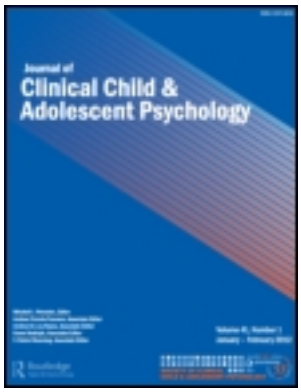

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The goal of the study is to examine whether family conflict generates peer-related stress and subsequent depressive symptoms among adolescents. In addition, in the context of the proposed mediation model, we examine whether negative cognitive styles about the self, cause, and consequences moderate the mediational pathway between peer stress and depressive symptoms. The study includes 179 adolescents (71 boys, 108 girls) ages 12 to 18, and the majority of the participants are Caucasian (79.5%). At the initial assessment, participants completed self-report measures regarding family conflict, negative cognitive style, stress, and depressive symptoms. Participants also completed 3 subsequent self-report assessments every 6 weeks in which information regarding stress and depressive symptoms were collected. Both within- and between-subject analyses indicate that dependent interpersonal peer stress partially mediates the relationship between greater family conflict and higher levels of depressive symptoms. Moreover, results of our moderated-mediation model indicate that negative cognitive styles regarding the self and cause, but not consequences, moderate the mediational pathway between peer stress and depressive symptoms. These findings underscore the importance of examining more comprehensive models that incorporate both cognitive and interpersonal vulnerability.

Adolescents are particularly susceptible to develop major depressive disorder (see Merikangas & Knight, 2009), and it has an insidious impact on psychosocial development throughout adolescence (Rudolph, 2008) and adulthood (Kessler & Wang, 2008). Although both cognitive (e.g., Abela & Hankin, 2009) and interpersonal (e.g., Prinstein, Borelli, Cheah, Simon, & Aikins, 2005) frameworks deconstruct the pathway leading to depression, the majority of research has examined these etiological models independently. At the same time, there are several theoretical reasons to integrate these approaches. Notably, in the elaborated cognitive vulnerability-transactional model, Hankin and Abramson (2002) asserted that an interdisciplinary approach to examining the etiology of depression is critical for understanding how cognitive vulnerability develops and operates within a depressogenic causal chain. More recently, Kercher and Rapee (2009) examined a ''Cognitive Diathesis-Stress Generation Pathway'' as cognitive diathesis-stress theories highlight potential processes that contribute to the emergence of depressive symptoms, whereas stress generation addresses factors that maintain such symptoms. Therefore, in line with research merging diverse theoretical models, the current study sought to examine an integrated cognitive-interpersonal etiological model of adolescent depression. Specifically, our goal is to examine the synergistic relationship of two theories—spillover effect (Repetti, 1989) and hopelessness theory (Abramson, Metalsky, & Alloy, 1989)—using a

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transactional approach in order to provide a more holistic understanding of how depressive symptoms may emerge in adolescence.

SPILLOVER EFFECT: DECONSTRUCTING AN ADOLESCENT'S FAMILY ENVIRONMENT

Within the context of an adolescent's life, family environment has a profound impact on psychosocial development and stress processes (e.g., Garber & Cole, 2010; Hammen, Shih, & Brennan, 2004; Sheeber, Hops, & Davis, 2001). In particular, adolescents are at increased risk for depression when there is (a) chronic family stress accompanied by negative parenting behaviors (e.g., Hammen, Shih, & Brennan); (b) lower levels of support, attachment, and approval (e.g., Hops, Lewinsohn, Andrews, & Roberts, 1990; Kandel & Davies, 1982); and (c) parental disengagement (Lovejoy, Graczyk, O'Hare, & Neuman, 2000). At the same time, family conflict, an interpersonal vulnerability factor, is one of the most robust predictors of adolescent depression (e.g., Cole & McPherson, 1993; Garber & Little, 1999; Mazza et al., 2009). Bond, Toumbourou, Thomas, Catalano, and Patton (2005) conducted a cross-sectional study ($n = 8{,}984$ adolescents) examining a broad range of risk and protective factors of depression, and family conflict had the strongest association with depressive symptoms. In addition, parent–child conflict during early- to mid-adolescence has been found to be associated with greater internalizing symptoms and subsequent depressive disorders (Rueter, Scaramella, Wallace, & Conger, 1999). Similarly, in a 7-year prospective study, family and marital conflict significantly predicted higher levels of depressive symptoms in adolescent girls but not boys (Mazza et al., 2009). As a whole, these findings suggest that conflict within the family environment leads to greater depressive symptoms; however, the mechanism through which family conflict leads to these symptoms remains unclear.

The spillover effect may provide a prism with which to better understand this process. Specifically, it seems plausible that conflict within the family environment may potentially generate a greater occurrence of peer-related stressors, which then leads to depressive symptoms. Such a framework suggests that problems or stress in one domain of life may spillover into another domain. More traditionally, the spillover effect has helped draw a connection between familial and work stress whereby stressful work environments contribute to greater emotional distress among family members (e.g., Repetti, 1989). However, the spillover model may also be germane to adolescent development as the preponderance of an individual's time is spent with one's family or peer group. It is important to note that family environments are youths' first opportunity to learn about relationships, and thus parental and marital conflict may negatively shape an adolescent's "mental model" of effective interpersonal skills and expectations of relationships (e.g., Stocker, Ahmed, & Stall, 1997). For example, Stocker and Youngblade (1999) found that marital conflict is strongly associated with problematic peer relationships among youth, suggesting that internal working models of peer relationships potentially contribute to maladaptive behaviors and/or dysfunctional approaches. Alternatively, Stocker and Youngblade suggested that merely witnessing ineffective expression of negative emotions and communication styles may contribute to emotion regulation deficits in children, which then increases susceptibility to peer conflict. Taken together, these findings strongly suggest that youth's approach to relationships and their subsequent vulnerability to experience peer-associated conflict and stressors may be potentiated by greater family conflict. Therefore, we hypothesized that a conflict-laden family environment would contribute to greater dependent interpersonal peer stressors—stressors thought to be dependent as they result, in part, from an individual's actions. Further, the presence of peer stress would then lead to higher levels of depressive symptoms over time, and moreover, peer stressors would mediate the prospective relationship between family conflict and depressive symptoms.

