

The Bi-Directional Relationship Between Parent–Child Conflict and Treatment Outcome in Treatment-Resistant Adolescent Depression

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Objective: To examine the bidirectional relationship between parent–child discord and treatment outcome for adolescent treatment-resistant depression. **Method:** Depressed youth who had not responded to an adequate course of a selective serotonin reuptake inhibitor (SSRI) were randomized to either a switch to another SSRI or venlafaxine, with or without the addition of cognitive behavior therapy (CBT) in the Treatment of SSRI-Resistant Depression in Adolescents (TORDIA) study. The Conflict Behavior Questionnaire was used to assess adolescent (CBQ-A) and parent-reported (CBQ-P) parent–child discord. The impact of remission on parent–child conflict, and the differential impact of medication and CBT on the CBQ-A and CBQ-P, were assessed using generalized linear models. **Results:** Although there were no differential treatment effects on parent or adolescent-report of conflict, remission was associated with improvement in the CBQ-P. In general, intake family conflict did not predict remission, except in the sub-group of participants whose parents reported clinically significant parent–child conflict at intake, for whom high levels of parent-reported conflict predicted a lower likelihood of remission. Conflict also did not moderate treatment response. **Conclusions:** Remission of depression may be sufficient to reduce parent-reported parent–child conflict. However, higher parent-reported conflict, in the clinically significant range, predicts a lower likelihood of remission from depression. Clinical trial registration information—Treatment of SSRI-Resistant Depression in Adolescents (TORDIA); <http://clinicaltrials.gov/>; NCT00018902. *J. Am. Acad. Child Adolesc. Psychiatry*; 2013;52(4):370-377. **Key Words:** parent–child conflict, selective serotonin reuptake inhibitor (SSRI), cognitive behavior therapy (CBT), venlafaxine, depression.

Parent–child conflict has been shown to be related to the onset, persistence, and recurrence of depressive disorders in childhood and adolescence.^{1,2} Conversely, the onset of depressive symptoms in a family member increases the likelihood of intra-familial interpersonal conflict.^{3–8} In fact, longitudinal studies support a bi-directional relationship between family conflict and depressive symptoms.⁹ Since family conflict is so intimately related to the course of depressive disorder in youth, a better understanding of its role in treatment response

could be helpful in improving treatment outcome of depressed adolescents.

The extant treatment literature provides support for a bi-directional relationship between family climate and depressive outcomes. High family conflict and low family cohesion predict a poorer response to the treatment of depression, and a greater likelihood of suicidal events.^{4,10–14} Interventions that target family interactions have been shown to decrease depressive symptomatology in unipolar and bipolar depressed youth,^{15–18} with some evidence that improvement in family climate mediates the impact of treatment and that higher levels of family conflict can be positive moderators of treatment outcome.^{19–21} Conversely,



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improvements in child and adolescent depressive symptomatology can reduce parent–child conflict, even when the intervention does not specifically target family interactions.^{5–7} In addition, family conflict can be a negative moderator of treatment outcome. Specifically, in the Treatment of Adolescent Depression Study (TADS), an unfavorable adolescent-reported family environment negatively moderated cognitive behavior therapy (CBT) treatment response.¹³

Most of the treatment studies that have examined the role of family conflict with respect to depressive outcome have reported only on baseline family conflict, rather than exploring the longitudinal inter-relationship of family conflict and youth depression over time. Therefore, we sought to examine the longitudinal inter-relationship between the course of depression and family conflict over time in the Treatment of Selective Serotonin Reuptake Inhibitor (SSRI)–Resistant Depression in Adolescents study (TORDIA).²² In this study, 334 depressed adolescents who had not responded to an adequate trial with an SSRI were randomized to a switch either to another SSRI or to venlafaxine, with or without the addition of cognitive behavior therapy (CBT). In TORDIA, we found that the addition of CBT to either medication switch was associated with a higher response rate, but there was no differential effect of medication.²² Predictors and correlates of poorer treatment response included more severe depressive symptomatology, history of non-suicidal self-injury, high or increasing alcohol or drug use during treatment, and subsyndromal manic symptoms.^{10,23,24} A history of maltreatment was a negative moderator of CBT response, whereas the greater the number of comorbid diagnoses, the greater the advantage conveyed by the addition of CBT to medication.^{10,25} Relevant to this study, adolescent-reported family conflict at intake was a predictor of poorer clinical response, suicidal events, and suicidal attempts.^{11,26} However, the interrelationship between treatment response and adolescent and parent reported family conflict over time has not yet been examined in this sample.

Based on the extant literature, we hypothesized the following: that higher levels of adolescent and parent-reported family conflict would predict lack of remission, and would negatively moderate CBT response; that remission would be associated with reductions in parent–child conflict; and that reductions in family conflict would be greatest in

those who received a combination of CBT and medication.

METHOD

Participants

Participants consisted of 334 adolescents between 12 and 18 years of age enrolled in the Treatment of SSRI-Resistant Depression in Adolescents (TORDIA) study. The participants had to have a clinical diagnosis of major depressive disorder (MDD) or dysthymia by *DSM-IV* criteria,²⁷ moderate-to-severe depressive symptoms, (≥ 40 on the Children's Depression Rating Scale–Revised [CDRS-R],²⁸ and ≥ 4 on the Clinical Global Impression–Severity Subscale [CGI-S]^{28,29}) which persisted despite an 8-week trial with an SSRI.²² Informed assent/consent was obtained from participants and families as per the Institutional Review Boards of all six sites.

Study Design

Participants were randomly assigned to four treatment groups in a 2×2 balanced design, with a switch in medication to either another SSRI ($n = 168$) or venlafaxine ($n = 166$), with or without receipt of cognitive behavior therapy (CBT). SSRIs included paroxetine ($n = 50$), citalopram ($n = 34$), and fluoxetine ($n = 84$). Randomization was balanced both within and across the six sites based on: incoming treatment medication, comorbid anxiety, chronic depression (duration ≥ 24 months), and suicidal ideation (Beck Depression Inventory [BDI] item 9 ≥ 2).³⁰ Participants received 12 weeks of acute treatment; responders continued with the same treatment for an additional 12 weeks, whereas nonresponders received open treatment.

Assessment Measures

Age, sex, race/ethnicity, parental education, and income were assessed by parent and youth report. The School Age Schedule for Affective Disorders and Schizophrenia for School-Aged Children–Present and Lifetime Version (K-SADS)³¹ was used to assess for comorbid diagnosis, onset of current MDD episode, onset of first MDD episode, and duration of depression. Depression remission was defined as at least 3 consecutive weeks without clinically significant depressive symptoms, corresponding to a score of 1 on the Adolescent Longitudinal Interval Follow-Up Evaluation.³² Severity and clinical improvement were assessed using the Clinical Global Impressions–Severity (CGI-S) and Improvement Subscales (CGI-I).²⁹ Overall functioning was assessed using the Child Global Assessment Scale (C-GAS).³³ The 17-item, clinician-rated CDRS-R assessed depressive symptoms in the past 2 weeks, based on information collected from both youth and a parent.²⁸ Self-reported depression,

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