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Integrating Pharmacotherapy and Psychotherapy for Paediatric Bipolar Disorder: Translating Science to Service

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ABSTRACT

Objective: For comprehensive management of paediatric bipolar disorder (PBD), it is imperative to combine psychopharmacotherapy with specific psychotherapy. This article proposes a model that incorporates (1) an overview of psychopathology, (2) a review of outcomes in psychopharmacotherapy trials, and (3) a summary of evidence-based forms of psychotherapy to complement pharmacotherapy. **Results**: The psychopathology of PBD is unique compared to that of adult bipolar disorder with prominent irritability, rapid cycling, high rates of co-morbid attention deficit hyperactivity disorder, mixed episodes and chronicity. Combination therapy with a second generation antipsychotic and a mood stabilizer is proving to be more effective than monotherapy with a mood stabilizer. Empirical findings for the support of family-focused, cognitive behavioral therapies with individual family or multifamily psychoeducation groups suggest that these psychosocial treatments are valuable complementary tools for clinicians who treat youths diagnosed with PBD. **Conclusion**: As pharmacotherapy and psychotherapy are most beneficial when applied together, the clinician's understanding of the science behind these forms of treatment is likely to be of great value in effectively providing services to youths diagnosed with PBD.

Key words: Bipolar Disorder, paediatric, ohild, pharmacotherapy, psychotherapy

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Paediatric bipolar disorder (PBD) significantly hinders a child's ability to function emotionally, academically, and socially. The intense mood episodes central to the disorder may be associated with irritability, aggressive and/ or impulsive behavior, and psychosis (Geller et al, 2002; Pavuluri et al, 2004c). These characteristics have the potential to create extreme challenges, both for the child as well as for others who interact with the child at home, at school, and in the community. Despite poor functionality and a high suicide rate (Baldessarini & Jamison, 1999), PBD often remains unrecognized or misdiagnosed. Research groups are currently striving to reach consensus in determining the phenomenology (National Institute of Mental Health Research Round Table, 2001 and 2002), understanding neurobiology, and developing treatment guidelines based on evidence. The current research review attempts to integrate meaningfully the critical findings from pharmacotherapy and psychotherapy studies in PBD. This review is intended to provide a framework for clinicians who work within a model where science is directly translated to service.

Therefore, the aims of this article are to:
1. Describe the psychopathology of PBD
2. Give an overview of psychopharmacotherapy trials in PBD
3. Provide specific psychotherapeutic modalities to complement medication management
4. Provide a comprehensive treatment approach

Phenomenology of PBD

Research groups across the world could assign the same diagnostic label (Paediatric Bipolar Disorder; PBD), governed by the same diagnostic standard (namely, the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision; DSM-IV-TR*), and yet yield dramatically different findings regarding the prevalence and nature of symptom presentation (American Psychiatric Association, 2000). Consensus research conferences have helped to reconcile ideological and methodological differences in the study of PBD to some extent (National Institute of Mental Health, 2001; 2002), but wide conceptual differences still exist (Harrington and Myatt, 2003; Leibenluft et al, 2003). While the lifetime prevalence of bipolar disorder of 1% was reported in an adolescent community study, data are limited by the lack of parent interviews (Lewinsohn, Klein, & Seeley, 1995). Subsyndromal symptoms were present in 5.7% of cases. The involvement of parent interviews, a younger age group, and a comprehensive understanding of developmentally specific manifestations of PBD (Geller et al, 2002; Findling et al, 2001; Wozniak et al, 1995) may yield a different prevalence that is yet to be determined. In clinical populations, the prevalence of BD varies between 0.6% and 15% depending on the instrument utilized to ascertain diagnoses and the type of referrals (Biederman et al, 1995; Lewinsohn et al, 1995; Strober et al, 1995).

Subgroups of bipolar disorder exist to better capture different presentations of the illness. The **bipolar I** diagnosis is considered when a child has experienced at least one manic or mixed episode. Youths diagnosed as **bipolar II** present with at least one episode of major depression, and hypomania. The diagnosis of **cyclothymia** applies to children with alternating hypomanic episodes and subsyndromal depressive episodes. The largest bipolar subgroup, the **bipolar not otherwise specified (BP-NOS)** subgroup (Lewinsohn et al, 2000), consists of children who do not meet full criteria nor have clearly defined episodes. These BP-NOS diagnosed youths suffer, nonetheless, from symptoms of a mood disturbance that result in functional impairment.

