NIH Public Access
Author Manuscript
J Am Acad Child Adolesc Psychiatry. Author manuscript; available in PMC 2016 February 01.

Published in final edited form as:
J Am Acad Child Adolesc Psychiatry. 2015 February ; 54(2): 116–125. doi:10.1016/j.jaac.2014.11.007.

Treatment Moderators of Child-and Family-Focused Cognitive-Behavioral Therapy for Pediatric Bipolar Disorder

Dr. Sally M. Weinstein, PhD, **Dr. David Henry, PhD**, **Ms. Andrea Katz, MA**, **Ms. Amy T. Peters, MA**, and **Dr. Amy E. West, PhD**
University of Illinois at Chicago, Chicago, IL

Abstract

Objective—Prior work has demonstrated the efficacy of child-and family-focused cognitive-behavioral therapy (CFF-CBT) versus enhanced treatment as usual (TAU; unstructured psychotherapy) for pediatric bipolar disorder (PBD). The current study builds on primary findings by examining baseline child, parent, and family characteristics as moderators of symptom response trajectories.

Method—Sixty-nine youth ages 7–13 (M = 9.19, SD = 1.61) with *DSM-IV-TR* bipolar I, II, or not otherwise specified (NOS) were randomly assigned, with family members, to CFF-CBT or TAU. Both treatments consisted of 12 weekly sessions and 6 monthly booster sessions. Participants were assessed at baseline, 4, 8, and 12 weeks, and 6-month follow-up on mania and depression symptoms and overall psychiatric severity. Parents and youth also provided self-report data on baseline characteristics.

Results—CFF-CBT demonstrated greater efficacy for youth depressive symptoms relative to TAU for parents with higher baseline depressive symptoms and lower income, and marginally for families with higher cohesion. In addition, youth with lower baseline depression and youth with higher self-esteem showed a poorer response to TAU versus CFF-CBT on mania symptom outcomes. Age, sex, baseline mania symptoms, comorbidity, and suicidality did not moderate treatment response.

© 2014 American Academy of Child and Adolescent Psychiatry. All rights reserved.

Correspondence to Sally M. Weinstein, PhD, Department of Psychiatry, University of Illinois at Chicago, 1747 W Roosevelt Road, Chicago, IL 60608; sweinstein@psych.uic.edu.
Dr. Henry served as the statistical expert for this research.

Disclosure: Dr. Weinstein has received honoraria from the University of Lund (academic talk) and American Foundation for Suicide Prevention (community talk). Dr. Henry has received funding from the National Institute of Justice, National Institute of Drug Abuse, NIMH, Centers for Disease Control and Prevention, and the McCormick Foundation. He has received honoraria and consultancies from Rush University, the Center for Alaska Native Health Research, and the University of Virginia Curry School of Education. He is also affiliated with the University of Alaska, Center for Alaska Native Health Research, where he holds a President's Professor appointment. Dr. West has received honoraria from the National Institutes of Health (grant review), Northwestern University (Grand Rounds), and University of Lund (academic talk). Ms. Katz and Ms. Peters report no biomedical financial interests or potential conflicts of interest.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Conclusion—Results indicate that CFF-CBT was relatively immune to the presence of treatment moderators. Findings suggest the need for specialized treatment to address symptoms of PBD in the context of parental symptomatology and financial stress.

Keywords

Pediatric bipolar disorder; cognitive-behavioral therapy; family-focused intervention; treatment moderators; randomized clinical trial

Introduction

Pediatric bipolar disorder (PBD) is a chronic and debilitating illness characterized by periods of episodic mood disturbance and pronounced impairments in social, academic, and family functioning.[1, 2] Given the significant psychosocial dysfunction and poor long-term prognosis associated with PBD, psychotherapy is considered an essential component of the treatment approach[3]. Although research is limited, randomized controlled trials have established the efficacy of family-focused individual and group treatments for youth with bipolar disorder (BD).[4–6] Yet beyond simply examining efficacy, the identification of patient and family characteristics that may influence or moderate treatment outcomes is critical for improving interventions for this vulnerable population. Indeed, the examination of treatment moderators has been prioritized by the National Institute of Mental Health (NIMH) to advance knowledge about optimal personalized treatment – i.e., what works, for whom, and under what conditions.[7] Personalized treatment approaches are particularly relevant in PBD; the complexity of PBD symptoms and variable response to even the best evidence-based treatments suggests the presence of pretreatment factors that may influence outcomes.

The child treatment literature points to several demographic, child, and parent characteristics related to differential response to psychotherapy for anxiety, depressive, behavior, and eating disorders, including child symptom severity and comorbidity [8–11], and parent marital adjustment and psychopathology.[8, 9] Numerous studies of youth depression highlight symptom severity[12–14], psychosocial impairment[14–16], comorbid disorders[15, 16], parental depression [12], and greater family difficulty (e.g., conflict, low cohesion)[13, 16] as predictors of poor psychosocial treatment prognosis overall. Specific to PBD, findings suggest that the effects of evidence-based treatments may in fact be enhanced among youth and families with greater baseline impairment. Families characterized as high in expressed emotion (EE; i.e., over-involvement and criticism) showed greater symptom improvement in response to family-focused treatment for adolescents (FFT-A) as compared to a brief educational control, whereas families with low EE responded equally to the treatment conditions.[17] Similarly, the effects of a group psychoeducational intervention for children with bipolar or depression spectrum disorders (Multi-Family Psychoeducational Psychotherapy, MF-PEP) versus waitlist control participants were greatest among youth with severe baseline functional impairment as compared to youth with mild impairment.[18] Thus, the effects of specialized treatment for PBD may be optimized among the higher-risk youth and families that these treatments are designed to target.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

In this study, we build on findings supporting the efficacy of child-and family-focused cognitive-behavioral therapy (CFF-CBT) for PBD[6] by exploring the factors associated with achieving optimal treatment effects. CFF-CBT is an adjunctive treatment that was developed to address the unique needs of the preadolescent PBD population and their families. CFF-CBT comprises three innovative aspects in the treatment of PBD: it is designed to be developmentally specific to symptoms of PBD experienced by school-aged children (e.g., rapid cycling, comorbid disorders, mixed mood states) and related psychosocial impairment (e.g., low self-esteem, interpersonal difficulties); it involves intensive individual work with parents in order to address their own therapeutic needs and impact on parenting (e.g., parental wellbeing, family stress),[19, 20] and it integrates psychoeducation and cognitive-behavioral therapy with complementary techniques from mindfulness-based and positive psychology interventions to target the range of needs of families affected by PBD. Grounded in the evidence on affective circuitry and psychosocial impairment associated with PBD, the core components of CFF-CBT aim to improve child affect dysregulation and self-esteem, parent wellbeing, and family coping with BD.

