], Bipolar II [BP-II], cyclothymia, and BP-NOS). Perhaps a more fitting reference would be bipolar spectrum disorders (Akiskal, 1983). Thus, we favor and will use the term *early-onset bipolar spectrum disorder* (EOBPSD) when describing bipolar disorder that occurs in persons younger than 18. Although controversy surrounds the existence of the so-called "soft phenotypes" within the bipolar spectrum (BP-II, cyclothymia, BP-NOS), as opposed to the "hard" or classic (nonmixed) manic–depressive illness BP-I phenotype (Akiskal, 2003), it is not currently known whether these subtypes actually do (or do not) represent a single clinical, developmental, or genetic continuum of related disorders (Akiskal, 2003). Therefore, in using the term *bipolar spectrum*, we, like Akiskal (2003), are referring to a theoretical cluster of "overlapping subtypes of bipolar disorder" (p. 2), which demands further examination.

Finally, as most research on bipolar disorder has been conducted with adults, typically without differentiating whether onset was in childhood, adolescence, or adulthood, we shall refer to these adult studies as addressing generic bipolar disorder (BPD).

Why Study EOBPSD?

In the past, bipolar disorder in children, particularly in its narrowly defined classic form of manic–depressive illness, was considered virtually nonexistent (Anthony & Scott, 1960). Although this "hard" phenotype is still rare in children, reports of "soft" EOBPSD phenotypes have become increasingly more frequent (NIMH, 2001). Although no epidemiological studies currently exist for children, Lewinsohn, Klein, and Klein's (1995) community school survey of 14- to 18-year-olds found lifetime prevalence rates for BP-I to be approximately 0.12% and BP-II and cyclothymia to be around 1%. They reported an additional 5.7% with subthreshold symptoms, multiple comorbidities, and associated psychosocial impairment, which may constitute a group of adolescents with BP-NOS. By comparison, the cross-national lifetime prevalence for adults with bipolar spectrum disorders ranges from 3 to 6% (Weissman et al., 1996). Lish, Dime-Meenan, Whybrow, Price, and Hirschfeld (1997) conducted a retrospective survey of 500 National Depressive and Manic–Depressive Association group members self-identified with "bipolar illness" (p. 263). Nearly one third (31%) recalled a variety of depressive and manic symptoms during childhood. An additional 28% reported onset during adolescence. Thus, over half of adults in this survey reported their symptoms began prior to adulthood. However, as Lish and colleagues acknowledge, because the diagnoses of their self-identified samples were not verified by a structured diagnostic interview, the occurrence of both "hard" or "soft" phenotypes and their age of diagnostic onset is unknown.

Second, EOBPSD is worthy of study due to the cost associated with its underdiagnosis and overdiagnosis. Underdiagnosis may occur if clinicians are reluctant to consider this diagnosis, in part because of its symptomatic overlap with ADHD, ODD, CD, and various anxiety disorders (Weller, Weller, & Fristad, 1995). With the recent academic, clinical, and public growth in attention to mania in children, and without an equivalent expansion of clinical training for assessment and diagnosis, overdiagnosis may also become quite common. Both over- and underdiagnosis have serious implications for the child and family, as misdiagnosis can lead to anxiety, helplessness, and hopelessness about the future. Under-diagnosis also may mean unnecessary suffering, delays in receiving effective treatment, and the recommendation of ineffective and sometimes harmful treatments. In contrast, overdiagnosis can set in motion stigmatization by self and others, long-term treatment including the use of multiple medications that frequently have significant side effects, and inappropriate medical, psychological, and community interventions.

Third, EOBPSD is associated with severe impairment and a negative prognosis. It wreaks havoc on family life, school functioning, and peer relationships (Lewinsohn et al., 2003). Over time, if left untreated, EOBPSD may have a prolonged course, be less responsive to treatment, and lead to legal difficulties, multiple hospitalizations, and increased rates of substance abuse and suicide (Findling et al., 2001; Geller et al., 2003; Lewinsohn et al., 2003).

Fourth, there are many unanswered questions about EOBPSD. Given its high rate of co-occurrence with other disorders and its atypical presentation compared to adults with BPD (Biederman, 1998; Carlson, 1999; Klein, Pine, & Klein, 1998), we need to examine what this phenomenon truly is; seek valid and reliable definitions, boundaries, classifications, and assessments for our subject matter; and explain why this phenomenon has become more recognized in the last decade.

Finally and most importantly, it is essential that we continue to study this relatively prevalent, often

misunderstood and devastating clinical phenomenon because there are very few empirically based treatments and no prevention strategies for EOBPSD. Yet, even with empirically supported treatments, clinicians may not utilize them and families with bipolar offspring may not access them. Lewinsohn et al. (1995) found that only 41% of young adults with BP-II and cyclothymia and 27% with subsyndromal bipolar symptoms utilized mental health treatments. Empirically supported treatments, therefore, must be transportable, available and accessible to the community if they are to be used outside of research settings.

Current Research and Clinical Progress

While there have been references to manic-like symptoms in children dating back to the eighteenth century (see Glovinsky, 2002, for an historical review through 1980), only recently have a cadre of research studies and clinical reports on EOBPSD become available. In 1997, the American Academy of Child and Adolescent Psychiatry (AACAP) published Geller and Luby's "Child and Adolescent Bipolar Disorder: A Review of the Past 10-Years" (Geller & Luby, 1997). In the same year, AACAP published the first assessment and treatment guidelines for this population: "Practice Parameters for the Assessment and Treatment of Children and Adolescents With Bipolar Disorder" (AACAP, 1997).

With widespread use of the Internet in the 1990s, parents of children with EOBPSD started online support groups. In 1999, many of these parents came together to form the Internet-driven national organization, Child and Adolescent Bipolar Foundation (CABF). The CABF website, *www.bpkids.org*, currently receives "over 100,000 unique visits per month" (Hellander, Sisson, & Fristad, 2003, p. 320). Three years later, in 2002, the Juvenile Bipolar Research Foundation (JBRF), *www.jbrf.org,* became the first charitable foundation dedicated to the support of research in EOBPSD.