Manic Episode:

Juvenile forms of bipolar disorder consist of mixed episodes of mania with depression. As outlined above, depending on the subtype of bipolar disorder, episodes of mixed states or of major depression may or may not occur with a manic episode. Mania in youths is characterized by a persistently elevated, expansive, or irritable mood present with symptoms such as inflated self-esteem or grandiosity, decreased need for sleep, pressured speech, racing thoughts, distractibility, increased goal-directed activity or psychomotor agitation, and risk-taking behavior (American Psychiatric Association, 2000). Irritability may often be the symptom that leads caregivers to seek treatment for a child (Carlson, 1983; Biederman et al, 2003), but its presence is not a clear indicator of mania. In fact, irritability is also associated with other childhood psychiatric illnesses, such as attention deficit/ hyperactivity disorder (ADHD), pervasive developmental disorder (PDD), schizophrenia, oppositional defiant disorder (ODD), and anxiety disorders (Geller et al., 2002). While it is acceptable to consider irritability (if present) as one of the two mood symptoms according to *DSM-IV* criteria (Biederman, 1998), the false positive rate is reduced if the diagnostician ensures the presence of coexisting symptoms of abnormally elevated mood and grandiosity (Geller et al, 2002). Another critical factor in the recognition of this disorder is the identification of pathological expressions of these features. Children can be silly and giddy with an elevated mood that is contextually appropriate such as when in anticipation of or during special events. It is when this extreme happiness crosses to inappropriate situations or persists for an abnormal duration causing functional disturbance that it becomes a concern. Similarly, grandiosity can be difficult to determine in youngsters. Genuine special talents, extended fantasy play due to lack of access to play toys and activities, and/ or insecurity masked with self-aggrandizement may all seem pathologically grandiose, although completely understandable when viewed in context. Therefore, it is critical to assess grandiosity if it is out of context, persistent, and is affecting the child's behavior (i.e. whether or not the child acts on the grandiose belief).

Other symptoms of mania are equally difficult to differentiate. Decreased need for sleep is often a critical associated feature in PBD, versus difficulties getting to sleep or having adequate sleep. Children diagnosed as manic do not experience fatigue despite reduced sleep hours. Given that children generally tend to talk fast, pressured speech should be gauged for a change from their baseline. Racing thoughts experienced by manic youths is often described as the child's "mouth not being able to keep up with (his/her) mind." Overall, with each of the symptoms of a manic episode, the clinical features reflect a change from the child's baseline functioning, occur within the context of a mood episode, and cause marked distress or impairment in the child's social, emotional, and academic functioning.

Depressive Episode:

A child with a major depressive episode presents with a depressed or irritable mood for the majority of the day, nearly every day; or a loss of interest or pleasure in activities that the child previously enjoyed. Additionally, the child experiences other symptoms such as significant weight loss (or failure to meet developmentally expected weight gain) or gain; hypersomnia or insomnia; psychomotor agitation or retardation; fatigue or loss of energy; feelings of worthlessness or excessive or inappropriate guilt; decreased ability to think or concentrate, or indecisiveness; and recurrent thoughts of death, recurrent suicidal ideation without a specific plan, or a single suicide attempt or specific plan for committing suicide (American Psychiatric Association, 2000). Depressed children may feel crabby and whine excessively. Also, they may visibly look unhappy, cry for no apparent reason, rapidly shift moods from irritable to tearful, and complain of somatic symptoms (Pavuluri, Naylor, & Janicak, 2002). Suicidal behavior should be monitored closely in these children, as they are at high risk for suicidal ideation, intent, plans, and attempts (Geller et al, 2002; Lewinsohn et al, 1995).

Rapid Cycling and Duration Criteria:

Rapid cycling as defined by the DSM-IV-TR should be differentiated from the rapid cycling variants of bipolar disorder as characterized by Geller and her colleagues (Geller et al, 1995). The DSM-IV-TR defines rapid cycling as having more than four episodes of either depression or mania or hypomania during a one-year period. In phenomenological studies conducted by Geller and colleagues (Geller et al, 1995), parents often described mood dysregulation as rapid mood swings. Given the consistency of such parental reports, they attempted to integrate this description into the phenomenology of PBD. For example, ultradian cycling is described as cycling within a single day (or more than 365 cycles a year). This cycling could be construed as mood or affect dysregulation. Ultra rapid cycles are those cycles occurring 5-365 times a year. Such episodes (discrete manic, mixed, hypomanic, and/or depressive episodes) clearly exist in some. Symptoms wax and wane with full respite in between episodes. However, it is not uncommon to note subsyndromal symptoms that persist in between episodes. The full extent of how much of the symptoms subside in between recurrent episodes depends on the severity of illness, availability of proper treatment, and the ability of the family to negotiate recovery with optimum treatment. However, PBD can often be non-episodic and chronic (Geller et al, 2004), especially if left untreated.