A recent trial examined CFF-CBT as compared to a dose-matched, enhanced TAU control, and findings demonstrated the efficacy of CFF-CBT in terms of symptom and global functioning outcomes.[6] The present study extends primary findings to examine whether baseline child, parent, and family variables moderated response to CFF-CBT versus TAU. We investigated moderators within the key categories identified by expert consensus, including demographics, illness severity and comorbidity, parental psychopathology, and psychosocial variables.[21] Within these categories, we focused on the treatment predictors/moderators that have emerged in the extant literature that most closely corresponded to the theoretical model and key treatment foci of CFF-CBT: indicators of child severity (symptoms, comorbid anxiety or disruptive behavior disorders, suicidality) and psychosocial functioning (self-esteem), parent wellbeing (operationalized as depressive symptomatology), and family functioning (operationalized as family cohesion). The current study expands prior research exploring moderators of an empirically-supported group intervention for children with bipolar and depressive disorders[18] and family-focused treatment for adolescent BD[17] by examining moderators of symptom trajectories in response to an individual family treatment for preadolescent youth with PBD. A better understanding of how baseline characteristics in the heterogeneous PBD population relate to symptom outcomes will improve treatment decision-making and approaches to enhance treatment response.

Guided by prior PBD research, we expected that youth with greater illness severity, lower self-esteem, higher parental depression, and lower family functioning at baseline would show greater reduction in symptom trajectories in response to CFF-CBT relative to TAU, given the explicit focus on these treatment targets in CFF-CBT. In addition, analyses examined potential demographic moderators (age, sex, and family income); these analyses were considered exploratory given mixed findings in past clinical trials for PBD and depression.[12,15,16,18]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Method

Participants

Participants were children (n = 69) diagnosed with a bipolar spectrum disorder recruited from a specialty mood disorders clinic in an academic medical center in a large Midwestern urban area from 2010–2014 (for details and consolidated standards of reporting [CONSORT] diagram, see [6]). Children meeting *DSM-IV-TR* criteria for bipolar spectrum disorders (BP-I, BP-II, and BP not otherwise specified [NOS]) aged 7–13 were eligible to participate. BP-NOS was defined using *DSM-IV-TR* criteria as the presence of depression and mania symptoms that met symptom severity threshold but not minimal duration criteria, or the presence of recurrent hypomanic episodes without intercurrent depressive symptoms. Inclusion criteria included: patients stabilized on medication (defined as Young Mania Rating Scale (YMRS[22]) ≤ 20 and Children's Depression Rating Scale-Revised (CDRS-R[23]) scores of < 80, indicating no severe symptoms requiring immediate, more intensive care), parental consent, and youth assent. These criteria were intended to exclude children who required acute stabilization in more intensive treatment before being able to participate in psychotherapy, but still include children who were actively symptomatic. Exclusion criteria included: youth IQ < 70 (KBIT-2[24]), active psychosis, active substance abuse, neurological/medical problems that complicate symptoms (Washington University in St. Louis Kiddle Schedule for Affective Disorders and Schizophrenia [WASH-U-KSADS][25]); active suicidality requiring hospitalization (Columbia Suicide Severity Rating Scale, C-SSRS[26]); and primary caregiver severe depression or mania.

Procedures

Diagnosis and Randomization—All study procedures were approved by the Institutional Review Board at the University of Illinois at Chicago. Eligibility was assessed by trained raters (licensed clinical psychologists and doctoral students). After the informed consent procedure and screening, parents were interviewed using the WASH-U-KSADS,[25] with portions of the Kiddie-SADS-Present and Lifetime Version (K-SADS-PL[25, 27]) used to define mood episodes with corroborating information from child report. Diagnostic interviews were reviewed during study meetings for final determination. Youth meeting diagnostic criteria for a bipolar spectrum disorder completed the baseline assessment and were randomized to study condition using Research Randomizer software.[28] Outcome assessments were conducted by a blinded rater at 4, 6, 12 (posttreatment) and 39 weeks (6-month follow-up).

Psychosocial Intervention—Participants randomized to CFF-CBT (n=34) were assigned a study therapist in the Pediatric Mood Disorders Clinic (PMDC) and received 12 60–90 minute weekly sessions in the core treatment phase and up to 6 monthly follow-up sessions in the maintenance phase over the course of 9 months. Study therapists were clinical psychology pre-and postdoctoral trainees (n= 23) who received a three-hour initial training on CFF-CBT and weekly expert supervision. Sessions alternated between parent, child, and family, and included seven components that comprise the treatment acronym "RAINBOW": **R**outine (developing consistent daily routines), **A**ffect Regulation (psychoeducation about feelings; mood monitoring; coping strategies to improve mood

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

regulation), **I** Can Do It! (improving child self-esteem and parent self-efficacy), **N**o Negative Thoughts/Live in the **N**ow (cognitive restructuring and mindfulness techniques to reduce negative thoughts), **B**e a Good Friend/**B**alanced Lifestyle (social skill-building and improving parent self-care), **O**h How Do We Solve this Problem? (family problem-solving and communication training), and **W**ays to Find Support (enhancing support networks; see [5]). Participants randomized to TAU (n=35) were assigned a therapist in the General Psychiatry Clinic (pre-and postdoctoral psychology trainees, psychiatry fellows, and social work interns), who received a one-hour training session on PBD. Sessions were matched for dosage but were otherwise unstructured. All sessions were audio-recorded to assess treatment content. Study therapists demonstrated strong fidelity to the CFF-CBT manual (93% of content delivered), and there was minimal overlap with TAU (4% of CFF-CBT contents were delivered in TAU sessions).[6] All participants received medication management in PMDC following an evidence-based algorithm.[29] Medication was not manipulated for the study, but any changes to the medication regimen were tracked at each assessment. Medication changes during the course of the study did not differ between CFF-CBT (23%, n=15 reported a medication change during the course of the study) and TAU (22%, n=14), $\chi^2 = .229$, n=64, p=.63.[6]

Measures

Outcome Measures

Outcome measures included multi-informant assessment of the range of PBD symptoms and overall impairment.

The Child Mania Rating Scale (CMRS[30]) is a 21-item parent-rated measure that assesses *DSM-IV-TR* mania symptoms. Items are rated on a Likert scale ranging from 0 (never) to 3 (very often) and summed to yield a total score; scores at/above 20 are considered clinically significant. The CMRS was selected because of its ability to capture symptom changes over time by reporters (parents) that have more comprehensive access to the child's behavior throughout the course of treatment and across different contexts. Research suggests that parent-report may result in more accurate assessment of mania[31], and the CMRS demonstrates strong psychometric properties, concurrent validity with the YMRS, and sensitivity to symptom change across treatment [30, 32].Reliability was high in this sample: Cronbach's alpha=0.90.

The Children's Depression Rating Scale-Revised (CDRS-R[23]) is a 17-item clinician-rated instrument for depression severity in children aged 6–17. The CDRS-R was administered to the youth, with collateral information collected separately from parents. The CDRS-R has shown strong validity, inter-rater and test-retest reliability,[23] and high internal consistency in this sample (alpha= 0.81).