THE INTERPLAY OF COGNITIVE AND INTERPERSONAL VULNERABILITY

Muller, Judd, and Yzerbyt (2005) posited the "potency of the mediating process depends on the moderator" (p. 856). Therefore, it may be critical to determine whether additional vulnerability factors moderate the mediational pathways in the proposed model just described. Notably, as family conflict may increase the occurrence of stress, and in particular peer stressors, such stress may also activate dormant, underlying cognitive vulnerability factors. Once activated, cognitive vulnerability factors may moderate the pathway between stress and depressive symptoms in the context of the mediational framework. To date, the hopelessness theory (Abramson et al., 1989), a cognitive diathesis-stress model of depression, has received widespread support among adolescents (e.g., Abela, 2001; Hankin, Abramson, & Siler, 2001), and it proposes that depressogenic attributional or negative cognitive styles interact with stressful life events contributing to the occurrence of a specific subtype of depression—hopelessness depression. Research has found that the interplay of depressogenic attributions and stress predicts higher levels of depressive symptoms in general (e.g., Stone,

Gibb, & Coles, 2010) and hopelessness depression symptoms specifically (e.g., Abela & Seligman, 2000). As the hopelessness theory is typically examined within a diathesis-stress framework, it suggests that the interaction of a cognitive diathesis and the occurrence of stressful life events increases the likelihood of experiencing depressive symptoms. Thus, in the absence of stress, vulnerable individuals who possess cognitive diatheses are no more likely than nonvulnerable individuals to experience depressive symptomology (Ingram & Luxton, 2005).

Although a large body of research has examined the hopelessness theory, and by extension the role that depressogenic attributional styles play in the manifestation of depressive symptoms (for review, see Abela & Hankin, 2008), little research has examined whether it arises in the context of an interpersonal model of depression such as the spillover effect framework (e.g., Kercher & Rapee, 2009). In general, the hopelessness theory of depression (Abramson et al., 1989) proposes that depressogenic attributions serve as a vulnerability to depression given the tendency (a) to make global and stable attributions about the causes of negative events, (b) to perceive negative events as having catastrophic consequences, and (c) to identify the self as being intrinsically flawed or inadequate following negative life events (Abela & Hankin, 2008; Calvete, Villardón, & Estévez, 2008). Whereas some research has examined attributional styles individually (i.e., cause, consequences, and/or self), other research has examined attributional styles using a composite score (e.g., Haeffel et al., 2008) or a weakest link approach (e.g., Abela & Sarin, 2002). A composite score methodology assesses an individual's average level of vulnerability across the three dimensions. In contrast, the weakest link hypothesis proposes that an individual's level of vulnerability is operationalized in relation to the most depressogenic attributional style. At present, research has not demonstrated a clear-cut advantage to using independent negative cognitive styles, composite scores, or a weakest link approach; nevertheless, research suggests that there may be age-related issues that are important to consider when making such a decision (e.g., Calvete et al., 2008; Gibb et al., 2006).

Recently, Stone et al. (2010) asserted that similar findings regarding the cause, the consequences, the self, the composite score, and the weakest link approach were obtained in their examination of hopelessness theory. Although the composite and weakest link approach may have great benefit in childhood when depressogenic attributional styles are thought to be unstable (e.g., Abela & Sarin, 2002), these cognitive styles are believed to become more entrenched and automatic during adolescence stabilizing around the age of 12 (Gibb et al., 2006). Moreover, it is conceivable that during adolescence, each attributional style may differentially impact the occurrence of depressive symptoms following stressful life events. Specifically, peer-related stressors may foster depressogenic attributions among adolescents in which they believe that (a) interpersonal stressors are the result of the *self* being deficient, unlovable, and/or unlikable; (b) the *cause* of these relational conflicts are beyond one's control or capacity to change; and/or (c) the *consequences* are catastrophic, resulting in social isolation or alienation. Once present, each of these depressogenic attributions may strengthen the association between peer stress and subsequent depressive symptoms. In the current study, we sought to examine whether individual depressogenic attributional styles (i.e., cause, consequences, and self) moderated the mediational pathway between peer stressors and subsequent depressive symptoms. In doing so, we examined the interplay of the spillover effect (i.e., the mediation model) and the hopelessness theory (i.e., the moderation model).

