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Parenting stress predicts depressive symptoms in adolescents with inflammatory bowel disease

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Abstract

Objectives—Adolescents with inflammatory bowel disease (IBD) are at an increased risk for depression and poor IBD management. Although depression and family factors have been associated in cross-sectional analyses, their casual association over time has not been studied. The primary objective of this study was to assess the temporal association between parenting stress and adolescent depressive symptoms in IBD.

Methods—Along with a primary parent, adolescents [$M_{\text{age}(\text{years})} = 15.5 \pm 1.4$] with a confirmed diagnosis of Crohn's disease or ulcerative colitis and prescribed a treatment regimen of a 5-ASA medication and/or 6-MP/azathioprine were recruited from routine gastroenterology clinic visits at two pediatric medical centers. The current study was nested in a larger IBD self-management trial. Adolescents completed the Children's Depression Inventory. Parents completed a sociodemographic questionnaire and the Pediatric Inventory for Parents. Gastroenterologists provided disease severity assessments on the basis of the clinic appointment corresponding to the study visit or the most recent clinic appointment. Questionnaires were readministered ~ 6 months after the initial visit.

Results—Participants include 93 families at baseline and 65 at follow-up (69.8% retention; $N = 65$). A cross-lagged panel analysis was used to detect causal directionality and change in adolescent depression and parenting stress over 6 months. Baseline parenting stress accounted for a significant amount of the variance in depressive symptoms at follow-up [$R\text{-change} = 0.03$, $F(1,58) = 35.6$, $P < 0.05$].

Conclusion—Parenting stress impacts adolescent depressive symptoms in IBD. Identification of parenting stress and adolescent depression should occur early and possibly in the context of routine clinic visits.

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Keywords

cross-lagged analysis; depression; diathesis-stress model; parent distress

Approximately 71 per 100 000 children and adolescents are diagnosed with inflammatory bowel disease (IBD) per year and experience uncontrolled and unpredictable periods of symptom exacerbation and remission [1]. Adolescents make up 25% of all IBD diagnoses in this pediatric population [2,3]. IBD can be particularly difficult for adolescents to cope with, given the unpredictable nature of symptom occurrence and the potentially embarrassing nature of these symptoms (i.e. diarrhea, poor weight gain, and delayed growth/puberty) [4]. IBD treatment, such as oral corticosteroids and immunomodulators, includes variable dosing schedules and multiple pills per day, which may further increase distress due to unpleasant side effects (e.g. unwanted facial hair growth, acne, weight gain, and mood swings). Such sequelae from IBD can have detrimental effects on self-esteem, functional abilities, and interpersonal relationships.

Youth with IBD experience higher rates of depression and other internalizing disorders when compared with healthy peers and youth with other chronic illnesses [5,6–10]. Adolescents with IBD have a 4.6-times greater odds of endorsing clinically significant levels of anxiety and/or depression than healthy peers [9]. Twenty percent of adolescents with IBD report clinically elevated emotional symptoms [8]. On the Child Depression Inventory [11], a well-validated measure of depressive symptoms in children and adolescents, 15.0–24.5% of adolescents with IBD exceed the clinical cutoff for depression [10,12,13]. Depressive symptoms are a significant concern as patients with IBD who are depressed are less likely to take their medicines [14,15], have a poorer quality of life [16], and are at an increased risk for suicidal behavior [17]. Given these severe and potentially irreversible consequences, it is important to proactively identify adolescents at risk for depression to provide preventive or early intervention services.

Early identification of those at risk for depression is challenging as depression is known to have a multifaceted etiology including, but not limited to, genetic predisposition and biological and environmental factors [18]. According to the diathesis-stress model, individuals with vulnerabilities toward a specific psychological disorder are more likely to experience that disorder when exposed to certain stressors [19]. This model has been examined in several pediatric chronic illness populations to better understand why some, but not all, youth with chronic illness experience depression [20]. Across several illness groups, family factors, such as parenting stress, have been identified as playing an important role in the development of child emotional problems [12,21–23].