Clinical Global Impressions Scales for Bipolar Disorder (CGI-BP[33]) is a clinician report of the overall severity of a child's psychiatric illness, modified for BD. Scores are calculated by summing across five subscales (mania, depression, attention-deficit/hyperactivity, psychosis, and sleep difficulties); inter-rater reliability for youth not experiencing an acute mood episode is 0.75.[33]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Baseline Characteristics

Parent/Family Characteristics: Parent depression was assessed at baseline via the Beck Depression Inventory-II (BDI-II[34]), a 21-item self-report measure of current depressive symptoms with demonstrated validity and one-week test-retest reliability.[34] Parents reported minimal baseline depression symptoms (M=10.41, SD=10.12; scores < 13 are subclinical), with 73% of parents reporting subclinical symptoms and 13% reporting moderate or higher symptoms (score > 20); current sample alpha=0.93. Respondents were primarily mothers (90%, n=62). *Family cohesion* was assessed via parent-report using the Family Adaptability and Cohesion Evaluation Scale (FACES), Cohesion subscale.[35] The FACES measures family relationships and attitudes; the Cohesion subscale includes 7 items about family involvement, closeness, and support, with responses ranging from 1 (Strongly Disagree) to 5 (Strongly Agree). This scale had adequate consistency within the sample (alpha=0.79). Lastly, annual family income was assessed via the Conners-March Developmental Questionnaire (CMDQ[36]). Responses on the ordinal scale were dichotomized as below/above $50,000.

Child Characteristics: Child PBD symptom severity at baseline was assessed using the CMRS and CDRS-R (see above). Comorbid diagnoses of anxiety disorder (separation, panic, specific/social phobia, obsessive-compulsive, generalized, or posttraumatic stress disorder) and disruptive behavior disorder (DBD; conduct, oppositional defiant, and attention-deficit/hyperactivity disorder) were made via WASH-U-KSADS[25] (dichotomized as present/absent). Suicidal ideation was assessed via the Columbia Suicide Severity Rating Scale (C-SSRS[26]), a semi-structured interview for ages six to elderly that assesses types of suicidal ideation and behaviors. The C-SSRS has shown good sensitivity and specificity for suicidal behavior across multiple studies.[26] A binary item was used to indicate presence of any current (past month) suicidal ideation. Child self-esteem was measured by child report (with assistance from study interviewer) on the Piers-Harris Self-Concept Scale (PHSCS-2[37]). The PHSCS-2 is a 60-item scale with good reliability and validity that assesses attitudes about physical appearance, intellectual and school status, behavior, satisfaction with self, and popularity. Items are rated as yes/no and yield a total score; reliability in this sample was alpha=0.90. Last, child age and sex were assessed via the CMDQ.[36]

Analytic Approach

Mixed-effects regression (growth curve) models (MRMs[38]) were conducted via SPSS MIXED to examine treatment moderators on key PBD symptom outcomes. Mixed effects regression models are well-suited for the analysis of longitudinal data, as they are robust to the data dependency that occurs with the repeated assessments of individuals over time. Additionally, MRMs are efficient in handling missing data by using all available data for a given participant to estimate group trends at each time point. Separate MRMs were evaluated for each outcome measure and included effects for Baseline Characteristic, Treatment (CFF-CBT [coded as 0], TAU), Wave (Baseline, 4, 8, 12, 39 weeks), and the Baseline Characteristic × Treatment × Wave interaction to examine moderation; models also included all associated lower-order interactions. Models included linear and quadratic effects for Wave; the model was re-fitted without the quadratic term if non-significant.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Interactions with continuous variables included mean-centered terms to reduce potential multicollinearity. Models were evaluated for the intent-to-treat sample and included all available data for randomized participants. Participants who dropped out of the study were contacted for follow-up assessments and included if available. For participants with outstanding assessment sessions at study completion (n=8), all available data was included.

Results

Descriptive Statistics

The intent-to-treat sample included 69 participants. Mean age was 9.19 years (SD=1.61; range 7–13), and n=29 (42.0%) were female. Furthermore, n=36 (52%) were European Caucasian, n=21 (30%) African American, n=7 (10%) Hispanic, n=3 (4%) American Indian or Alaskan Native, n=1 (1%) Native American or Pacific Islander; and n=3 (4%) identified as "Other." The majority of youth met criteria for BP-NOS (n=43, 62%), with n=22 (31%) diagnosed as BP-I and n=4 (6%) diagnosed as BP-II. Significantly more CFF-CBT participants completed the core treatment (n=30) than TAU (n=17), χ^2=13.46, p<.001; however, attrition by the 6-month assessment did not differ by condition (n=10 in CFF-CBT, n=19 in TAU; χ^2=2.51, ns). For study CONSORT diagram, see [6].

Table 1 displays baseline descriptive statistics for all measures, stratified by condition. A series of t-tests and chi-square analyses also examined whether outcome and moderator variables differed by treatment condition. Findings indicated equivalence across conditions with the exception of child mania symptoms; youth in CFF-CBT demonstrated lower mania symptoms versus TAU. In addition, analyses examined correlations among potential moderators. Child depression symptoms and self-esteem were inversely correlated (r=−.59, p<.001); no other correlations were significant, signifying distinct constructs.

Parent/Family Moderators

Separate MRMs examined the moderating effects of baseline parent/family characteristics (parent depression symptoms, family cohesion, and family income) on child mania symptoms, depression symptoms, and overall psychiatric severity. Results of the moderator effects from all MRMs are presented in Table 2, and effect sizes are reported using Cohen's *d*.[39] Consistent with hypotheses, results revealed a three-way interaction between baseline parent depression symptoms, treatment, and wave on child depression symptoms, indicating that parent wellbeing differentially influenced response to CFF-CBT versus TAU; this effect was medium-sized. To illustrate the effect for interpretation purposes, mean splits dichotomized high versus low parent depression scores; estimated mean CDRS scores over time for high/low parent depression scores, stratified by condition, are plotted in Figure 1. For parents with higher baseline symptoms, CFF-CBT seemed to be more effective in improving youth depression symptoms versus TAU. Youth responded similarly to CFF-CBT regardless of parent symptomatology, whereas striking differences were observed in TAU: youth of parents with higher symptoms showed a poorer treatment response versus parents with low symptomatology. Baseline parent depression also moderated child psychiatric severity outcomes, although effects were marginal (p=.08); patterns were identical to those

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

observed for child depression outcomes. For mania outcomes, however, parent depression did not predict or moderate treatment response.