Differential Diagnosis and Co morbidity:

A current controversy in the study of PBD involves the diagnostic differentiation of similar clinical features, such as distractibility, impulsivity, and irritability shared by other childhood disorders like attention-deficit/ hyperactivity (ADHD), oppositional-defiant (ODD), and conduct disorders (CD). While phenomenological studies continue to explore the often subtle, sometimes drastic, differences in externalizing behaviors characteristic of these disorders, another critical area under investigation is the psychopharmacological and psychosocial treatment of these disorders. As treatment options often differ according to psychiatric diagnosis, the assessment of clinical features should be conducted with caution given the frequent comorbidity. Rates for comorbid disorders range between 11% to 75% for ADHD; 46.4 to 75% for oppositional defiant disorder (ODD); 5.6 to 37% for conduct disorder (CD); 12.5- 56 % for anxiety disorders; and 0 to 40% for substance abuse disorders (Biederman et al, 1997; Geller et al, 1998b, 2002; Findling et al, 2001; Lewinsohn et al, 1995; McClellan et al, 1999; Wozniak, 1995). Children with PBD tend to have higher rates of ADHD than adolescents with BD, while the latter have higher rates of substance abuse (Findling et al, 2001; Lewinsohn et al, 1995; McClellan et al, 1999).

Depending on age and methodological differences of studies, the prevalence of psychotic features in PBD has been shown to range from 16 to 88% (Wozniak et al, 1995; Carlson et al, 2000). The assessment of psychosis in the context of a mood episode is essential in order to distinguish PBD from paediatric schizophrenia, especially in adolescents. Mood congruent delusions and hallucinations are common in PBD compared to those in schizophrenia (Pavuluri et al, 2004a). In addition to the episodic presentation (as opposed to chronic course) of psychotic episodes in PBD, a family history of affective psychosis adds value during the assessment process. A study by Potash and colleagues (Potash et al, 2001) found that psychotic probands were more likely than nonpsychotic probands to have a relative with affective disorder with psychosis.

Suicidal Behavior:
In comparison to other psychiatric disorders in youth, suicidal behaviors occur more frequently with PBD (Baldessarini & Jamison, 1999). Accordingly, the evaluation of suicidal risk factors, ideation, behavior, and attempts should be viewed as an essential component to the assessment protocol for PBD.

Treatment of PBD

Pharmacotherapy Option:

Given the severity of mood dysregulation, aggression, and impulsivity, psychotropic medications almost always play a central role in the treatment of PBD. Optimal treatment of PBD youths involves the collaboration of mental health professionals and family members with a combination of psychopharmacological and psychosocial treatment methods. This dual treatment should be tailored to the individual child and be based on his/her symptom profile at the time of presentation or previous response to medications. A comprehensive pharmacotherapy algorithm was developed by Pavuluri and colleagues that incorporates evidence from clinical trials available to date (Pavuluri et al, 2004a). This algorithm is based on principles that include: (1) prescription hygiene, (2) mood stabilization, (3) overcoming obstacles in mood stabilization by addressing break-through symptoms, and (4) problem solving, (e.g. addressing treatment of comorbid conditions and/or adverse events of medications).

Prior to pharmacotherapy, a baseline evaluation should be conducted. This protocol should include a comprehensive psychiatric evaluation; recent physical examination; complete blood count; comprehensive metabolic profile, including liver and thyroid/d function tests; urine pregnancy test in teenage girls; and a drug screen. If lithium or clonidine is to be utilized in treatment, a baseline ECG is recommended (Pavuluri et al, 2002). Prescription hygiene is employed, i.e., all medications such as antidepressants or stimulants that are worsening the mania are discontinued.