GOALS OF THE CURRENT STUDY

The aim of the current study is to build on existing cognitive (i.e., hopelessness theory) and interpersonal vulnerability (i.e., spillover effect) models of depression in adolescents and, in doing so, address an important theoretical gap. We utilize a multiwave, longitudinal design in order to delineate the time-lagged relationship among cognitive and interpersonal vulnerability, stress, and subsequent depressive symptoms. Initially, we examine the role that family conflict plays in the spillover effect framework, and we hypothesize that dependent interpersonal (i.e., peer-related stressors) stressors would mediate the relationship between greater family conflict and higher levels of depressive symptoms (see Figure 1). In addition, using a moderated-mediation data analytic approach (see Figure 2), we examine an integrated cognitive-interpersonal vulnerability model of adolescent depression. Meaning, in the context of the spillover effect framework, we hypothesize that greater family conflict would contribute to higher levels

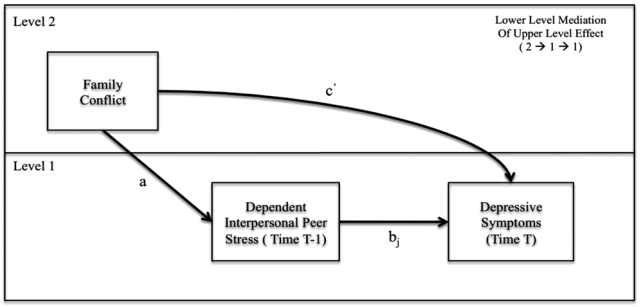

FIGURE 1 Time-lagged multilevel model examining the relationship among family conflict, stress, and depressive symptoms.

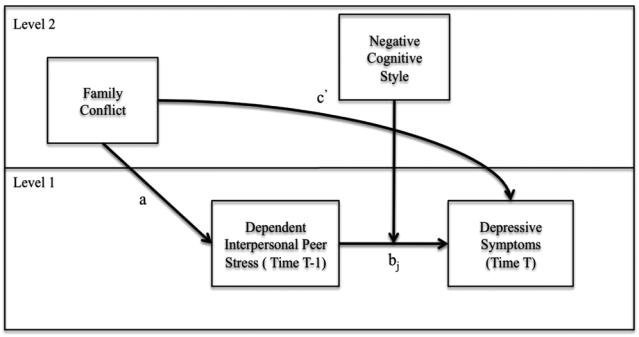

FIGURE 2 Integrated cognitive-interpersonal adolescent model.

of dependent interpersonal peer-related stress and subsequent depressive symptoms. Further, the occurrence of such stress may also activate depressogenic attributional styles (i.e., regarding the self, causes, and consequences), and thus we believe that negative cognitive styles will moderate the mediational pathway between peer stress and depressive symptoms.

METHOD

Participants

The current study recruited participants from a high school in Montreal, Canada, and the sample included 179 adolescents (71 boys, 108 girls). Ages ranged from 12 to 18 ($M = 13.87$, $SD = 1.41$), and the ethnic distribution indicated the following: 79.5% Caucasian, 10.2% Asian, 5.1% Black, 1.7% Hispanic, and 3.4% reported other.

Procedure

The university ethics board provided approval for the study, and the treatment of participants was in accordance with APA ethical standards. Prior to the onset of the study, letters of informed consent were sent home to parents describing the aims of the current study as well as requesting consent for their child's participation. All assessments occurred during school hours on school grounds. During the initial consent, participants completed a demographics form and the following questionnaires: (a) Center for Epidemiologic Studies Depression Scale (Radloff, 1977), (b) Adolescent Life Event Questionnaire–Revised (Hankin & Abramson, 2002), (c) Family Environment Scale (Moos & Moos, 1983), and (d) Adolescent Cognitive Style Questionnaire (Hankin & Abramson, 2002). Participants also completed three subsequent waves of assessment, which occurred every 6 weeks for 4.5 months (Times 2–4). A 6-week interval was selected as we were interested in examining the time-lagged relationship between the occurrence of stress and subsequent depressive symptoms. Consequently, the timing of assessments enabled participants to recall events that had occurred in their lives as well as experience changes in both stress and depressive symptom levels. The rates of retention for each follow-up were 90%, 87%, and 88%. Each participant completed a minimum of three of the four assessments. At the conclusion of the study, all participants were provided a cognitive-behavioral skills workshop that (a) discussed the project aims, (b) taught evidenced-based skills to challenge maladaptive patterns of thinking, and (c) offered counseling services within the greater Montreal area for individuals in need.

Measures

Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). The CES-D is a 20-item self-report measure that assesses levels of depressive symptoms. Examples of questions include "I felt sad," "I felt hopeless about the future," and "I felt lonely." Items on the scale ranged from 0 to 3 with possible total scores ranging from 0 to 60, and higher scores reflect greater depressive symptomology. The CES-D has been shown across numerous studies to have strong test–retest reliability and high correlations with other measures of depressive symptoms (Radloff, 1991). Whereas the CES-D was administered every 6 weeks, participants reported how they felt during the past week by using the following scale: rarely (<1 day), some or a little of the time (1–2 days), occasionally or a moderate amount of time (3–4 days), and most or all of the time (5–7 days). Across administrations the Cronbach's alpha ranged from .92 to .93, which indicates high internal consistency.

Adolescent Life Events Questionnaire–Revised (Hankin & Abramson, 2002). The Adolescent Life Events Questionnaire–Revised is a 57-item self-report instrument that was developed to assess a broad range of negative life events occurring in the past month. In previous research, a consensus team, which included two psychologists and two advanced doctoral students, rated whether items were (a) dependent (i.e., at least in part dependent on the actions of the individual), (b) interpersonal, and (c) noninterpersonal (see Auerbach, Eberhart, & Abela, 2010). It is important to note that only interpersonal events were rated as dependent as more contextual information would be needed for coding noninterpersonal events. Items were retained only if there was unanimous agreement, and items were excluded when a consensus could not be reached. Twenty-nine items were rated as both dependent and interpersonal, and exemplar items are "You fought with

your parents over your personal goals, desires, or choice of friends'' and ''You had an argument with a close friend.'' As the current study was interested in exploring family conflict as a contributor to peer stressors, 18 of the 29 items were used to create a separate subscale regarding peer-specific dependent interpersonal stressors (e.g., ''A close friend did not treat you as well as he/she used to''). For all items, participants rated how often such events occurred on a Likert scale ranging from 1 (*never*) to 5 (*always*), and higher scores reflected a greater number of negative life events.