Models examining family cohesion revealed that youth from families with higher cohesion showed a marginally stronger response to CFF-CBT versus low cohesion families in terms of overall psychiatric severity (p=.05) and child depression (p=.08) outcomes, relative to TAU; effects were medium-sized. Moderation effects on mania symptoms were not significant. Last, family income significantly moderated treatment effects on child depression, with a medium-sized effect. As Figure 2 demonstrates, the interaction was driven by the stronger treatment response for lower-income families to CFF-CBT versus TAU. Trajectories in CFF-CBT did not vary by income, but differences were observed in TAU. No two-or three-way interactions were significant for mania symptoms or overall severity outcomes. Post hoc analyses explored whether family income findings were driven by differences in session attendance or child/family characteristics (i.e., illness severity, self-esteem, parent depression, family cohesion) by income status. Parental depression was marginally greater among low versus high income families (p=.074); no other variables differed by income status.

Child Moderators

Models examined the moderating effects of child depression severity (on CMRS and CGI-BP outcomes only) and mania severity (on CDRS-R and CGI-BP), as well as self-esteem, current suicidality, and comorbid anxiety or DBD on all outcomes (Table 2). Baseline child depression significantly moderated mania response trajectories, with a medium-sized effect. As shown in Figure 3, youth with lower depression (dichotomized via mean split as above) demonstrated a poorer response to TAU versus higher depression youth. Effects on overall severity were not significant. An identical pattern of effects was observed for child self-esteem on mania symptom trajectories: youth with higher self-esteem showed a poorer response to TAU versus youth with lower self-esteem, whereas trajectories in CFF-CBT were similar across youth. Moderation effects of self-esteem on depression and overall psychiatric severity were not significant. In contrast, baseline mania severity did not predict or moderate treatment response for depression symptoms or overall psychiatric severity. Additionally, current suicidality did not predict or moderate treatment response for any outcome, nor did comorbid anxiety. Given the high prevalence of comorbid DBD in this sample (87% met criteria), moderation analyses could not be conducted due to limited variability. Last, exploratory analyses examined age and sex as treatment moderators, but neither was a significant predictor or moderator of mania, depression, or overall psychiatric severity outcomes. Sex × Treatment × Wave on overall severity was marginally significant (p=.08); examination of slopes indicated a slight advantage for girls in CFF-CBT versus TAU, but girls and boys responded similarly within each condition.

Discussion

The search for moderators is essential to inform the development of personalized treatment approaches and improve long-term outcomes in difficult-to-treat populations.[7] We identified several key child and parent/family moderators of treatment response among pre-adolescents

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

with BD, a complex and vulnerable clinical population, with consistently strong effect sizes. In line with expectations, parent functioning significantly moderated treatment effects of CFF-CBT on child symptom trajectories as compared to an enhanced treatment-as-usual. Children of parents with higher depressive symptoms showed greater response to CFF-CBT versus TAU in terms of their own depression symptoms, and marginally, overall psychiatric severity. Of note, this was not a clinical sample of parents; the majority of parents had subthreshold levels of depressive symptoms. Findings thus suggest that parent functioning deficits, even at a subclinical level, interfered with treatment in TAU. However, children with less symptomatic parents responded similarly to the treatments. Our findings stand in contrast to the youth depression literature, where the superiority of CBT versus control on youth depression outcomes was eliminated in the presence of maternal depressive symptoms.[12] Yet results converge with the broader themes in the PBD literature, suggesting that parents and youth with greater impairment pretreatment respond better to specialized interventions for BD versus usual care.[17, 18]

Although analyses did not examine mechanisms, findings suggest that directly addressing parental wellbeing, which is integrated throughout CFF-CBT, may be a necessary element of effective treatment for PBD in the presence of parent symptomatology. Our treatment is unique in the focus on parent functioning, recognizing the primary role parents play in coping with PBD among children. Specifically, one-third of sessions target parent wellbeing, with therapeutic content including: the acceptance of parents' difficult feelings related to their child's diagnosis ("A"); enhancing self-efficacy ("I"); improving parents' negative thinking via cognitive-and mindfulness-based strategies ("N"); increasing parental self-care and life balance ("B"); and accessing positive supports ("W"). These themes were minimally addressed in TAU, as only 4% of CFF-CBT ingredients were delivered in TAU. Without addressing parents' needs, they are unlikely to be effective treatment consumers when experiencing their own depressive symptoms. Moreover, findings suggest that even subclinical symptoms in parents – which may go undetected by clinicians-are relevant for treatment outcomes; this is consistent with research documenting that subthreshold depressive symptoms are associated with significant impairment [40–42]. Results underscore the importance of assessing and addressing these subclinical symptoms in treatment for PBD to optimize youth outcomes. Although not tested directly in this study, the focus on parents in CFF-CBT may result in a myriad of changes that contribute to youth improvement, including improved self-efficacy and coping, consistency of parenting strategies, and parent-child interactions. It will be important for research to explore these factors to elucidate mechanisms of intervention efficacy in PBD.

Similarly, we saw that children from low-income families showed greater response to CFF-CBT relative to TAU. Prior research has demonstrated mixed support for income as a treatment moderator for youth mood disorders. Higher-income families responded better to CBT for youth depression versus control,[15] whereas income did not influence response to group treatment versus waitlist control for PBD.[18] Current findings may be explained, in part, by the marginally greater depressive symptoms among parents of lower income status. Although analyses did not examine which processes may mediate the superior response to CFF-CBT versus TAU among lower income families, it is possible that the structured curriculum and focus on psychoeducation, parent management strategies, and parental

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

support in CFF-CBT may be better equipped to manage PBD symptoms in the context of financial difficulty and related parental distress, versus usual care. Future research is needed to fully understand the influences of socioeconomic factors on treatment for PBD, but current results suggest that families respond equally to CFF-CBT regardless of income level; this has important implications for dissemination into community settings targeting financially underserved populations.

In contrast to predictions, families with higher cohesion responded marginally better to CFF-CBT than low-cohesion families, relative to TAU, and showed the strongest treatment response across conditions. However, effects only approached significance and thus must be interpreted cautiously. Although the direction was unexpected, findings are not surprising, as it may be more difficult for families with vulnerabilities in cohesion to implement the family-focused activities in CFF-CBT (e.g., problem solving, collaborative coping plans). Similarly, past research has shown that depressed adolescents from families with disengagement and communication difficulties were more likely to benefit from fluoxetine alone, whereas youth with better family functioning showed greater benefit from combined fluoxetine and CBT,[43] suggesting that a certain threshold of family functioning may relate to effective family therapy participation. Thus, an enhanced focus on family support and cohesion at treatment outset may improve outcomes. While families with increased cohesion showed marginally greater improvement than families with lower cohesion in CFF-CBT, it is important to note that overall, all families benefited from CFF-CBT compared to TAU. The marginal differences in treatment trajectories suggest that even at lower levels of cohesion, families were amenable to treatment and that CFF-CBT engages important family-level skills that, while harder for some, are imperative for treatment success.