On April 27, 2000, the National Institute of Mental Health (NIMH) Developmental Psychopathology and Prevention Research Branch and the Child and Adolescent Treatment and Preventive Intervention Research Branch invited some top researchers in the field to a small roundtable meeting for an "open-ended discussion of possible approaches to outstanding issues for research on the diagnosis of prepubertal bipolar disorder" (NIMH, 2001, p. 871). In the last 5 years, specific treatments for children with EOBPSD have emerged, including the first randomized controlled treatment studies of lithium (Geller et al., 1998), quetiapine augmentation of divalproex (DelBello, Schwiers, Rosenberg, & Strakowski, 2002), and multifamily psychoeducation (Fristad, Goldberg-Arnold, & Gavazzi, 2003).

Why Now and Why in America?

What can account for the substantial recent increase in interest in EOBPSD in the U.S.A.? First and foremost, we may be witnessing a "pendulum effect," whereby the subjective opinions and perceptions of researchers, clinicians, the media, and the public have shifted from the position that childhood mania was virtually nonexistent to the current "flavor of the month" position whereby it appears that "every child" has EOBPSD. With continued scientific progress, we anticipate the pendulum will swing back to the "middle" as we refine our ability to accurately identify "true" cases of EOBPSD.

A second reason for the current interest in this phenomenon may be related to the emerging concept of the bipolar spectrum (Akiskal, 1983) in adult psychopathology (see the special issue of the *Journal of Affective Disorders*, January 2003, which focused on bipolar spectrum disorders in adults). This concept has led to a broader view of what constitutes bipolar disorder in adults and youth, including both "hard" (psychotic and nonpsychotic BP-I) and "soft" (BP-II, cyclothymia, BP-NOS) phenotypes (Akiskal, 2003). Along with this expansion of diagnostic criteria for bipolar disorder, there has been an increase in the number and use of diagnostic structured interviews in the field in general. With broader criteria and more comprehensive methods to assess a wider range of symptoms, more children and adolescents are receiving diagnoses of bipolar disorder. Whether or not all these varieties of EOBPSD are valid will depend on their future ability to meet the five classic criteria for diagnostic validity described by Robins and Guze (1970) and extended by Cantwell (1975): clinical phenomenology, psychosocial factors, demographic factors, biological factors, family genetic factors, family environmental factors, natural history, and response to therapeutic intervention.

Two related trends noted during the 1980s and 1990s also may be involved. During this time period the field witnessed a dramatic increase in the recognition of childhood depression and ADHD, with an accompanying rise in prescriptions for

selective serotonin reuptake inhibitors (SSRIs) and stimulants (Magno-Zito et al., 2003). There is some evidence that SSRIs (Biederman et al., 1998) and stimulants (DelBello et al., 2001) can induce a manic episode in children predisposed to bipolar disorder. It is suggested that some children with genetically transmitted "latent bipolarity" are initially diagnosed with ADHD and/or depression then treated with stimulants and SSRIs, respectively. They respond to these pharmacological "stressors" with a manic episode. In the past, before the era of stimulants and SSRIs, many of these children may have carried their latent vulnerability throughout childhood. With the advent of increased biological and psychosocial stressors in late adolescence and adulthood they may have become symptomatic at a later age.

A final explanation for the substantial recent increase in interest in EOBPSD is related to what has been called the generational or cohort effect. Gershon, Hamovit, Guroff, and Nurnberger (1987) reported rates of bipolar, schizoaffective, and unipolar disorders were higher in cohorts born after 1940 than those in cohorts born earlier. Several biological and environmental factors many explain this effect. One is genetic anticipation, meaning the tendency for certain genetic illnesses to have an increased risk of transmission when two predisposed individuals reproduce (Findling et al., 2003). The mechanism of transmission involves a particular DNA code, a "triple-repeat sequence," which, via reproduction expands, resulting in a greater risk of transmission to offspring. Mendlewicz et al. (1997) have identified such DNA codes (i.e., expanded trinucleotide cytosine–adenine–guanine repeats) in families with BPD. Furthermore, the probability of genetic anticipation may have increased over the last 20 years. Pharmacologic and psychosocial treatment advances for adults with BPD may have improved their interpersonal functioning and reduced their risk of death by suicide, such that more adults with BPD may now live longer and have more children.

Precursors to Psychosocial Treatment

Without reliable and valid methods to assess, diagnose, and classify EOBPSD, differentiate it from alternative diagnoses and medical conditions, accurately ascertain its "true" comorbidity, appraise its impact on family, school, and peer domains, and provide effective pharmacotherapy, evidence-based psychosocial treatments will not be forthcoming. These are all significant and somewhat controversial issues in the field, each of which demands a thorough examination in itself. However, because a detailed appraisal of these issues is beyond the scope of this current paper, we will briefly outline and then refer the reader to more specialized reviews of these topics.

EOBPSD are some of the most difficult childhood disorders to assess, diagnose, and classify. Mackinaw-Koons and Fristad (2002) noted several conditions a clinician must resolve or observe before concluding that behaviors are truly "manic." First and foremost, because of their high level of comorbidity and overlapping symptoms with several other childhood disorders, making a careful differential diagnosis is essential. Clinicians need to distinguish EOBPSD from normal temperamental differences in childhood such as overactivity, poor concentration, recklessness, bragging, and imaginative play (Weckerly, 2002); ADHD, ODD, and CD; learning disabilities and various anxiety, psychotic, Axis II, and pervasive developmental disorders; and from the effects of certain addictive substances, prescribed medications, medical conditions, and poor child-rearing.

Second, the presence of manic symptoms must represent a significant change from baseline, even if that baseline is already disrupted by another condition such as ADHD. In that case, the overlapping manic symptoms of distractibility, psychomotor agitation, and involvement in dangerous activities/pressure to keep talking must increase, along with a change in mood, above and beyond the baseline ADHD symptoms of inattention, hyperactivity, and impulsivity. Third, although manic symptoms in children may not be clearly episodic, there must be evidence of waxing and waning mood symptoms that frequently arise "out of the blue," and are often unrelated to environmental events. Fourth, during the altered (expansive, euphoric, or irritable) mood state, additional manic symptoms (e.g., grandiosity, racing thoughts, decreased need for sleep) must be present. Finally, a thorough family, developmental, medical, social, and school history is required to understand symptom manifestation in the larger context of the child's life. In addition, a mood lifeline, clearly demarcating onset, duration, severity, impairment, and offset of mood symptoms during the child's life, can further distinguish the various EOBPSD subtypes (BP-I, BP-II, cyclothymia, BP-NOS) from one another.