Family Environment Scale (Moos & Moos, 1983). The Family Environment Scale is a 90-item self-report measure that examines family dynamics. In the current study, we were particularly interested in the impact of family conflict, and thus, we only utilized the nine-item Family Conflict subscale. The Family Conflict subscale assesses the amount of expressed anger and conflict among family members, and respondents must indicate whether a given statement is true (1) or false (2). Sample items include ''In our family, we believe you don't ever get anywhere by raising your voice'' and ''Family members sometimes hit each other.'' Higher scores are indicative of greater family conflict, and in the current study, the Cronbach's alpha was .78 indicating moderate internal consistency.

Adolescent Cognitive Style Questionnaire (ACSQ; Hankin & Abramson, 2002). The ACSQ assesses cognitive vulnerability including negative inferences for cause, consequence, and self. The ACSQ is composed of 12 hypothetical negative life event scenarios (a) across interpersonal and achievement domains and (b) relevant to adolescents. Examples of events include ''You take a test and get a bad grade,'' ''You don't get chosen for an extracurricular activity that you want to be a part of,'' and ''You get into a big fight with your parents.'' First, participants are provided with the hypothetical event and are asked to provide one cause for the event. Then, participants rate the degree to which the cause of the event is internal, stable, and global. In line with the hopelessness theory of depression (Abramson et al., 1989), the negative attributions for cause, or the Generality subscale, was calculated by taking the average of the Global and Stable subscales and did not include the internal item responses. Second, participants rate the likelihood that future consequences will result from the negative event (i.e., negative attributions of consequences). Last, participants rate whether the occurrence of the event signifies whether the self is flawed (i.e., negative attributions of self). Items on the ACSQ range from 1 to 7, and the average response across items within a given subscale is computed. The Cronbach's alpha for the negative attributions dimensions included .90 cause, .86 consequences, and .88 self.

Overview of Data Analytic Approach

To examine our proposed mediation models, we utilize idiographic, time-lagged multilevel modeling in which time is nested within individuals and followed the guidelines for multilevel mediation analyses established by Bauer, Preacher, and Gil (2006). Such an approach is ideal for examining mediation models that include repeated measures, and given that the model is estimated in a single equation, one can directly estimate the covariance of the random effects that are encompassed in different Level 1 and Level 2 models (see Auerbach, Eberhart, et al., 2010). Consequently, Bauer and colleagues' (2006) data analytic approach is preferable to mediation models that utilize a step-by-step process, which makes the implicit assumption that each of the steps is independent (e.g., Baron & Kenny, 1986; Kenny, Korchmaros, & Bolger, 2003). To examine our lower level mediation model with a single equation, it was necessary to use a selection variable, Z, by stacking Y and M (i.e., the dependent variable and mediator) for each unit *i* (i.e., individuals) within *j* (i.e., time; Bauer et al., 2006). By using a single outcome variable, we can then fit a multivariate model using a univariate model approach. However, given that Z may represent different outcome variables (i.e., dependent interpersonal stress and depressive symptoms), it is also necessary to create two separate selection variables, S_M and S_Y. Thus, when Z represents the mediator (i.e., stress), S_M is set to equal 1, whereas S_Y is set to equal 0. In contrast, if Z is the outcome variable (i.e., depressive symptoms), then S_Y is equal to 1 and S_M is 0. An example of the single, simultaneous model is next[1]:

$$Z_{ij} = S_{Mij}(d_{Mj} + a_j X_{ij}) + S_{Yij}(d_{Yj} + b_j M_{ij} + c'_j X_{ij}) + error_{Zij}$$

To examine whether dependent interpersonal $stress_{(Time\ T-1)}$ mediates the relationship between family conflict and depressive $symptoms_{(Time\ T)}$, analyses were carried out using SAS (version 9.2) mixed procedure and maximum likelihood estimation. Our dependent variable is within-subject fluctuations in depressive $symptoms_{(Time\ T)}$ which is a Level 1 variable. The primary predictor of depressive $symptoms_{(Time\ T)}$ is family conflict, a between-subject and Level 2 variable, and the mediator was within-subject fluctuations of dependent interpersonal $stress_{(Time\ T-1)}$, a Level 1 variable. The mediation effect of stress is given by a^*b_j (see Figure 1), and the 95% confidence interval (CI) for the mediation

[1]The example of the single, simultaneous models is drawn from Bauer, Preacher, and Gil (2006, p. 146).

effect is computed following the formula presented in Bauer et al. (2006). The mediation effect is considered statistically significant if zero is not included in the CI. The following additional effects are also included in this initial mean structure. First, to control for individual differences in baseline levels of depressive symptoms, participant's initial depressive is included in the model. Second, to account for individual variability in the average level of depressive symptoms at his or her mean level of dependent interpersonal stress a random effect for intercept is included in the model. The inclusion of the random effect for intercept accommodates for the between-subject differences in the depressive symptoms after the fixed effects of the predictors (i.e., family conflict, interpersonal stress, and negative cognitive style) are accounted for in the model. Third, given that dependent interpersonal stress is a within-subject predictor with an effect expected to vary from participant to participant, a random effect for slope is included in the model. Last, preliminary analyses indicated that neither age nor gender moderated our effects; however, both age and gender are included as covariates in all estimated models.