Child depression and self-esteem also moderated treatment response but in intriguing directions. Youth with lower depression, and those with higher self-esteem, demonstrated poorer mania outcomes in TAU relative to CFF-CBT. Youth responded equally to CFF-CBT regardless of impairment, whereas dramatic differences were observed within TAU. Our findings stand in contrast to past research indicating that greater baseline impairment was a positive moderator of CBT or specialized treatment in randomized trials for youth mood disorders.[16, 18, 44] Of note, child functioning measures were obtained via youth self-report and responses to a semi-structured interview and may minimize pretreatment distress levels. Nonetheless, findings suggest that youth reporting less acute symptoms may in turn receive a less powerful intervention in an unstructured and non-manualized treatment; in contrast, all youth receive similar interventions focused on affect regulation, self-esteem, and interpersonal functioning in CFF-CBT given the manualized approach. As such, the feedback loop inherent in clinical practice—tailoring the intervention based on assessment of current functioning and needs—may not operate to the youth's benefit in treatment for PBD. Rather, a standardized approach to the amelioration of PBD symptoms may optimize treatment outcomes. Findings also underscore the importance of multi-informant assessment to inform treatment planning.

Last, several factors did not influence treatment outcomes, including other indicators of youth severity (child mania symptoms, comorbid anxiety, suicidality), age, and sex. Numerous studies have shown that suicidality predicts poorer outcomes in treatment for

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

adolescent depression.[15, 16, 45] However, our younger sample may allow for intervention at an earlier point in the continuum of suicidality severity, thus resulting in more favorable treatment outcomes. Findings for comorbid anxiety, as well as the inability to examine moderation of comorbid DBD due to limited variability, parallel recent PBD research[46, 47]. The lack of effects for age and sex are consistent with past treatment studies with children,[8, 18] suggesting that age and sex within younger populations do not affect treatment outcomes, although limited variability in age range may influence findings.

Of interest were findings that parent factors moderated treatment outcomes for depressive symptoms, whereas child factors differentially influenced mania trajectories. Such patterns may speak to the complexity and heterogeneity in clinical presentation in PBD[48] and multiple levels of influence on symptoms. Measurement differences (i.e., parent versus clinician report of outcomes) may also account, in part, for findings. It is important to note that our analyses of primary outcomes revealed a lack of treatment main effects on the CDRS[6]. Results suggest the presence of moderators that influenced depression outcomes but were masked when examining main effects. Findings underscore the need to examine moderators when considering intervention efficacy.

Results must be viewed in the context of study limitations. The sample size and attrition by follow-up may have limited power to detect small effects, although the detection of multiple interactions suggests that any absence of hypothesized interaction effects was not due to insufficient power. In addition, differential drop-outs in the acute phase of TAU versus CFF-CBT may have influenced treatment outcomes. A third limitation concerns the lack of objective assessment of mania; consistency in outcome measurement for mania and depression symptoms would have strengthened findings. Fourth, our sample primarily included subthreshold parental depressive symptoms, so findings may not generalize to severe parental depression. Fifth, as an outpatient treatment study, findings may not generalize to more severe clinical populations (e.g., inpatient or intensive outpatient settings). Last, analyses did not examine causal pathways nor did they include longitudinal measurement of parent symptoms. The examination of treatment mediators must be explored in future research to identify how moderation and mediation processes operate in concert.

Despite limitations, these findings have powerful implications for treatment decision-making and methods to enhance treatment response in PBD. Results indicate that CFF-CBT is efficacious even in the presence of potentially complicating child and parent factors, where standard psychotherapy may fall short. Results highlight the importance of specialized manual-based treatment for PBD to address the constellation of difficulties at the child and parent level that accompanies this disorder.

Acknowledgments

This research was supported by the National Institutes of Mental Health (NIMH) K23 grant MH079935 (A.E.W.) and Young Investigator Award YIG-1-140-11 from the American Foundation for Suicide Prevention (S.M.W.).

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

References

1. Goldstein TR, Birmaher B, Axelson D, et al. Psychosocial functioning among bipolar youth. Journal of affective disorders. 2009; 114(1–3):174–183. [PubMed: 18715651]
2. West AE, Pavuluri MN. Psychosocial treatments for childhood and adolescent bipolar disorder. Child and adolescent psychiatric clinics of North America. 2009; 18(2):471–482. x–xi. [PubMed: 19264274]
3. McClellan J, Kowatch R, Findling RL. Work Group on Quality I. Practice parameter for the assessment and treatment of children and adolescents with bipolar disorder. Journal of the American Academy of Child and Adolescent Psychiatry. 2007; 46(1):107–125. [PubMed: 17195735]
4. Miklowitz DJ, Axelson DA, Birmaher B, et al. Family-focused treatment for adolescents with bipolar disorder: results of a 2-year randomized trial. Archives of general psychiatry. 2008; 65(9): 1053–1061. [PubMed: 18762591]
5. Fristad MA, Verducci JS, Walters K, Young ME. Impact of multifamily psychoeducational psychotherapy in treating children aged 8 to 12 years with mood disorders. Archives of general psychiatry. 2009; 66(9):1013–1021. [PubMed: 19736358]
6. West AE, Weinstein SM, Peters AT, Katz AC, Pavuluri M, Henry D. A randomized controlled trial of child and family focused cognitive behavioral therapy (CFF-CBT) for pediatric bipolar disorder. The Journal of the American Academy of Child and Adolescent Psychiatry. 2014; 53:1168–1178.
7. National Institute of Mental Health. Strategic Plan: Envisioning a world in which mental illnesses are prevented and cured. (Publication No. 08–6368). http://www.nimh.nih.gov/about/strategic-planning-reports/index.shtml. Published August 2008. Retrieved September 23, 2014
8. Beauchaine TP, Webster-Stratton C, Reid MJ. Mediators, moderators, and predictors of 1-year outcomes among children treated for early-onset conduct problems: a latent growth curve analysis. Journal of consulting and clinical psychology. 2005; 73(3):371–388. [PubMed: 15982136]
9. Ginsburg GS, Kingery JN, Drake KL, Grados MA. Predictors of treatment response in pediatric obsessive-compulsive disorder. Journal of the American Academy of Child and Adolescent Psychiatry. 2008; 47(8):868–878. [PubMed: 18596553]
10. Compton SN, Peris TS, Almirall D, et al. Predictors and moderators of treatment response in childhood anxiety disorders: results from the CAMS trial. Journal of consulting and clinical psychology. 2014; 82(2):212–224. [PubMed: 24417601]
11. van den Hoofdakker BJ, Nauta MH, van der Veen-Mulders L, et al. Behavioral parent training as an adjunct to routine care in children with attention-deficit/hyperactivity disorder: moderators of treatment response. Journal of pediatric psychology. 2010; 35(3):317–326. [PubMed: 19633060]
12. Brent DA, Kolko DJ, Birmaher B, et al. Predictors of treatment efficacy in a clinical trial of three psychosocial treatments for adolescent depression. Journal of the American Academy of Child and Adolescent Psychiatry. 1998; 37(9):906–914. [PubMed: 9735610]
13. Birmaher B, Brent DA, Kolko D, et al. Clinical outcome after short-term psychotherapy for adolescents with major depressive disorder. Archives of general psychiatry. 2000; 57(1):29–36. [PubMed: 10632230]
14. Emslie GJ, Mayes T, Porta G, et al. Treatment of Resistant Depression in Adolescents (TORDIA): week 24 outcomes. The American journal of psychiatry. 2010; 167(7):782–791. [PubMed: 20478877]
15. Curry J, Rohde P, Simons A, et al. Predictors and moderators of acute outcome in the Treatment for Adolescents with Depression Study (TADS). Journal of the American Academy of Child and Adolescent Psychiatry. 2006; 45(12):1427–1439. [PubMed: 17135988]
16. Rohde P, Seeley JR, Kaufman NK, Clarke GN, Stice E. Predicting time to recovery among depressed adolescents treated in two psychosocial group interventions. Journal of consulting and clinical psychology. 2006; 74(1):80–88. [PubMed: 16551145]
17. Miklowitz DJ, Axelson DA, George EL, et al. Expressed emotion moderates the effects of family-focused treatment for bipolar adolescents. Journal of the American Academy of Child and Adolescent Psychiatry. 2009; 48(6):643–651. [PubMed: 19454920]
18. Macpherson HA, Algorta GP, Mendenhall AN, Fields BW, Fristad MA. Predictors and moderators in the randomized trial of multifamily psychoeducational psychotherapy for childhood mood