To date, only one set of official assessment guidelines, the AACAP practice parameters (AACAP, 1997), has been published. Although comprehensive and state-of-the-art at the time, in light of the rapid

progress the field has made, these are now dated. As a response to this relative dearth in contemporary assessment guidelines, a group of researchers (including the second author) in conjunction with CABF, the Internet-based support and advocacy organization, recently met in Cincinnati to develop a consensus set of contemporary assessment and treatment guidelines that will be published in the near future (R. Kowatch, personal communication, July 9, 2003).

In terms of classification and diagnosis, *DSM-IV* (American Psychiatric Association [APA], 1994) lists four possible bipolar diagnoses: BP-I, BP-II, cyclothymia, and BP-NOS. Although BP-I, especially in its classic manic–depressive manifestation, can be diagnosed in childhood, it is relatively rare before and even during adolescence (Lewinsohn et al., 1995; NIMH, 2001). In a similar fashion to research on adult BPD (Akiskal, 2003), what has been more recently recognized in youth are the "softer" bipolar spectrum phenotypes of BP-II, cyclothymia, and BP-NOS (Lewinsohn et al., 1995, 2000, 2003; NIMH, 2003). As opposed to adults with "classic" manic depression, who more often present with episodic mania/hypomania characterized by the hallmark symptoms of elevated/elated mood, and grandiosity (Goodwin & Jamison, 1990), children and adolescents, frequently present with shorter episodes or a nonepisodic continuous pattern of mood instability characterized by irritable rages (Geller et al., 2003). Currently, the three main diagnostic and classification issues regarding EOBPSD are whether the diagnosis has to include discrete episodes, the minimum duration of episodes, and whether there are specific hallmark symptoms of mania (Leibenluft, Charney, Towbin, Bhangoo, & Pine, 2003).

In an attempt to resolve the lack of agreement amongst researchers on the nature and definition of EOBPSD, NIMH roundtable members (NIMH, 2001) concluded that children impaired by mood instability could be placed into one of two diagnostic categories: (1) children who meet full *DSM-IV* (APA, 1994) criteria for BP-I or BP-II; and (2) children who are severely impaired by mood instability but do not meet *DSM-IV* criteria for BP-I or BP-II and therefore are typically given the diagnosis BP-NOS. Because of the unclear criteria for BP-NOS, the roundtable considered it a "working diagnosis" to advance future research. However, one limitation of the BP-NOS diagnosis is that it is rather nondescript and may be misused as a general "catch-all" category when in fact it may contain several valid subtypes of EOBPSD.

More recently, Leibenluft et al. (2003) have suggested an innovative resolution by proposing a classification scheme for a range of narrow to broad phenotypes of EOBPSD. This taxonomy, which requires further validation, is based on the duration of episodes (≥7 days, ≥4 days, 1–3 days, continuous), presence of hallmark symptoms of mania (i.e., elevated/expansive mood or grandiosity) or presence of nonhallmark symptoms (i.e., irritability and hyperarousal). Using these three criteria, Leibenluft et al. proposed four phenotypes: a narrow phenotype (≥7 day or ≥4 day episodes with hallmark symptoms), an intermediate hypomania/mania-NOS phenotype (1- to 3-day episodes with hallmark symptoms), an intermediate irritable hypomania/mania phenotype (≥7-day or ≥4-day episodes without hallmark symptoms), and a broad severe mood and behavioral dysregulation phenotype (nonepisodic, but waxing and waning of mood without hallmark symptoms and without episodic decreased need for sleep). As they themselves acknowledge, the latter category will be replete with comorbidity but is required because the available *DSM-IV* diagnoses do not capture the "relatively homogenous population [of children and adolescents] with mood disturbance, hyperarousal and decreased frustration tolerance" (p. 435).

Diagnosis of EOBPSD is made even more complicated by the need to differentiate manic-like overlapping symptoms from other disorders such as ADHD, ODD, CD, schizophrenia, schizoaffective disorder, posttraumatic stress disorder (PTSD), and borderline personality disorder. In addition, the diagnostic process must also take into consideration a variety of prescribed medications, illegal substances, and known medical conditions that may trigger mania. For a more detailed discussion of diagnostic issues, see the AACAP Practice Parameters (AACAP, 1997) and upcoming assessment guidelines (R. Kowatch, personal communication, July 9, 2003).

Children and adolescents diagnosed with EOBPSD typically have a large number of co-occurring conditions, leading Carlson, Loney, Salisbury, Kramer, and Arthur (2000) to state "multiple comorbidity is so common, that prepubertal children meeting symptom criteria for mania without concurrent psychiatric disorders are extremely rare" (p. 176). The most frequently observed co-occurring disorders with EOBPSD are ADHD, ODD, CD, various anxiety disorders, and substance abuse (Biederman et al., 1996; Findling et al., 2001; Geller et al., 2000; Lewinsohn et al., 2003; West et al., 1995; Wozniak et al., 1995).

The evaluation, accurate identification, and effective treatment of comorbid conditions in children and adolescents with EOBPSD are essential, as these conditions may warrant clinical attention in their own right. Additionally, comorbid conditions could act as stressors, triggering future episodes of mania or depression (e.g., an increase in separation anxiety disorder symptoms related to summer camp attendance that impairs a child's ability to fall asleep at night could prompt a manic episode).

In sum, a comprehensive, detailed, and accurate assessment, diagnosis, and classification are indispensable precursors to the psychosocial treatment of EOBPSD, which should then drive the subsequent selection, ordering and implementation of certain treatments strategies and tactics (Rush, 1999). Using Rush's paradigm, Findling et al. (2003) present a variety of strategies and tactics for different case conceptualizations of EOBPSD including the prepubertal, nonpsychotic complex-cycling patient; the patient with ADHD; the euphoric patient, the patient with psychosis; and the patient with depression.