Mood Stabilizers:

Mood stabilizers are the first line medication of choice in the pharmacological treatment of PBD. Data on the immediate and long-term effects of mood stabilizers on youths diagnosed with PBD is less developed than the adult literature.
Lithium is the only mood stabilizer approved by the U.S. Food and Drug Administration (FDA) for prescription to children for acute mania and maintenance. There are two placebo-controlled trials of lithium in PBD (Geller et al, 1998; Kafantaris et al, 2004). In the study examining subjects with a spectrum of mood disorders and substance abuse, Geller et al (1998) found that after 6 weeks of treatment, subjects treated with lithium showed a statistically significant decrease in positive urine toxicology screens and a significant improvement (46% in the lithium treated group vs. 8% in the placebo group) in global assessment of functioning. In this study, the adolescent's diagnosis of BPD preceded their substance abuse by several years. This study demonstrated the efficacy of lithium carbonate for the treatment of bipolar adolescents with comorbid substance use disorders, but did not measure the effect of lithium on mood in these adolescents. Kafantaris examined lithium for adolescent mania in a series of studies and found it effective in combination with second generation antipsychotics (SGAs) (Kafantaris et al, 2001 a, b), but also found that it did not remain as effective in maintenance by itself compared to placebo (Kafantaris et al, 2004).

Another mood stabilizer, **divalproex sodium** (Valproate), has been studied and found efficacious in the treatment of the adult bipolar population (Pope et al, 1991; Bowden et al, 1994). Kowatch and his colleagues (Kowatch et al, 2000) studied outpatient youths who were randomly assigned to 6-8 weeks of open treatment of lithium, divalproex sodium, or **carbamazepine**. The Clinical Global Impression (CGI) Scale and the Young Mania Rating Scale (Y-MRS) were used as the primary measures of efficacy with response defined as a $\geq$ 50% change from baseline to exit in Y-MRS scores. Accordingly, the response rates were: carbamazepine 38%, lithium 38%, and divalproex sodium 53%. Each of these mood stabilizers produced a large effect size ($d > 0.8$) when applied to the child and adolescent monotherapy for bipolar I or bipolar II disorder with a current mixed or manic episode. Side effects in adolescent and teenage females treated with divalproex sodium may consist of menstrual irregularities and/or polycystic ovarian syndrome (PCOS) (O'Donovan, Kusumakar, Graves, & Bird, 2002).

Wagner and colleagues (Wagner et al, 2002) have recently published the results of an open-label study of valproate in 40 children and adolescents aged 7 to 19 years with manic or mixed episodes. There was 55% response to a 50% or greater improvement in YMRS scores. Pavuluri and colleagues (in press) studied mixed mania in an open 6-month trial of divalproex sodium and reported a 73.5% response rate in both manic and depressive symptoms. In this study, it is critical to note that 38% of the subjects also received a stimulant to treat comorbid ADHD.

Although further research with youths is warranted, some studies offer support for **lamotrigine** as an adjunctive treatment for PBD (Kusumakar & Yatham, 1997; Suppes et al, 1999). It may be particularly useful as an antidepressant and may be used, for mixed or depressive episodes where depression is predominant, in combination with a second generation antipsychotic (SGA) or with lithium (Saxena et al, 2004; Pavuluri & Naylor, in press). In comparison adult bipolar disorder, limited support exists for the use of lamotrigine in the treatment of PBD due to the uncommon risk of potentially lethal cutaneous reactions like toxic epidermal necrolysis and Stevens - Johnson syndrome (Kowatch et al, in press). Very gradual titration of lamotrigine over the course of 6-8 weeks is recommended in order to avoid the risk of these reactions.

Frazier and colleagues (Frazier et al, 1999) reported that **olanzapine** monotherapy was effective in 61% of studied cases with significant improvement in manic symptoms. **Combination therapies**, however, yielded consistently better results. Lithium and divalproex (Findling et al, 2003), lithium and another mood stabilizer (Kowatch et al, 2003), divalproex and quetiapine (DelBello et al, 2002), lithium and a SGA (Kafantaris et al, 2001a,b), lithium and risperidone or divalproex and risperidone (Pavuluri et al, 2004d) were some of the combinations that were published with response rates ranging between 70-80%.