As the strength of any mediating model often depends on the moderator (Muller, Judd, & Yzerbyt, 2005), we also examined whether attributional style, a cognitive vulnerability factor, strengthened the mediational pathways. More specifically, we estimated models in which attributional style, a between-subject predictor and Level 2 variable, moderated the pathway between dependent interpersonal $\text{stress}_{(\text{Time T-1})}$ and depressive $\text{symptoms}_{(\text{Time T})}$. Similar to the mediation model just described, the moderated-mediation models included the same covariates and random effects for intercept and slope. Similar to the mediation analyses, preliminary analyses indicated that neither age nor gender moderated the findings.

Between-subject effects were also estimated to examine changes in depressive symptoms over time. Moreover, fixed effects were utilized from within-subject analyses to examine the predictive capacity of the proposed model previously described. Specifically, fixed effects accounting for the relationship between family conflict, negative cognitive style, peer stress, and depressive symptoms were used to examine between-subject differences in depressive symptoms. Two- and three-way repeated analyses of variance (ANOVAs) with time, family conflict, and negative cognitive style as factors were utilized to predict differences in depressive symptoms for individuals reporting high versus low vulnerability. Median splits were utilized to operationalize high versus low family conflict as well as high versus low negative cognitive style (i.e., higher scores on negative cognitive style denote greater vulnerability).

RESULTS

Descriptive Statistics

Bivariate Pearson correlations as well as descriptive statistics including mean, standard deviation, and range are included in Table 1.

Main Effect Model: Family Environment Conflict Predicting Depressive Symptoms

Preliminary multilevel models estimated whether family environment conflict predicts higher levels of depressive symptoms over time. The model includes both age and gender as covariates, and all models include an autoregressive covariance structure and random intercept. Results indicate that greater family environment conflict predicts higher levels of depressive symptoms over the follow-up period ($b = 3.87$, $SE = 0.71$), $t(168) = 5.48$, $p < .001$.

Mediation: Family Conflict as a Predictor of Peer Stress and Depressive Symptoms

Given the prospective relationship between family conflict and depressive symptoms, we examined whether dependent interpersonal peer $\text{stress}_{(\text{Time T-1})}$ mediates the relationship between family environment conflict and depressive $\text{symptoms}_{(\text{Time T})}$ using Bauer and colleagues' (2006) single equation simultaneous approach (see Figure 1). All models include an autoregressive heterogeneous covariance structure ($p < .0001$) as well as appropriate random effects for slope and intercept. Of primary importance, greater levels of family conflict predict higher levels of dependent interpersonal peer stress

TABLE 1
Pearson Correlations, Means, Standard Deviation, and Range for Depressive Symptoms, Dependent Interpersonal Stress, Family Conflict, Negative Cognitive Styles at the Initial Assessment

Variables	*1.*	*2.*	*3.*	*4.*	*5.*	*6.*
1. Depressive Symptoms	—					
2. Dependent Interpersonal Peer Stress	.49***	—				
3. Family Conflict	.42***	.31***	—			
4. ASCQ–Self	.32***	.18**	.19*	—		
5. ASCQ–Cause	.34**	.26***	.33**	.73***	—	
6. ASCQ–Consequences	.23**	.22**	.23**	.80***	.83***	—
M	14.49	54.15	12.85	2.37	2.36	2.90
SD	12.22	15.82	2.58	1.19	1.02	1.00
Low	0	13	9	1	1	1
High	52	82	18	5.44	5.22	5.89
Range	52	69	9	4.44	4.22	4.89

Note: ACSQ = Adolescent Cognitive Style Questionnaire (Hankin & Abramson, 2002).

*$p \leq .05$. **$p < .01$. ***$p < .001$.

TABLE 2
Time Lagged Mediation Model for Family Conflict, Dependent Interpersonal Stress, and Depressive Symptoms

Predictor	*Parameter Estimate (b)*	*Standard Error*	*t Value*	*df*
Dependent Interpersonal Stress$_{(\text{Time T-1})}$				
Age	0.34	0.46	0.75	861
Gender	3.28	1.28	2.56*	861
Family Conflict	2.57	0.67	3.85***	861
Depressive Symptoms$_{(\text{Time T})}$ model				
Age	–0.25	0.38	–0.65	861
Gender	4.18	1.09	3.82***	861
Initial Depressive Symptoms	0.38	0.05	7.49***	861
Initial Dependent Interpersonal Stress	–0.23	0.07	–3.29**	861
Dependent Interpersonal Stress$_{(\text{Time T-1})}$	0.13	0.06	2.44*	861
Family Conflict	2.13	0.61	3.49***	861

Note: Dependent interpersonal stress measures peer-related stressors. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

over time (**path a**: $b = 2.57$, $SE = 0.67$), $t(861) = 3.85$, $p < .001$. When controlling for the proportion of variance accounted for by dependent interpersonal peer stress$_{(\text{Time T-1})}$ in predicting higher levels of follow-up depressive symptoms$_{(\text{Time T})}$ (**path b_j**: $b = 0.14$, $SE = 0.06$), $t(861) = 2.44$, $p < .05$, high levels of dependent interpersonal peer stress$_{(\text{Time T-1})}$ partially mediate the relationship between high levels of family conflict and high levels of depressive symptoms$_{(\text{Time T})}$ (**path c'**: $b = 2.13$, $SE = 0.61$), $t(861) = 3.49$, $p < .001$. The 95% CI (**path a*b$_j$**: $b = 0.35$, $SE = 0.18$; 0.005, 0.69) suggests that the mediation effect is significant (see Table 2).