Finally, perhaps one of the most important precursors to effective psychosocial treatment is pharmacotherapy. While numerous double-blind placebo-controlled studies have shown lithium, valproate, and carbamazepine to be effective for the acute treatment and maintenance of BP-I in adults, few have investigated pharmacotherapy for BP-II and BP-depression (Keck & McElroy, 2002). There is an even greater dearth of pharmacological studies for EOBPSD (James & Javaloyes, 2001), with most employing case studies, discontinuation studies, or uncontrolled open-label trials (Geller & Luby, 1997; Ryan, 2003).

Only one double-blind placebo-controlled trial has shown efficacy for lithium in the treatment of adolescents with BP-I or BP-II and secondary substance dependency disorders (Geller et al., 1998). Most children with EOBPSD require multiple medications. Kowatch, Sethuraman, Hume, Kromelis, and Weinberg (2003), in a prospective, open treatment of 7- to 18-year-olds with BP-I or BP-II, found an 80% response rate to two mood stabilizers after not responding to a single mood stabilizer. Similarly, DelBello et al. (2002), using a double-blind placebo-controlled design, reported adolescents with BP-I responded significantly better to valproate and quetiapine than to valproate and a placebo. Finally, Kafantaris, Coletti, Dicker, Padula, and Kane (2001) found adolescents with BP-I with psychotic features responded better to a combination of lithium and an antipsychotic medication than lithium alone.

In addition to the need for more double-blind placebo-controlled trials for all subtypes of EOBPSD, another important and controversial area in pharmacotherapy involves whether the use of antidepressants or stimulants can trigger mania. There have been reports of SSRIs-inducing mania in children and adolescents (Achamallah & Decker, 1991; Biederman et al., 2000; Christensen, 1995; Oldroyd, 1997; Venkataraman, Naylor, & King, 1992), but other studies do not report this relationship (Geller et al., 2002; Geller, Fox, & Clark, 1994; Geller, Fox, & Fletcher, 1993). Similarly, there is some evidence for stimulant-induced mania (DelBello et al., 2001; Koehler-Troy, Strober, & Malenbaum, 1986; Rosse, Johri, & Deutsch, 1997), whereas other studies do not report this relationship (Biederman et al., 1998; Carlson et al., 2000; Carlson & Kelly, 1998; Geller et al., 2002).

Because of various methodological problems with these studies, it is not entirely clear at present whether antidepressants or stimulants induce mania in children and adolescents with EOBPSD. Until this issue is resolved, clinical wisdom warrants the initial use of mood stabilizers followed by the gradual ("start low, go slow") introduction of antidepressants or stimulants in patients who have had previous manic episodes (for a more detailed review of the findings and issues in pharmacotherapy of EOBPSD, see Findling et al., 2003; Ryan, 2003).

Summary

In the last two decades, EOBPSD has gained more widespread acceptance by American academicians, clinicians, and the public at large. Although effective biopsychosocial interventions are sorely needed, many questions remain about the assessment, diagnosis, classification, comorbidity, and pharmacotherapy of EOBPSD. Effective psychosocial treatment will be facilitated by the use of valid and reliable methods of assessment, diagnosis, and classification; accurate differential diagnosis; ascertainment of related comorbid conditions; and the use of effective pharmacological treatments.

ISSUES RELEVANT TO PSYCHOSOCIAL TREATMENT OF EOBPSD

A literature search of PSYCHINFO (pre-1966 and 1966-to-2003) and Medline (1967-to-2003) did not reveal any publications (except our own) for

in-progress or currently developed psychosocial treatments, empirically based or otherwise, for EOBPSD. Given this, on what can we base our treatment decisions? In an attempt to answer this question, we will explore an assortment of topics with direct or indirect implications for treating EOBPSD. These include biological and psychological risk factors; psychosocial treatment of adult BPD, childhood unipolar depression, and child anger-management treatment programs as irritability and "affective storms" are prevalent and highly problematic symptoms of EOBPSD.

Biological and Psychosocial Risk Factors

Biological Factors

The exact cause(s) of EOBPSD is not presently known, but substantial evidence in the adult literature suggests a biological basis. Craddock and Jones' (1999) review of the genetic research identifies a 0.5–1.5% risk of developing BPD in the general population. This risk increases to 5–10% if a first-degree relative has BPD. Additionally, Todd, Geller, Neuman, Fox, and Hickok (1996) found that families of BPD and major depressive disorder (MDD) probands have an increased prevalence of alcoholism in the absence of a mood disorder, suggesting that family history of alcoholism may also play a role in illness onset.

Badner's (2003) review of the monozygotic (MZ) and dyzygotic (DZ) twin studies reveals a BPD concordance rate, when using narrow (BP-I) diagnostic criteria, of 50–67% for MZ twins compared to 17–24% for DZ twins. When using broad diagnostic criteria, these concordance rates increase to 70–87% for MZ and 35–37% for DZ twins. Mendlewicz and Rainer (1977) reported significantly higher rates of affective spectrum disorders (i.e., BPD, MDD, schizoaffective psychosis, and cyclothymia) in biological relatives of adopted probands (18%) compared with adoptive relatives (7%).

Neurochemical studies reveal the possible action of various neurotransmitters (serotonin and dopamine), hormones (cortisol, corticotropin-releasing hormone, thyrotropin-releasing hormone, and somatostatin) and intracellular calcium in the blood system (see a review by Findling et al., 2003). Pharmacological studies, previously reviewed, and studies of relapse related to medication noncompliance (Keck & McElroy, 2002) both suggest the involvement of biological mechanisms. Neuroimaging studies of adults, children, and adolescents with BPD implicate the frontal–striatal–limbic regions of the brain (DelBello & Kowatch, 2003). Finally, as EOBPSD progresses, it may, like adult BPD, become less affected by external environmental stressors and more spontaneously activated by internal biological mechanisms; a process called "stress sensitization" (Kraepelin, 1921; Post, 1992).