PBD presents a multitude of clinical challenges beyond stabilization of mania that must be factored into both the acute and maintenance phases of treatment. If there are prominent symptoms of depression, lithium or lamotrigine (Bowden, 2002; Calabrese et al., 2000) are chosen as primary mood stabilizers either alone or as adjuvant to another partially effective agent; second choice would be a combination of lithium plus lamotrigine; third choice is a small dose of a selective serotonin reuptake inhibitor (SSRI) (in desperate circumstances of severe depression). A long acting medication such as **escitalopram** in lower doses like 2.5 to 5 mg (in a time limited manner and under close supervision) combined with psychoeducation often is effective alongside a mood stabilizer (Wilens et al, 2003). Given the black box warning associated with SSRI use in children, it is important to balance the risks versus benefits of the above treatments.

In moderate to severe presentations of associated aggression, the combination of mood stabilizer and SGA (risperidone, quetiapine, aripiprazole, ziprasidone, olanzapine) may be used after excluding ineffective medications and after adequate trial of chosen medications. Clonidine can be used to subdue rage attacks when things are out of control (Prince et al., 1996; Pliszka et al., 2000). However, children may become disinhibited or become more aroused after persistent use, although this particular observation needs to be further examined.

Strategies for treatment resistance include alternative monotherapy, at least two trials of combination regimes of mood stabilizers plus SGA, and finally, triple therapy to treat comorbid ADHD.

While sleep difficulties are prominent, if left untreated, they can cause severe circadian dysrhythmia and mood dysregulation. While it is standard practice to increase the evening dose of the sedating mood stabilizer, alternatives may be needed. For instance, Melatonin 1-3 mg (Smits et al, 2003), tiagabine 2-4 mg (Gustavson et al, 1997; Mathias et al, 2001) or trazodone 25-50 mg (Saletu-Zyhlarz et al, 2001, 2003; Balon, 1994) can be administered to establish a sleep routine that is critical in the management of PBD.

Treatment of Co morbid Conditions**:**

While ADHD is a distinct disorder separate from PBD, it is not understood if the ADHD-like symptoms in PBD warrant additional treatment beyond mood stabilization. In our study, several subjects continued to show symptoms of inattention post-mood stabilization that warranted stimulant medication (Pavuluri et al, 2004a). Cognitive difficulties such as shifting attention and executive function seen in both ADHD and PBD can be addressed by stimulants. Stimulants are almost always given in a long acting form unless an additional after-school dose is required to sustain the benefits. Among psychostimulants, **long acting methylphenidate** or **mixed amphetamine salts** are equally effective (Sheffer et al, in press). Sheffer and colleagues compared divalproex sodium with placebo and divalproex sodium with mixed amphetamine salts and found an excellent response of 80% with the latter combination. If the stimulant add on strategy is ineffective, **atomoxetine** is factored into the algorithm as an alternative treatment. There are no data establishing the safety or efficacy of atomoxetine in treating youth with comorbid ADHD and PBD. Atomoxetine is a selective norepinephrine reuptake inhibitor with potential antidepressant effects and could theoretically trigger or exacerbate symptoms of mania in patients with PBD. Thus, atomoxetine should be used with great care in youth with PBD. Anxiety disorders, including generalized anxiety disorder and separation anxiety disorder, are relatively common, especially in BD type I.

Psychotherapeutic interventions, such as cognitive behavioral therapy (CBT), remain the first choice of treatment in children and adolescents with comorbid PBD and an anxiety disorder. A small dose of an SSRI such as escitalopram as adjuvant medication may be effective if mania has stabilized, though there are no controlled trials for anxiety comorbid with bipolar disorder. SSRIs are the only medications consistently shown to be effective in controlled trials for childhood anxiety disorders (Birmaher et al, 2003; Black & Uhde, 1994). This treatment intervention requires educating the family about the risk of a switch to mania and close monitoring of the treatment response is necessary. **Guanfacine** goes up in the list of choices if vigilance and autonomic hyperarousal are prominent. **Benzodiazepines** (Graae et al, 1994; Simeon et al, 1992) and **buspirone** follow as alternative choices. Risk for developing dependence needs to be considered for long-term use of benzodiazepines in adolescents. Buspirone may not be effective in all cases. **Propranolol** may be considered in cases of performance anxiety. Medication is often utilized in small doses to reduce risks of exacerbating bipolar disorder and to enable patients to benefit from supplemental psychotherapeutic intervention.

Prescribing information for all of the PBD medications is beyond the scope of this article and is summarized in the "Handbook of Pharmacotherapy, A Life Span Approach" (Pavuluri & Janicak, 2004).