To provide a more stringent examination of the proposed mediation model, additional analyses were conducted examining the reverse model. The same multilevel model, idiographic, time-lagged data analytic approach was utilized with the exception that (a) depressive symptoms$_{(\text{Time T-1})}$ was the mediator and (b) the dependent variable was dependent interpersonal peer stress$_{(\text{Time T})}$. It is important to note that higher levels of family conflict were associated with greater levels of depressive symptoms (**path a**: $b = 4.02$, $SE = 0.72$), $t(862) = 5.57$, $p < .001$; however, depressive symptoms$_{(\text{Time T-1})}$ did not predict higher levels of peer stressors$_{(\text{Time T})}$ (**path b_j**: $b = -0.06$, $SE = 0.04$), $t(862) = -1.49$, *ns*. As this pathway is critical for mediation to occur, the mediation model is not significant suggesting that our proposed model is unidirectional in nature.

Moderated-Mediation: An Integrated Model Examining Family Conflict and Attributional Style

In our next set of analyses, we sought to examine our hypothesized moderated-mediation model or integrated model (see Figure 2). Thus, in a single, simultaneous model, we examined whether negative cognitive style (i.e., self, cause, or consequences) moderated the mediational pathway between dependent interpersonal peer stress$_{(\text{Time T-1})}$ and subsequent depressive symptoms$_{(\text{Time T})}$ within the context of our broader mediation model just outlined. First, in line with our hypothesis, greater family conflict predicts higher levels of dependent interpersonal peer stress over time (**path a**: $b = 2.38$, $SE = 0.07$), $t(806) = 3.49$, $p < .001$. In addition, negative self-cognitive style moderated the pathway between dependent interpersonal peer stress$_{(\text{Time T-1})}$ and higher levels of depressive symptoms, and further, peer stress (**path b_j**: $b = 0.04$, $SE = 0.02$), $t(806) = 2.30$, $p < .05$, partially mediates the relationship between family conflict and depressive symptoms$_{(\text{Time T})}$ (**path c'**: $b = 1.96$, $SE = 0.63$), $t(806) = 3.09$, $p < .001$. The 95% CI does not include zero (**path a*bj**: b = 0.08, $SE = 0.04$; 0.004, 0.16), indicating that the test of the indirect suggests that the mediation effect is significant. Second, when examining the negative cause cognitive style as a moderator, the results indicate that greater family conflict predicted higher levels of depressive symptoms (**path a**: $b = 2.42$, $SE = 0.69$), $t(801) = 3.50$, $p < .001$. Further, the negative cause cognitive style moderated the relationship between peer stressors$_{(\text{Time T-1})}$ and depressive symptoms$_{(\text{Time T-1})}$, and peer stress (**path b_j**: $b = 0.05$, $SE = 0.02$), $t(801) = 2.88$, $p < .01$, partially mediates the relationship between family conflict and depressive symptoms$_{(\text{Time T})}$ (**path c'**: $b = 1.64$, $SE = 0.64$), $t(801) = 2.56$, $p < .01$. When examining the 95% CI (**path a*bj**: b = 0.12, $SE = 0.06$; 0.01, 0.23), the results of the test of the indirect effect suggest that the mediation effect is significant. Last, our findings did not support the role of a negative consequences cognitive style. Not surprisingly, greater family conflict predicted higher levels of depressive symptoms (**path a**: $b = 2.39$, $SE = 0.68$), $t(813) = 3.49$, $p < .001$. However, dependent interpersonal stressors$_{(\text{Time T-1})}$ did not interact with a negative consequence cognitive style to predict higher levels of depressive symptoms$_{(\text{Time T})}$ (**path b_j**: $b = 0.03$, $SE = 0.02$), $t(813) = 1.74$, *ns*. Therefore, this moderated-mediation model is not significant.

Between-Subject Analyses: Family Conflict, Peer Stress, Attributional Style, and Depressive Symptoms

We first examined whether there was any change in depressive symptoms over the course of our study using

a repeated measures ANOVA with negative cognitive style, family conflict, age, and gender as covariates to account for the individual (between-subjects) differences. Results indicated significant linear change in the depressive symptoms over time, $F(3, 131) = 2.728$, $p = .047$. To further understand the mechanisms underlying depressive symptom change, we next examined our proposed mediation models.

We utilized the fixed effects from the mediation and moderated-mediation models described in our within-subject analyses presented earlier to calculate predicted depressive symptoms for individuals with high versus low family conflict and high versus low cognitive vulnerability (i.e., a median split delineated low and high groups). By calculating the predicted estimates for depressive symptoms,[2] analyses account for the impact of peer stressors in the manifestation of depressive symptoms at each time point, and thus, provide a framework to examine the predictive validity of our proposed models. In our first set of analyses, we examined predicted estimates for a model in which peer stress mediates the relationship between family conflict and subsequent depressive symptoms. A two-way ANOVA with time and family conflict as factors suggest that the Time × Family Conflict, $F(2, 133) = 23.01$, $p < .001$, is significant. Figure 3 suggests that adolescents reporting higher family conflict are more likely to report higher levels of depressive symptoms over time. Second, we examined a three-way ANOVA with time, family conflict, and negative self cognitive style as factors. A preliminary analysis indicates a significant Time × Self-Cognitive Style, $F(1, 130) = 9.16$, $p < .01$. Moreover, the Time × Family Conflict × Self-Cognitive Style, $F(1, 130) = 3.84$, $p = .05$, is also significant. Figure 4 indicates that individuals reporting both high family conflict and a depressogenic self-cognitive style report the highest level of depressive symptoms over time. Critically, such symptoms are also at a clinically significant level. Last, to examine the moderating role of the negative cause cognitive style we examined a repeated measures ANOVA with time, family conflict, and negative cause cognitive style as factors. Preliminary two-way analyses indicated a significant Time × Cause Cognitive Style, $F(1, 131) = 19.70$, $p < .001$. Moreover, the thee-way Time × Family Conflict × Cause Cognitive Style, $F(1, 131) = 8.29$, $p < .01$, is also significant. Similar to the previous analyses, Figure 5 suggests that for adolescents reporting high family conflict and a high