Psychosocial Factors

As the previous section illustrates, BPD appears to have significant biological origins. As a result, psychological theories and interventions were considered superfluous and largely were neglected for many years (Frank, Swartz, & Kupfer, 2000). Despite the significant array of psychiatric medications available for the treatment of adult BPD, relapse rates of 40% in 1 year, 60% in 2 years, and 73% in 5 years have been reported (Gitlin, Swendsen, Heller, & Hammen, 1995). Residual symptoms, poor outcomes for bipolar depression, and high medication noncompliance rates in adults with BPD (see a review by Keck & McElroy, 2002) have prompted researchers to examine psychosocial exacerbations of and interventions for BPD.

Cause Versus Course

Whereas EOBPSD appears to have a biological cause, its development over time, or course, may be affected somewhat more by psychosocial factors. For example, Lewinsohn et al. (2003) found that adolescents with EOBPSD did not differ from adolescents with no history of mental illness on sex, age, race, or parental education, but they were less likely to be living with biological parents.

High levels of familial expressed emotion (EE; i.e., critical comments, hostility, and emotional overinvolvement) have been found to predict relapse rates in adults with BPD (Simoneau, Miklowitz, & Saleem, 1998). In children and adolescents with MDD, high familial EE has been associated with a more insidious illness onset (Asarnow, Carlson, & Guthrie, 1987) and a slower recovery course (Asarnow, Goldstein, Tompson, & Guthrie, 1993). While no studies to date have assessed EE in families with EOBPSD, Geller, Bolhofner, et al. (2000) found low maternal-child warmth and high maternal-child and paternal-child tension were significantly greater for an EOBPSD group, compared to an ADHD group and a matched clinical control group without a history of mood disorder or ADHD. Furthermore, children and adolescents with EOBPSD who experienced low

maternal-child warmth were 4.1 times more likely to relapse at a 2-year follow-up compared to those with high maternal warmth (Geller et al., 2003).

The emotional, physical, and financial stress of parenting or being raised with a child with EOBPSD, with or without high EE, also could lead to increased mental health problems for parents and siblings. These problems could, in a transactional manner, act as a further stressor for the child with EOBPSD. Sisson and Fristad (2001) examined a variety of factors related to raising a child with EOBPSD and found caregivers experienced an overall high level of parenting stress. In addition, increased disagreement between parents/caregivers on child-rearing matters has been linked to higher rates of child problem behaviors (Jouriles et al., 1991), lower levels of family problem-solving (Vuchinich, Vuchinich, & Woods, 1993), and overall parental ineffectiveness (Deal, Halverson, & Wampler, 1989). Finally, Smith and Fristad (2003) found parents reported siblings of a child with EOBPSD experienced a high degree of negative stressful experiences.

Negative life events (stress) has been correlated with a threefold increase in time to recover (Johnson & Miller, 1997) and a fourfold increase in relapse rates (Ellicott, Hammen, Gitlin, Brown, & Jamison, 1990) for adults with BPD. When faced with negative life events, adults with BPD who possess a negative cognitive style characterized by dysfunctional attitudes or depressogenic attributions are more likely to develop additional manic and depressive symptoms (Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999). Finally, disruption in social rhythms, particularly the sleep–wake cycle, may increase risk for future manic episodes in adults (Malkoff-Schwartz et al., 1998). Wehr and Sack (1987) proposed that sleep loss in vulnerable adults is a main causal pathway in the manifestation and recurrence of mania.

Summary

Both biological and psychosocial risks are involved in the development and maintenance of EOBPSD. In addition to providing a theoretical foundation on which to design psychosocial interventions for EOBPSD, a knowledge of contemporary findings about cause and course is also invaluable to mental health professionals' conceptualization of individual cases, as well as their formulation of initial and ongoing treatment planning.

Relevant Psychosocial Treatments

Adults With BPD

The literature on psychosocial treatment of BPD is relatively new and focuses not on the disorder, *per se*, but instead is directed toward management of psychosocial risks that may affect the course and severity of impairment caused by bipolar symptoms (Callahan & Bauer, 1999). Otto, Reilly-Harrington, and Sachs (2003) identified three adjunctive psychosocial approaches, including cognitive–behavioral therapy (CBT; Lam et al., 2000), family-focused therapy (FFT; Miklowitz & Goldstein, 1997), and interpersonal therapy with a social rhythm component (IPSRT; Frank et al., 1994). An integrated family and individual therapy (IFIT) also has been recently developed by Miklowitz et al. (2003).

To date, three controlled studies have demonstrated the efficacy of CBT, evidenced by improved medication adherence and fewer hospitalizations (Cochran, 1984); fewer manic, hypomanic, and depressive episodes (Lam et al., 2000); and a reduction in new episodes in addition to an increase in the euthymic interval (Hirshfeld et al., 1998; cited in Otto et al., 2003; Hirshfeld, Gould, Reilly-Harrington, & Sachs, 1997). One randomized clinical trial of FFT and pharmacotherapy has demonstrated reduced manic and depressive symptoms and protection against the recurrence of depression, resulting into longer intervals without relapse and rehospitalization (Miklowitz et al., 2000). Preliminary evidence from a controlled treatment study of IPSRT indicates this adjunctive intervention helps adults with BPD to achieve more stability in their daily routines, lower levels of symptomatology, lower recurrence rates (Frank, 1999; Frank et al., 1997), and attain clinical remission in half the time (21 vs. 40 weeks) compared to adults randomized to intensive clinical management (Hlastala et al., 1997). Finally, an open trial of IFIT, using an historical control sample for comparison, reported longer delays in relapse and greater reductions in depressive symptoms for the treatment group (Miklowitz et al., 2003).

As we are interested in these studies for what they suggest to us about the psychosocial treatment of EOBPSD, an examination of their treatment delivery mechanisms and treatment components follows. CBT trials utilized 6–20 sessions of individual or group outpatient therapy over a course of 2–6 months. Effective treatment components included education about bipolar disorder and its

management, improving medication compliance, cognitive restructuring, problem-solving, management of routines and sleep, assertiveness training, and activity management. FFT treatment was provided via up to 21 sessions of family or marital therapy, conducted in the patient's home. Treatment components included psychoeducation about the disorder, communication-enhancement training, problem-solving skills training, and between-session assignments. IPSRT used an undetermined number of sessions and involved the following treatment components: psychoeducation about bipolar symptoms, prescribed medications, and side effects, and the possible role of disruptions to social and circadian rhythms in triggering episodes; and developing strategies to self-monitor daily mood and activities, manage symptoms, stabilize daily rhythms, resolve a specific interpersonal problem area (grief, role disputes, role transition, or interpersonal deficits), and prevent future social and circadian rhythm disruptions. IFIT consisted of up to 25 biweekly sessions of individual IPSRT therapy and up to 25 biweekly family (or couples) FFT therapy sessions during the alternative weeks. While each of these four interventions has unique characteristics, the overlap in active treatment components is striking. Treatment components can be put into one of two broad categories: information sharing or skill building.