Psychosocial Treatment

As a supplement to pharmacotherapy, psychosocial treatment of PBD is critical to achieve reduction and management of PBD symptoms. Psychosocial concepts like social support, academic functioning, and community influence are important to keep in mind when formulating a treatment plan. Family support is crucial to allow a youth diagnosed as PBD to comprehend, adjust, and work to reduce the impact of features of his or her illness. When a PBD youth is stable on medication, new cognitive and behavioral skills may be introduced through psychosocial therapy so that the child may handle his or her mood episodes and reactions to them. Psychoeducation includes teaching parents and youths about the symptoms, course, treatment, and systems of care associated with PBD. With psychoeducation skill-building strategies that encourage communication, problem solving, emotion regulation, and impulse control are emphasized (Kowatch et al, in press). These psychosocial concepts are addressed through different forms of treatment like cognitive behavior therapy, multifamily psychoeducation groups, family-focused therapy, child and family focused cognitive behavior therapy, and interpersonal psychotherapy. As with pharmacotherapy research, much of the psychosocial therapy research involves the exploration of bipolar adult models with youths diagnosed with PBD.

Fristad and colleagues (Fristad et al, 2002) have applied psychoeducational models used with bipolar adults in their development of a 6-week **multifamily psychoeducation group (MFPG)** intervention. Findings from a study of this intervention suggest that both families of youths diagnosed as PBD and of youths diagnosed with major depressive disorder / dysthymic disorder (MDD-DD) benefit from MFPG with gained knowledge, skills, support, and positive attitudes. In studies with adults, Miklowitz and colleagues (Miklowitz et al 2000; 2003) have demonstrated that **family-focused treatment (FFT)** reduced the number of and extended the time before relapses or recurrences of mood episodes, particularly of depressive episodes. In theory, a decrease in symptomatology is anticipated with FFT as the bipolar patient gains an awareness of how to cope with the illness, experiences decreased levels of expressed emotion from caregiving relatives, and achieves enhanced family problem-solving and communication skills (Pavuluri et al, 2004b). Promising findings have been produced by this research group with a pilot application of FFT to bipolar adolescents. **Child- and family-focused cognitive-behavioral therapy (CFF-CBT),** otherwise called **RAINBOW therapy,** builds upon the foundations of FFT and adds a quality of developmental sensitivity. The CFF-CBT model takes into consideration the affective circuitry of the brain and its putative dysfunction specific to PBD, psychopathology unique to PBD, and social (family and school) stressors linked to PBD. Preliminary study of CFF-CBT suggests that the treatment significantly reduced severity of symptoms and significantly improved overall functioning of PBD youths (Pavuluri et al, 2004b). The key ingredients of RAINBOW therapy are:

1. **Routine**: Establishing a strict routine to allow a stable circadian rhythm and sleep hygiene while cutting down room for negotiation towards distracting agendas.
2. **Affect regulation / anger control:** Establishing techniques to self-monitor mood with mood charts, educating about the disorder and the role of medications in controlling affect.
3. **I for "I can do it**": Helping the child to string together a positive self story while the therapist generates positive self statements to encourage and build self esteem
4. **N for "No negative thoughts**": Restructuring negative thinking by guiding the child to think of how to change unhelpful thoughts or experiences to helpful ones; helping parents to "live in the *now.*"
5. **Be a good friend / balanced life style:** Parents are encouraged to organize play dates and to help children build positive ties while parents supervise them in real life setting. Parents are also advised to focus on obtaining a balanced life style in caring for themselves as they are in this treatment and management process for a long haul.
6. **"Oh, how can 'we' solve it?":** This point is the key for CFF-CBT for PBD where families engage in collaborative problem solving with children after the rages pass through interpersonal and situational problem solving methods **(Options for solutions).** Immediate consequences for undesirable behavior are not recommended in PBD and a reward system can get very intense. Teaching through "pep talks" is the key to influence children.
7. **Ways to ask and get support:** Children are encouraged to draw a support tree and, on each branch, name all the people close to them in their life. The tree is intended to crystallize in the child's mind that there is a supportive safety network or system around them to nurture them when they are in need. They will be coached to reach out for support.

It is critical to validate parents in the process of treatment while building their skills in effectively coping with their child with PBD. The number of empirically validated psychosocial treatment forms available for bipolar youths highlights the usefulness of the psychosocial approach. Again, as apparent through the success of both forms of treatment, the optimal recommended course of care for PBD involves the utilization of both pharmacotherapy and psychotherapy with encouraged involvement of family members.

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