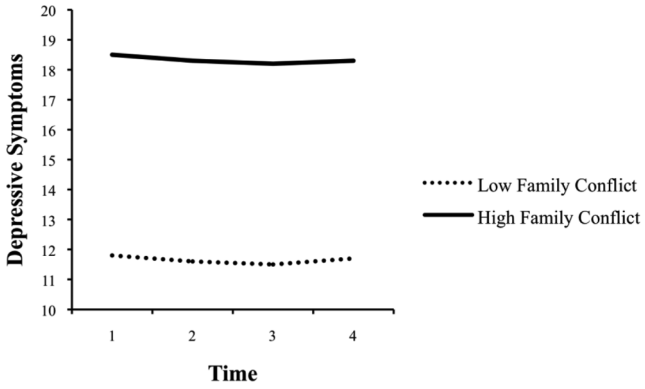

FIGURE 3 Predicted depressive symptoms during follow-up period for adolescents reporting high versus low family conflict. *Note.* CESD ≥ 16 accepted cutoff for clinically significant depressive symptoms.

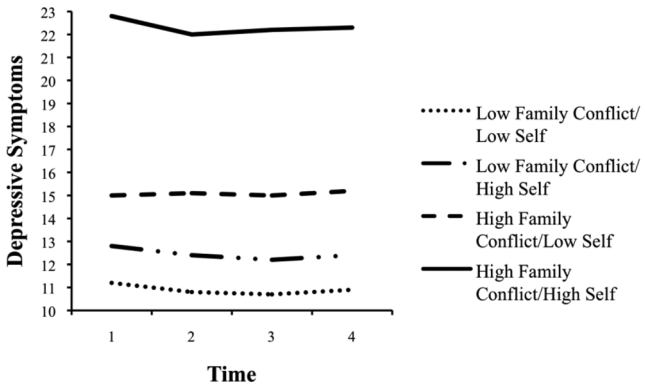

FIGURE 4 An examination of the moderating effect of negative self cognitive style on predicting depressive symptoms over time. *Note.* Higher levels of the depressogenic cognitive style for self denote greater cognitive vulnerability; CESD ≥ 16 accepted cutoff for clinically significant depressive symptoms.

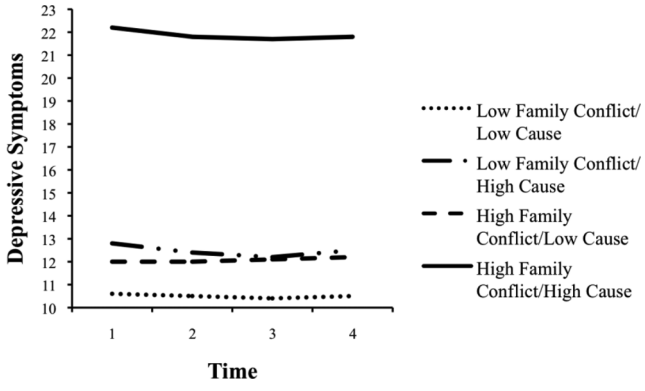

FIGURE 5 An examination of the moderating effect of negative cause cognitive style on predicting depressive symptoms over time. *Note.* Higher levels of the depressogenic cognitive style for cause denote greater cognitive vulnerability; CESD ≥ 16 accepted cutoff for clinically significant depressive symptoms.

[2]There is a robust association between the observed and predicted values of depressive symptoms over time ($r = .86$), suggesting it is appropriate to use predicted values in order to account for the impact of peer stress in predicting changes in depressive symptoms during the course of the study.

depressogenic cause cognitive style, predicted depressive symptoms are a clinically significant level.

DISCUSSION

In line with research examining cognitive-interpersonal integration (e.g., Hankin & Abramson, 2002; Kercher & Rapee, 2009), the study integrated two prominent cognitive and interpersonal theoretical models: the spillover framework (Repetti, 1989) and the hopelessness theory (Abramson et al., 1989). Using a 4.5-month multiwave, longitudinal design and an idiographic, time-lagged data analytic approach, results supported the integration of the cognitive and interpersonal etiological models of depression. Specifically, when examining the spillover framework, results indicated that greater family conflict contributed to greater dependent interpersonal peer stressors and subsequent depressive symptoms (see Figure 3). In addition, when examining the integrated model, results indicated that both negative cognitive style for self and cause moderated the mediational pathway between peer stress and depressive symptoms (see Figures 4 and 5). These findings contribute to a growing body of research, which underscores the importance of examining integrated cognitive-interpersonal models of depression.

Interpersonal Spillover Effect

Seminal research on stress has dramatically reshaped and improved our understanding about the onset, recurrence, and severity of depression (for review, see Hammen, 2006; Hammen & Shih, 2008). More recent studies, particularly in younger samples, have begun to examine the role that underlying interpersonal vulnerability and risk factors play in triggering interpersonal stress (e.g., Auerbach, Bigda-Peyton, Eberhart, Webb, & Ho, 2011; Stocker et al., 1997; Stocker & Youngblade, 1999). Consistent with the spillover framework, we found that greater family conflict triggered greater dependent interpersonal peer stressors. Such stress then contributed to higher levels of depressive symptoms. Moreover, results from the reverse model suggest that the effect is unidirectional in nature in that peer stressors contribute to subsequent depressive symptoms; however, depressive symptoms do not predict higher levels of peer stressors. In addition to underscoring the importance of interpersonal vulnerability and risk factors, the model also highlights the impact of ''interpersonal spillover'' (Flook & Fuligni, 2008; Repetti, 1989). Meaning, these findings suggest that in adolescence, conflict in the family environment confers vulnerability to experience an increased frequency of peer stressors. As a whole, peer stressors seem to be mediating the relationship between family conflict and subsequent depressive symptoms, which further substantiates the need to cultivate supportive and prosocial family environments for the development of healthy adolescents (Mazza et al., 2009).