Parents play an instrumental role in adolescent IBD management, with recent research indicating that 85% continue to rely on their parents [24], who serve as the primary source of emotional support for adolescents with IBD [25]. When experiencing parenting stress, parents may be less able to provide their adolescents with the instrumental and emotional support necessary to cope with IBD, which could increase the risk for adolescent depressive symptoms. Previous research has documented a link between parenting stress and child depressive symptoms in IBD [12]. However, because of the cross-sectional nature of

previous work, temporal relationships have yet to be determined. The use of illness-related measures of parenting stress has become more common [12,26–32], and these measures have been used to elucidate that parenting stress is common in pediatric IBD [6,33], with stress occurring because of the frequency, as well as the perceived difficulty, of various illness-related tasks. Parents often struggle with making frequent medical decisions, managing the unpredictable nature of IBD symptoms [33], and worry about the impact of IBD on their child [34]. This stress may serve as a catalyst for the development of adolescent depression by unintentionally creating an environment that fosters negative attitudes toward IBD.

The current study advances the IBD literature by applying the diathesis-stress framework to pediatric IBD. Using a cross-lagged panel correlation analysis, we examined the temporal association between parenting stress and adolescent depressive symptoms in pediatric IBD over the course of 6 months. Given the diathesis-stress framework and research in other chronic illness populations, we hypothesized that pediatric parenting stress contributes to the development of elevated depressive symptoms in adolescents with IBD, above and beyond the effects of covariates known to be related to depression or parenting stress (e.g. adolescent age, sex, ethnicity, family income, disease severity). Examining this model in the context of IBD is unique given the unpredictable and intermittent nature of IBD symptoms and management demands.

Methods

Participants

Adolescents aged between 13 and 17 years with a confirmed diagnosis of either Crohn's disease or ulcerative colitis and prescribed a treatment regimen of a 5-ASA medication and/or 6-MP/azathioprine were recruited as part of a larger longitudinal, multisite study examining IBD self-management. Exclusion criteria included a diagnosis of a neurocognitive disorder, a comorbid chronic medical condition, or lack of English fluency in either the adolescent or their parent as documented in the medical chart. Of the 106 eligible adolescents and their parents approached for participation in a larger observational study, 87.7% of families agreed to participate and completed baseline (T_1) assessment ($N = 93$). Reasons for declining participation were blood draw requirement (i.e. part of larger study requirement), not enough time, and/or not interested in research participation. A 69.8% retention rate was obtained 6 months later (T_2 ; total $N = 65$). Baseline sample characteristics are outlined in Table 1. Site demographic differences were not detected and the sample was generally representative of the pediatric IBD population reported in the literature [8,35]. Differences between lost to follow-up and retained participants were not found on baseline measures.

Procedure

All study procedures were approved by the Institutional Review Boards at study sites. Study recruitment occurred at two large pediatric medical centers in the Midwest ($n = 42$) and the Northeast ($n = 51$) USA. Study personnel identified adolescents meeting study inclusion criteria through medical chart review. Eligible families were contacted during regularly

scheduled gastroenterology clinic appointments, during scheduled infliximab infusions, or over the telephone and given a thorough study description. After parents verified inclusion/exclusion criteria, informed consent/assent was obtained from adolescents and their parents at the initial study visit (T₁). Measures were readministered ~ 6 months after the initial visit (T₂), which was in conjunction with their follow-up clinic appointment. Each adolescent's gastro-enterologist provided disease severity assessment data based on the clinic appointment corresponding to the study visit or from the most recent clinic appointment. Participants were modestly reimbursed for their time and effort for study completion.