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

disorders. Journal of clinical child and adolescent psychology: the official journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53. 2014; 43(3):459–472.

19. Keenan-Miller D, Peris T, Axelson D, Kowatch RA, Miklowitz DJ. Family functioning, social impairment, and symptoms among adolescents with bipolar disorder. Journal of the American Academy of Child and Adolescent Psychiatry. 2012; 51(10):1085–1094. [PubMed: 23021483]
20. Schenkel LS, West AE, Harral EM, Patel NB, Pavuluri MN. Parent-child interactions in pediatric bipolar disorder. Journal of clinical psychology. 2008; 64(4):422–437. [PubMed: 18357574]
21. Jensen PS, Hoagwood K, Petti T. Outcomes of mental health care for children and adolescents: II. Literature review and application of a comprehensive model. Journal of the American Academy of Child and Adolescent Psychiatry. 1996; 35(8):1064–1077. [PubMed: 8755804]
22. Young RC, Biggs JT, Ziegler VE, Meyer DA. A rating scale for mania: reliability, validity and sensitivity. The British journal of psychiatry : the journal of mental science. 1978; 133:429–435. [PubMed: 728692]
23. Poznanski EO, Grossman JA, Buchsbaum Y, Banegas M, Freeman L, Gibbons R. Preliminary studies of the reliability and validity of the children's depression rating scale. Journal of the American Academy of Child Psychiatry. 1984; 23(2):191–197. [PubMed: 6715741]
24. Kaufman, AS.; Kaufman, NL. Kaufman Brief Intelligence Test–Second Edition (KBIT-2). Circle Pines, MN: American Guidance Service; 2004.
25. Geller, B.; Williams, M.; Zimerman, B.; Frazier, J. Washington University in St. Louis Kiddie Schedule for Affective Disorders and Schizophrenia (WASH-U-KSADS). St. Louis, MO: Washington University; 1996.
26. Posner K, Brown GK, Stanley B, et al. The Columbia-Suicide Severity Rating Scale: initial validity and internal consistency findings from three multisite studies with adolescents and adults. The American journal of psychiatry. 2011; 168(12):1266–1277. [PubMed: 22193671]
27. Mattos P, Rohde LA. The Kiddie-Sads-Present and Lifetime Version. Journal of Attention Disorders. 2007; 11(2):100–100. [PubMed: 17709813]
28. Urbaniak, GC.; Plous, S. Research randomizer. Social Psychology Network; http://www.randomizer.org/. Published 1997
29. Pavuluri MN, Henry DB, Devineni B, Carbray JA, Naylor MW, Janicak PG. A pharmacotherapy algorithm for stabilization and maintenance of pediatric bipolar disorder. Journal of the American Academy of Child and Adolescent Psychiatry. 2004; 43(7):859–867. [PubMed: 15213587]
30. Pavuluri MN, Henry DB, Devineni B, Carbray JA, Birmaher B. Child mania rating scale: development, reliability, and validity. Journal of the American Academy of Child and Adolescent Psychiatry. 2006; 45(5):550–560. [PubMed: 16601399]
31. Youngstrom E, Meyers O, Demeter C, et al. Comparing diagnostic checklists for pediatric bipolar disorder in academic and community mental health settings. Bipolar disorders. 2005; 7(6):507–517. [PubMed: 16403176]
32. West AE, Celio CI, Henry DB, Pavuluri MN. Child Mania Rating Scale-Parent Version: A valid measure of symptom change due to pharmacotherapy. Journal of affective disorders. 2011; 128(1): 112–119. [PubMed: 20858565]
33. Spearing MK, Post RM, Leverich GS, Brandt D, Nolen W. Modification of the Clinical Global Impressions (CGI) Scale for use in bipolar illness (BP): the CGI-BP. Psychiatry research. 1997; 73(3):159–171. [PubMed: 9481807]
34. Beck, AT.; Steer, RA.; Brown, GK. Manual for the Beck Depression Inventory-II. San Antonio, TX: Psychological Corporation; 1996.
35. Olson D. FACES IV and the Circumplex Model: validation study. Journal of marital and family therapy. 2011; 37(1):64–80. [PubMed: 21198689]
36. Conners, C.; March, J. The Conners/March Developmental Questionnaire. Toronto, ON: MultiHealth Systems Inc; 1996.
37. Piers, EV. The Piers-Harris children's self concept scale. Los Angeles, CA: Western Psychological Services; 2002.
38. Laird NM, Ware JH. Random-effects models for longitudinal data. Biometrics. 1982; 38(4):963–974. [PubMed: 7168798]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