Integrated Cognitive-Interpersonal Model

By integrating the hopelessness theory and the spillover framework, we sought to better ''why'' depressive symptoms emerge. In the context of the family conflict spillover effect model, results indicated that depressogenic attributional styles for self and cause moderated the mediational pathway between peer stress and depressive symptoms. These findings suggest that a family environment embroiled in conflict and turmoil generates dependent interpersonal peer stress; in addition, individuals who view themselves as flawed or deficient may blame themselves for their turbulent family environment as well as peer relational difficulties, which contributes to higher levels of depressive symptoms. Alternatively, depressogenic attributions regarding the global and stable cause of events contribute to a sense of hopelessness as spillover from the family environment may intrude into various domains of one's life (Flook & Fuligni, 2008). Given the chronicity of such stress as well as one's beliefs about the cause of these negative life events, depressive symptoms emerge. The findings highlight the synergistic relationship between interpersonal and cognitive vulnerability, and in doing so provide a more holistic understanding regarding the etiology of adolescent depression.

Of interest, the moderated-mediation model including negative cognitive style for consequences was not significant. Although these findings are in contrast to our hypothesis, recent research on future orientation among adolescents may help explain these unanticipated results. In general, adolescents exhibit weak orientation for the future, which may be attributed to an undeveloped working memory system (Cauffman, Steinberg, & Piquero, 2005) as well as an immature prefrontal cortex coupled with profound hormonal change (Steinberg, 2008). Relative to older individuals, adolescents are characterized as having less concern about the future as well as an inability to accurately forecast consequences regarding their decisions (Steinberg et al., 2009). Although youthful ''myopic tendencies'' are associated with high-risk behaviors (e.g., precocious sexual behaviors and substance use; Auerbach, Tsai, & Abela, 2010; Steinberg, 2007), it also may inhibit adolescents from perceiving negative events, particularly interpersonal stressors, as having dire and catastrophic long-term consequences. Adolescents' temporal perspective may be developmentally limited, and thus, for many, cultivating catastrophizing thoughts about the future may be ''out

of reach.'' Although these findings should be replicated, it provides tacit support for examining negative cognitive styles separately (as opposed to using composite scores or weakest link approach) as they may differentially interact with relational stressors when contributing to the occurrence of depressive symptoms.

Limitations

Although there are a number of strengths with regards to examining our proposed integrated cognitive-interpersonal model of depression, the study is not without limitations. First, the sample was recruited from a high school in Montreal, Quebec, and participation was based on consent from the student and parent/guardian as well as the logistical constraints imposed by the average school day (e.g., testing, detention, student truancy/absences). All students were encouraged to participate; however, reasons for nonparticipation were not assessed. Given that we did not assess nonparticipation, differences between the samples could not be ascertained and may limit the generalizability of our findings. Second, the current study estimated complex idiographic, time-lagged multilevel models, which allowed for relatively strong inferences regarding the temporal unfolding of stress and depressive symptoms. At the same time, the data analytic approach employed does not determine ''model fit,'' which limits our capacity to directly compare our mediation models. Third, the present study utilized self-report measures that are prone to response biases and have diagnostic limitations. Future research would benefit from utilizing more sophisticated assessment techniques such as peer and parent ratings, semistructured diagnostic interviews, and direct behavioral observation in order to examine both state and trait variables of interest. In addition, the present study examined idiographic symptom fluctuation; however, we did not assess for clinically significant mood or anxiety disorders. At the same time, our findings do suggest that the models predict clinically significant depressive symptoms (see Figures 3, 4, and 5). Therefore, future research should explore whether the integrated cognitive-interpersonal model predicts onset, recurrence, and severity of depression. Fifth, past research examining cognitive vulnerability has found similar results when examining the cause, the consequences, the self, the composite score, and weakest link approach (Gibb et al., 2006). In the current article, results indicated nonsignificant findings for consequences, which may suggest that there is benefit to examining such styles separately. Nevertheless, as there may also be an advantage to examine composite and weakest link models, future research may also consider exploring these approaches when testing integrated models for depression. Last, the homogeneity of ethnic distribution may limit the generalizability of our findings to more diverse samples.

Implications for Research, Policy, and Practice

To date, cognitive-behavioral therapy (CBT) has been the most widely studied nonpharmacologic intervention for the treatment of depression in youth; however, recent meta-analyses have revealed modest effect sizes ($d = 0.53$ in Klein, Jacobs, & Reinecke, 2007). A potential limitation of the CBT approach for depressed adolescents, which may account, in part, for these modest effects, is not adequately addressing interpersonally salient issues. Specifically, Hammen's (1991) research on the stress generation framework began disentangling the complex and reciprocal relationship between interpersonal stress and depressive episodes, and subsequent research has found that interpersonal stressors are more strongly linked to adolescent depression as compared to noninterpersonal stressors (Rudolph, 2009). Given the interplay between cognitive and interpersonal vulnerability factors, the current findings suggest that CBT in adolescents may be enhanced by also targeting salient interpersonal vulnerability factors.

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