Childhood Unipolar Depression

Most literature on nonadult depression focuses on adolescents. A few studies have examined childhood depressive disorders and several have included mixed-age groups. Despite evidence that depression can occur in early childhood, especially among children of parents with depression (Beardslee, Versage, & Gladstone, 1998), no randomized controlled psychosocial treatment studies of preschool children diagnosed with depression have been published.

Psychosocial treatments developed for childhood unipolar depression include CBT, family therapy, and psychoeducation. Most frequently, these have been adaptations of treatments shown to be successful in adult populations. CBT has been examined in children (Kahn, Kehle, Jenson, & Clark, 1990; Stark, Reynolds, & Kaslow, 1987; Weisz, Thurber, Sweeney, Proffitt, & LeGagnoux, 1997) and in mixed child and adolescent samples (Vostanis, Feehan, Grattan, & Bickerton, 1996; Wood, Herrington, & Moore, 1996). Overall findings suggest CBT leads to significant improvement in self and clinician-reported symptoms of depression, self-esteem, and overall functioning. Stark, Swearer, Kurowski, Sommer, and Bowen (1996) examined a multidimensional treatment program that incorporated CBT skills for children, parent training, family therapy, and teacher consultation. They reported significant decreases in depressive symptoms and depressive cognitions immediately posttreatment and 7 months later. Pilot research on Interpersonal Family Therapy (IFT) with families of depressed children suggests it leads to the reduction of depressive symptoms and maladaptive family interactions (Kaslow & Racusin, 1994). Finally, two other groups also have investigated psychoeducation and found significant reductions in self-reported symptoms of depression, negative automatic thoughts, and internalized coping (Asarnow, Scott, & Mintz, 2002) and increases in parental knowledge of mood problems, positive family interactions, children's perceptions of parental support, and treatment utilization (Fristad et al., 2003).

Again, we will review the mechanisms of treatment delivery and treatment components for the treatment of child depression. For CBT, effective treatment delivery included child-only clinic and school-based interventions, individual sessions, small group (2–5 children) sessions (50–60 min) for 5–12 weeks, structured treatment manuals, role-play group discussion, and homework assignments. Effective treatment components incorporated activity-scheduling logs, instruction in self-monitoring and increasing pleasant activities, changing objective conditions to increase rewards, group problem-solving discussions, relaxation training, positive imagery, cognitive restructuring, skills generalization, problem-solving, watching videotapes of self displaying nondepressed behaviors, education about thoughts, feelings, and behaviors, social skills training, setting realistic standards and goals, and increasing self-reinforcement while decreasing self-punishment.

For family therapy, effective treatment delivery utilized 12–30 weeks of individual, family, and small group sessions, teacher consultations, games, and appropriate homework assignments to make sessions engaging and to encourage generalization. Effective treatment components for the child included education about emotions, activity scheduling, progressive relaxation, anger management, problem-solving, social skills training, cognitive restructuring, and self-monitoring. Parents learned positive-behavior management techniques, including noncoercive methods of discipline, anger-management skills, empathic

listening, positive reinforcement, and pleasant-activity scheduling. Families focused on learning communication, problem-solving, and conflict resolution skills, identifying and modifying dysfunctional patterns of interaction, maladaptive beliefs and negative messages within the family, recognizing the effects of depression on the entire family, understanding the child's symptoms and stressors, developing strategies for affect regulation, and supporting interpersonal and social functioning.

Effective psychoeducational treatment delivery incorporated the use of a single 90-min parent workshop, small parent group and child group sessions that last 50–75 min for 6–9 weeks, structured manuals, role plays, and games. Effective treatment components included educating parents about symptoms, comorbid conditions, risk factors for suicide, myths about mood disorders, medication management, side effects, working with school and treatment providers, effects of mood disorders on family life, parenting techniques, and stress management. Children were educated about symptoms, comorbid conditions, medication management, and side effects. Both parents and children were trained in social skills, anger management, conflict resolution, communication, problem-solving, and generalization of their new skills. As with treatments for adults with BPD, CBT, family therapy, and psychoeducation interventions for childhood depression each have distinct features, yet the similarity of techniques utilized is notable; they involve either information sharing or skill building.

Anger Management Training for Children

In contrast to the classic symptoms of euphoria and expansive mood experienced by many adults with BPD, most children with EOBPSD present with periods of intensely irritable mood (Geller et al., 2003). This irritability is frequently severe and persistent. It can lead to explosive, physically violent, and destructive temper-tantrums, often in response to minor stressors or parental limit-setting (Weckerly, 2002). As irritability is a highly problematic symptom of EOBPSD, the inclusion of anger-management training should be an important component of psychosocial treatment for this population.

Although numerous empirically supported treatments (ESTs) exist for ODD and CD (see a review by Brestan & Eyberg, 1998), only one anger-management treatment program, *The Anger Coping Program* (Lochman, Curry, Dane, & Ellis, 2001), has been identified as probably efficacious (Lonigan, Elbert, & Johnson, 1998) for school-aged children. The *Anger Coping Program* is composed of eighteen 60- to 90-min weekly sessions of cognitive–behavioral group intervention. Treatment targets the social–cognitive distortions and deficits of aggressive children via teaching, modeling, role-playing, and practicing within and between sessions. Treatment components include self-control statements and thoughts, perspective taking skills, the identification of physiological signs of anger, problem-solving skills, and generalization. Controlled outcome studies have demonstrated this program to be effective in reducing disruptive–aggressive off-task behavior; lowering parents' ratings of aggression; increasing children's reported self-esteem; and, at 3-year follow-up, improving problem-solving skills, enhancing self-esteem, and lowering levels of substance abuse (see a review by Lochman, Whidby, & FitzGerald, 2000). Interestingly, continued reduction in disruptive–aggressive off-task behavior and parents' ratings of aggression, at 3-year follow-up, only occurred if children and their parents received a 12-month booster session. Once again, the treatment components employed in Lochman's *Anger Coping Program* are similar to those used in the treatment of adult-onset BPD and childhood unipolar depression. They mainly include elements of information sharing and skill building.