39. Cohen, J. Statistical power analysis for the behavioral sciences. Hillside, NJ: Lawrence Erlbaum Associates; 1988.
40. Gotlib IH, Lewinsohn PM, Seeley JR. Symptoms versus a diagnosis of depression: differences in psychosocial functioning. Journal of consulting and clinical psychology. 1995; 63(1):90–100. [PubMed: 7896995]
41. Lewinsohn PM, Solomon A, Seeley JR, Zeiss A. Clinical implications of “subthreshold” depressive symptoms. Journal of abnormal psychology. 2000; 109(2):345–351. [PubMed: 10895574]
42. Pincus HA, Davis WW, McQueen LE. ‘Subthreshold’ mental disorders. A review and synthesis of studies on minor depression and other ‘brand names’. The British journal of psychiatry : the journal of mental science. 1999; 174:288–296. [PubMed: 10533546]
43. Feeny NC, Silva SG, Reinecke MA, et al. An exploratory analysis of the impact of family functioning on treatment for depression in adolescents. Journal of clinical child and adolescent psychology : the official journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53. 2009; 38(6):814–825.
44. Barbe RP, Bridge J, Birmaher B, Kolko D, Brent DA. Suicidality and its relationship to treatment outcome in depressed adolescents. Suicide & life-threatening behavior. 2004; 34(1):44–55. [PubMed: 15106887]
45. Asarnow JR, Emslie G, Clarke G, et al. Treatment of selective serotonin reuptake inhibitor-resistant depression in adolescents: predictors and moderators of treatment response. Journal of the American Academy of Child and Adolescent Psychiatry. 2009; 48(3):330–339. [PubMed: 19182688]
46. Cummings CM, Fristad MA. Anxiety in children with mood disorders: a treatment help or hindrance? Journal of abnormal child psychology. 2012; 40(3):339–351. [PubMed: 21912843]
47. Boylan K, Macpherson HA, Fristad MA. Examination of disruptive behavior outcomes and moderation in a randomized psychotherapy trial for mood disorders. Journal of the American Academy of Child and Adolescent Psychiatry. 2013; 52(7):699–708. [PubMed: 23800483]
48. Geller B, Craney JL, Bolhofner K, Nickelsburg MJ, Williams M, Zimerman B. Two-year prospective follow-up of children with a prepubertal and early adolescent bipolar disorder phenotype. The American journal of psychiatry. 2002; 159(6):927–933. [PubMed: 12042179]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Clinical Guidance

- Findings point to the relevance of assessing parent depression; an intensive focus on parent wellbeing through individual sessions, alongside child/family work, is indicated for caregivers with even subclinical symptoms. Tailoring treatment to address parent self-efficacy, negative cognitions related to coping with PBD, self-care, and support systems may be particularly efficacious.
- Clinicians may consider specialized PBD treatment for families with lower income. A structured curriculum with content focused on psychoeducation, parent management strategies for affect dysregulation, parental well-being, and family coping may enhance outcomes for families experiencing financial difficulty and related parental distress.
- Effective participation in family therapy for PBD may be improved by targeting family cohesion at treatment outset, including promoting family strengths, increasing positive family activities, and improving communication.
- Manualized treatment for PBD may be indicated for youth presenting with less acute symptoms. Clinicians are cautioned against diverting focus from core PBD symptoms and associated difficulties with problem solving, self-esteem, and social functioning in treatment planning.

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

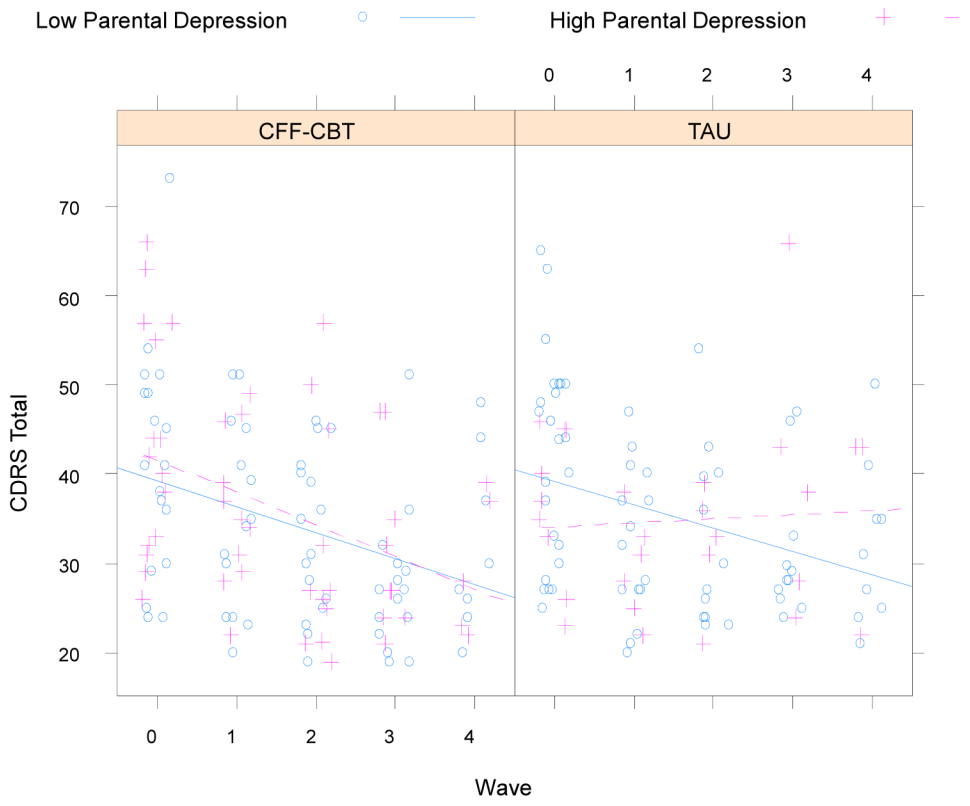

Figure 1.

Adjusted (estimated) depressive symptoms across treatment as a function of parental depression and treatment condition. Note: CDRS = Children's Depression Rating Scale-Revised; CFF-CBT=child-and family-focused cognitive-behavioral therapy; TAU=treatment as usual; Wave: 0=baseline; 1=4 weeks; 2=8 weeks; 3=12 weeks/posttreatment; 4=6 month follow-up.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

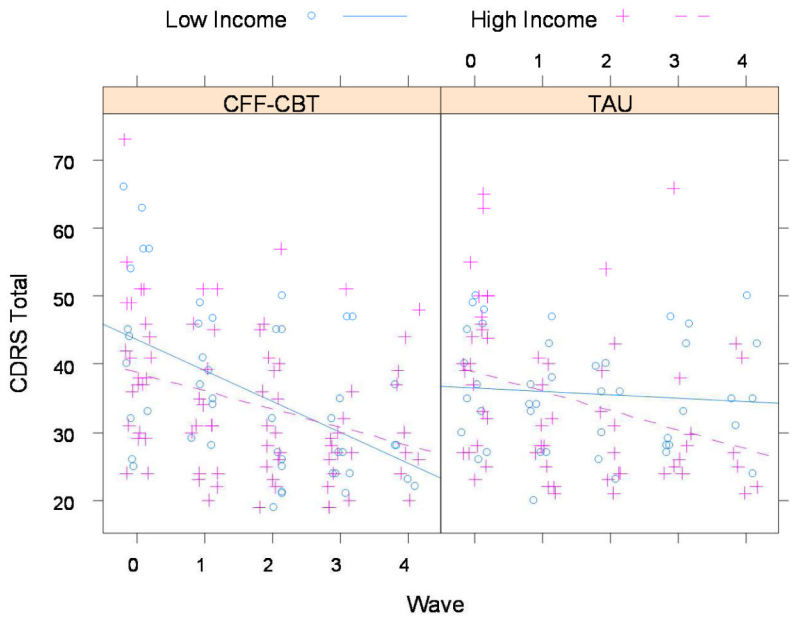

Figure 2.