Summary

EOBPSD can be conceptualized as a biopsychosocial disorder as its cause appears to be biological and its course seems to be affected by psychological factors such as low maternal-child warmth, high maternal-child and paternal-child tension, and poor peer relationships, whereas an intact biological family serves as a protective factor. Research on the treatment of adult-onset BPD, childhood unipolar depression, and anger management training in children suggests two family-based treatment components, information sharing and skills development, that may be beneficial in the treatment of EOBPSD.

TREATMENT PROGRAMS FOR EOBPSD

Burns, Hoagwood, and Mrazek (1999) recently published a paper based on their executive summary for the U.S. Surgeon General in preparation for his Report on Mental Health (U.S. Public Health

Service, 2000). In that paper, Burns and colleagues made several recommendations relevant to future research in childhood mood disorders. These include the need to determine treatment effectiveness for mood-disordered youth with comorbid disorders; include families in treatment; develop interventions for younger children (below age 10); include indicators of functional status in addition to symptom severity measures; and use manualized interventions.

Psychoeducation

The Multi Family Psychoeducation Group (MFPG) and Individual Family Psychoeducation (IFP) programs developed by Fristad and colleagues meet these recommendations. Furthermore MFPG and IFP adopt a biopsychosocial theoretical approach to treatment and include many treatment components and delivery systems found to be effective for adult-onset BPD, childhood unipolar disorder, and child anger management training. Both MFPG and IFP are adjunctive treatments developed to supplement existing pharmacological, psychological, and educational interventions. Both are time-limited, with the expectation that ongoing work of a similar nature will be required to maintain treatment gains over time for this chronic illness. It is anticipated that increasing parents' and children's knowledge of EOBPSD and its treatment and expanding their repertoire of individual and family coping skills will lead to more effective and efficient use of existing and future treatments. Furthermore, because MFPG and IFP are manual-based and divided into specific psychoeducational and coping skills sessions, both programs can be used in a flexible, as-needed manner (see Kendall, Chu, Gifford, Hayes, & Nauta, 1998, for a discussion of flexibility and creativity with manual-based treatments).

Overall treatment goals for children and parents are to (1) increase knowledge of mood disorders, symptoms, and common co-occurring disorders; (2) increase understanding of pharmacological, mental health, and community-based (e.g., school) interventions; (3) help families differentiate the child from his/her symptoms; (4) convey to children and parents that they are not to blame for the child's symptoms, but they are responsible for attempting to manage those symptoms; (5) improve manic and depressive symptom management; (6) increase coping and self-preservation skills; (7) enhance individual and family communication and problem-solving skills; (8) improve peer and family relationships; (9) increase concordance between caregiving adults; and (10) increase social support.

MFPG

This group treatment format was developed to include children with bipolar and depressive spectrum illnesses and their parents. MFPG sessions last 90 min and begin with a brief "check-in" meeting of parents and children, during which the previous week's family projects are discussed. Children and parents then go to their respective groups and discuss that particular session's topics. The final 15–20 min of the children's session is spent engaging in recreational activities and noncompetitive group games to foster appropriate social interactions and to develop friendships. Each session ends with the children rejoining the parent group, where they explain their session's topic and discuss that particular week's family projects.

MFPG was designed initially as a 6-week, 75-min group treatment. Results from a randomized controlled trial of 35 7- to 12-year-old children and their 47 participating parents indicate the following posttreatment results: parents' knowledge about childhood mood disorders increased; parent–child relationships, as rated by parents, improved; children's ratings of perceived social support from parents increased (Fristad et al., 2003); and parents' "consumer skills" (i.e., their ability to obtain appropriate services) improved (Goldberg-Arnold, Fristad, & Gavazzi, 1999). A full-scale, randomized study ($N = 165$) of an expanded 8-week 90-min session format is now under way to further establish efficacy of the MFPG program.

IFP

More recently, as not all families or treatment settings can utilize group treatment, MFPG has been translated into "individual family psychoeducation," or IFP. Although advantages of the group format are lost (i.e., increased social support, peer feedback, in vivo peer practice, and discussions with peers who experience similar problems), advantages of "individual family work" are gained. These include ease in scheduling and the opportunity to individualize content to address specific needs of the child or family. Furthermore, during IFP's development many practical and cost-effective issues were taking into consideration for the eventual transportability of this program into private practice and community mental health settings (e.g., using inexpensive,

easy-to-use, parent–child friendly manuals, 50-min sessions). IFP involves sixteen 50-min sessions, alternating between parent-only sessions and, after the requisite "check-in," child-only sessions. The "group games" treatment component utilized in MFPG is not included. However, an additional unit on "Healthy Habits" is included that focuses on improving sleep hygiene (due to the need to minimize circadian rhythm disruption; Rao, 2003), nutritional eating practices (for weight control, as many mood stabilizers, especially antipsychotics, cause significant weight gain; Nasrallah, 2003) and developmentally appropriate exercise routines (for weight control and for their antidepressant effect; Pollock, 2001). A randomized pilot study ($N = 20$) of IFP is being conducted currently by Fristad and colleagues.

Treatment Format

The first half of both MFPG and IFP is more psychoeducational and devoted to providing families with knowledge about manic and depressive symptoms and other disorders including ADHD, ODD, anxiety disorders, and psychotic symptoms. Information about treatment is also provided, including the topics of medication, side effects, and side effect management; mental-health and community-based treatment teams and services, including school and special education services; and how to "build, not burn bridges" with service providers. During these first few sessions, families develop their own treatment goals ("Fix-it Lists") and parents use mood-medication logs to monitor the effects of their child's medication on mood, and complete assignments to chart services and members of their treatment and education teams to identify any gaps in coverage.

Families also learn to differentiate the child from his/her symptoms, using the "Naming the Enemy" exercise (Fristad, Gavazzi, & Soldano, 1999) which is intended to help them establish the symptoms as the "common enemy" of the child and family. This extremely powerful exercise involves the child and family listing, in one column on a single page of paper, the child's symptoms and, in the other column, the characteristics and strengths of the child. After these lists are completed, the paper can be folded in half to show how symptoms sometimes "cover up" the child and lead parents, teachers and peers to see only the symptoms. The paper can then be folded the other way to show how treatment can help "put the symptoms behind the child and allow their true character and strengths to come into view." In subsequent sessions, family members are cued to discuss the child's symptoms as distinct from the child's willful behaviors or personality.

The second half of treatment is dedicated to helping children and parents develop particular coping skills to manage bipolar and comorbid symptoms. One session involves teaching children "feelings management" skills via the identification and awareness of individual triggers (e.g., sibling bugging them), feelings (e.g., anger), physiological signals (e.g., tightened muscles), and behavioral responses (e.g., kicking sibling). This is followed by the abstract or concrete construction of a "Tool-Kit" containing "tools" from four realms: creative (e.g., dance), physical (e.g., jump rope), social (e.g., talk to a friend), and rest and relaxation (e.g., mom gives a back-rub) to help children cope with various problem situations and negative (i.e., "mad, sad, or bad") feelings.

Separate parent and child sessions are used to teach families basic cognitive–behavioral therapy principles, via a "Thinking–Feeling–Doing" exercise, that illustrates how thoughts, feelings, and behaviors about events ("triggers") are related and how they can be modified by generating "helpful" instead of "hurtful" thoughts, feelings, and behaviors (Davidson & Fristad, in press). Problem-solving skills also are taught to parents and children (using the sequence "Stop–Think–Plan–Do–Check") to help them explore solutions to common family problems associated with mood disorders. In addition, therapists teach parents and use role-play and activities to help children identify and understand the effect of "hurtful" and "helpful" verbal and nonverbal communication on family conflicts and relationships.

Additional parent sessions are devoted to discussing and developing strategies to deal with negative family cycles, the stress of parenting a child with a mood disorder and specific issues in managing manic, depressive, and psychotic symptoms. At the end of the final session, families receive feedback on the child's strengths, areas to grow and brief recommendations; are given a list of general resources (e.g., books, websites on mood disorders, mental health, and parenting) and resources specific to their community (e.g., local education services, therapeutic day schools, crisis centers, home-based services, respite, and camps); then "graduate."

All of the previously described treatment components are part of both MFPG and IFP except "Healthy Habits" which, along with IFP, was developed later. Finally, we have developed a collection of

therapeutic tools, behavior management suggestions, clinical caveats, and a list of "predictable problems and promising possibilities" for the mental health professional who works with families with EOBPSD. For a more detailed description of these clinical tools and of the MFPG parent and child sessions, see Fristad and Goldberg-Arnold (2003) and Goldberg-Arnold and Fristad (2003), respectively.

Other Psychosocial Treatments in Development

Although Fristad and colleagues' MFPG study is, to date, the only published empirical study on EOBPSD (Fristad et al., 2003), Pavuluri, Naylor, and Janicak (2002) have developed a child- and family-focused cognitive behavior therapy (CFF-CBT or "Rainbow Program") for 8- to 12-year-olds with bipolar disorder. Treatment is delivered via 12 individual parent and child sessions. Additionally, the child's school receives a work folder of the individual sessions and the school staff has a teleconference with the treatment therapist. Parent and child treatment components include psychoeducation regarding the neuropsychiatric basis of bipolar disorder and the importance of routines; and skill building to improve affect-regulation/anger control, increase positive self-statements, restructure negative thinking/living in the now, promote a balanced lifestyle (parents), increase interpersonal and situational problem-solving, and develop support systems.

Miklowitz's group is developing a manually driven family-focused psychoeducation intervention for 13- to 17-year-olds with BP-I. Based on FFT for adults (Miklowitz & Goldstein, 1997), treatment components include "psychoeducation, family-problem-solving, communication enhancement training to reduce expressed emotion, managing crises" (p. 7) and the rehearsal of coping strategies for future relapses.

Summary

Consistent with existing biopsychosocial theories and research on EOBPSD, as well as therapeutic techniques used in treating adult-BPD, childhood unipolar depression, and anger-management training in children, Fristad and colleagues have developed and are testing two variations of a psychosocial intervention that combines psychoeducation and skill building for families of children with EOBPSD. Similar psychosocial treatments for children and adolescents with EOBPSD are currently being developed and tested by other investigators.

CONCLUSION

Once considered virtually nonexistent as a clinical phenomenon, EOBPSD has recently received a great deal of attention from the scientific and public communities in America. In the last 20 years, we have come to understand that EOBPSD may be expressed in a similar manner to the adult phenotype (BP-I, BP-II, or cyclothymia) or more typically as a mixed, chronic, rapid cycling, phenotype dominated by intense irritable mood swings and high rates of comorbidity. Furthermore, we have begun to comprehend its potential biological origins and psychosocial risks, developmental course, long-term prognosis, and pervasive psychosocial impairments.

Our assessment technologies and diagnostic abilities have evolved and we are building on the foundation of previous research conducted on the treatment of adult-onset BPD, childhood unipolar depression, and anger-management training in children to develop and test pharmacological and psychosocial treatments for EBOPSD.

Considerable progress has been made, but we have a great deal further to go. To move the field forward, future studies should focus on developing effective medication regimens for EOBPSD; creating school-based interventions to increase academic success and improve social–emotional functioning; determining the incremental effects of psychosocial programs in conjunction with pharmacotherapy; evaluating the cost-effectiveness and transportability of psychosocial interventions into community mental health settings; developing and testing psychosocial treatment programs for families with younger children (age 7 and below) who suffer from EOBPSD; creating prevention programs for families with children at risk for EOBPSD; and examining the efficacy of all these programs in families of varying ethnic and socioeconomic backgrounds.

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