Adjusted (estimated) depressive symptoms across treatment as a function of income and treatment condition. Note: CDRS = Children's Depression Rating Scale-Revised; CFF-CBT=child-and family-focused cognitive-behavioral therapy; TAU=treatment as usual; Wave: 0=baseline; 1=4 weeks; 2=8 weeks; 3=12 weeks/posttreatment; 4=6 month follow-up.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

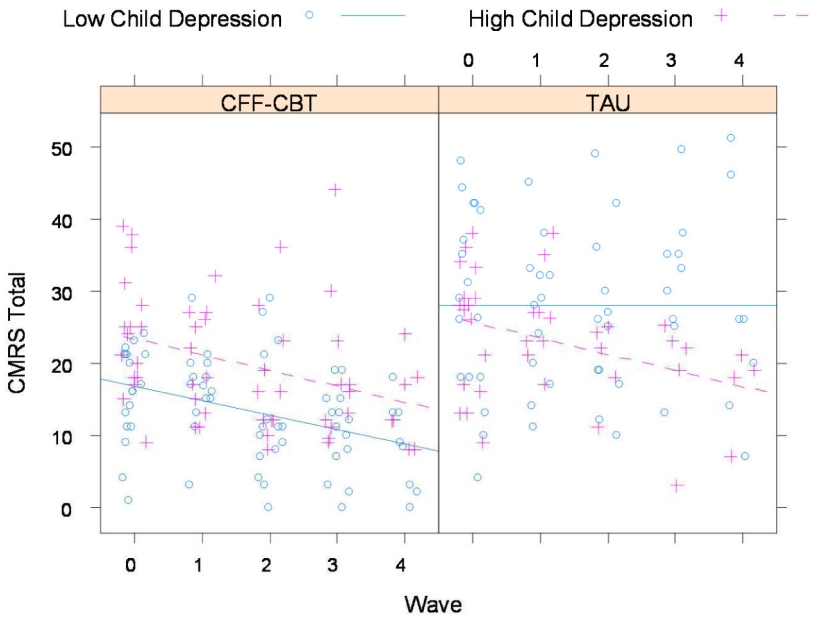

Figure 3.

Adjusted (estimated) mania symptoms across treatment as a function of child depression and treatment condition. Note: CFF-CBT=child-and family-focused cognitive-behavioral therapy; CMRS=Child Mania Rating Scale; TAU=treatment as usual; Wave: 0=baseline; 1=4 weeks; 2=8 weeks; 3=12 weeks/posttreatment; 4=6 month follow-up.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Table 1

Descriptive Statistics for Moderator and Outcome Variables at Baseline, by Treatment Condition

	CFF-CBT (n = 34)		TAU (n = 35)	
Variable	**M**	**SD**	**M**	**SD**
Age	9.26	1.93	9.11	1.25
Child Mania Rating Scale*	19.82	8.59	26.82	11.18
Children's Depression Rating Scale	42.26	12.47	40.71	10.65
Clinical Global Impressions Scale-Severity	4.06	0.67	4.12	0.48
Piers-Harris Self-Concept Scale	42.91	10.11	43.85	10.74
Beck Depression Inventory (Parent)	12.30	10.82	8.77	8.99
FACES Cohesion Scale	27.61	4.48	29.06	4.66
	n	**%**	**n**	**%**
Female Sex	16	47	13	37
Family Income (<$50,000/year)	12	43	12	39
Comorbid Anxiety	13	38	13	38
Comorbid Disruptive Behavior	27	79	32	94
Child Suicidal Ideation (any current)	16	37	11	32

Note: Percentages calculated based on number of available cases. CFF-CBT = Child-and Family-Focused Cognitive Behavioral Therapy; FACES = the Family Adaptability and Cohesion Evaluation Scale; TAU = treatment as usual.

* $p < .05$ on t-test or chi-square analyses.

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

Table 2

Moderator Effects From Mixed Effects Regression Models on Child Treatment Outcomes

Parent/Family Models	Depression Symptoms				Mania Symptoms				Overall Psychiatric Severity			
	Estimate	*SE*	*t* (df)	*d*	Estimate	*SE*	*t* (df)	*d*	Estimate	*SE*	*t* (df)	*d*
1 Parent Depression × Treatment × Wave	0.28	0.11	2.34* (205)	0.57	−0.05	0.10	−0.54 (194)	0.13	0.02	0.01	1.76^{t} (198)	0.44
2 Cohesion × Treatment × Wave	0.28	0.16	1.77^{t} (171)	0.43	−0.04	0.12	−0.36 (162)	0.09	0.03	0.01	1.94^{t} (166)	0.48
3 Income × Treatment × Wave	−3.31	1.52	−2.18* (170)	0.53	1.27	1.20	1.06 (165)	0.26	−0.17	0.14	−1.27 (171)	0.32
Child Models												
4 Depression × Treatment × Wave	---	---	---	---	−0.12	0.06	−2.20* (170)	0.54	−0.01	0.01	−1.21 (181)	0.30
5 Mania × Treatment × Wave	0.04	0.08	0.57 (175)	0.14	---	---	---	---	0.00	0.01	0.06 (172)	0.01
6 Self-Esteem x Treatment × Wave	0.06	0.08	0.82 (178)	0.20	0.15	0.06	2.48* (164)	0.61	0.01	0.01	1.66 (168)	0.41
7 Suicidal Ideation × Treatment × Wave	0.84	1.60	0.53 (175)	0.13	−1.66	1.18	−1.40 (166)	0.34	−0.08	0.14	0.58 (171)	0.14
8 Anxiety × Treatment × Wave	−0.57	1.59	−0.36 (176)	0.27	−1.38	1.23	−1.12 (166)	0.09	−0.01	0.14	−0.06 (174)	0.01
9 DBD × Treatment × Wave	n/a				n/a				n/a			
10 Sex × Treatment × Wave	1.78	1.55	1.15 (170)	0.28	−0.25	1.21	−0.20 (160)	0.05	0.27	0.14	1.97^{t} (166)	0.49
11 Age × Treatment × Wave	0.20	0.57	0.35 (186)	0.08	−0.05	0.45	−0.12 (175)	0.03	−0.06	0.05	−1.24 (180)	0.31

Note: Each model included additional variables; only effects examining moderation are included. DBD = disruptive behavior disorder; n/a = model could not be calculated.

*p<.05;

^{t}p<.10.