Measures

Pediatric inventory for parents

The Pediatric Inventory for Parents (PIP) [31] is a 42-item parent-report questionnaire using five-point Likert scaling that measures both the frequency (PIP-F) and the difficulty (PIP-D) of illness-related parenting stress across four factors: communication (e.g. arguing with family members, speaking with child about his/her illness), medical care (e.g. helping my child with medical procedures, handling changes in my child's daily medical routines), role functioning (e.g. being unable to go to work/job, feeling uncertain about disciplining my child), and emotional functioning (e.g. learning upsetting news, feeling numb inside). Higher scores reflect greater parenting stress. This dual approach to measuring parenting stress (i.e. PIP-F and PIP-D) is guided by a transactional model of stress and coping [36], which theorizes that stress is the result of not only the occurrence of a stressor (frequency) but also the cognitive appraisal of that stressor (difficulty). Adequate validity and internal consistency has been demonstrated previously ($\alpha = 0.80\text{--}0.96$) [31] and also confirmed within the current study [33] (Table 1).

Children's depression inventory

The Children's Depression Inventory (CDI) [37] is a well-validated 27-item self-report questionnaire of depressive symptomatology. Using a three-point Likert scale that ranges from 0 (no symptom) to 2 (distinct symptom), the CDI assesses negative mood, interpersonal problems, ineffectiveness, anhedonia, and negative self-esteem. The CDI total raw scores range from 0 to 54, with a clinical cutoff raw score of 13 or higher, which is indicative of clinically elevated depressive symptoms. A high degree of internal consistency and test–retest reliability was demonstrated within the current sample (Table 1).

Pediatric Crohn's disease activity index

The well-validated Pediatric Crohn's Disease Activity Index (PCDAI; [38,39] assesses Crohn's disease activity using both subjective (e.g. pain) and objective criteria (e.g. physical exam), laboratory findings, and growth parameters. Disease activity scores ranged from 0 to 100: less than 10 = inactive, 10–29 = mild, and 30 or higher = moderate/severe. Internal consistency within the current sample was high (Table 1).

Lichtiger colitis activity index

Using both subjective and objective criteria, the Lichtiger Colitis Activity Index (LCAI) [40] assesses eight ulcerative colitis symptoms (score 0–21): daily stool frequency, nocturnal

diarrhea, visible blood in stool, fecal incontinence, abdominal pain/cramping, general well-being, abdominal tenderness, and need for antidiarrheal medication. Higher scores represent more severe disease. LCAI scores of 2 or lower indicate quiescent disease, a score of less than 10 indicates a response to therapy, and a score of 10 or higher indicates active disease and no response to therapy [41]. Internal consistency within the current sample was acceptable (Table 1).

Demographic questionnaire

At the first study visit, parents completed a demographic questionnaire assessing family income, parent age, marital status, education, and number of family members at home.

Data analytic plan

Using zero-order correlations and *t*-tests, demographic (adolescent age, sex, family income) and disease-specific characteristics (diagnosis, medication type, disease severity) were examined as possible covariates for the cross-lagged panel analysis. Given that the majority of the sample had inactive or mildly severe disease, continuous ratings from the PCDAI and LCAI were summed and then combined to reflect an overall disease severity rating [13,33] (Table 1). Covariates were identified if they were significantly associated ($P < 0.05$) with adolescent depression and parenting stress at both T_1 and T_2 [42].

A cross-lagged panel model was used to examine causal directionality and change in adolescent depression and parenting stress over a 6-month time frame using a longitudinal study design. Three statistical assumptions (i.e. instrument reliability, synchronicity in measurement, and stationarity in data) must be met before causal inferences can be substantiated [43]. Internal consistency reliability increases between two assessment points can artificially inflate cross-lagged comparisons [42]. As such, internal consistency α values were calculated to determine stability of instrument reliability at T_1 and T_2 for both depression (Dep) and parenting stress (Stress; Table 1). Autocorrelations (i.e. test-retest reliabilities) were also calculated (Dep₁Dep₂; Stress₁Stress₂) and should be considered acceptable (Table 1). The next phase examined the assumption of synchronicity, which is simultaneous measurement of both variables over time (Dep₁Stress₁; Dep₂Stress₂). *F*-tests were conducted to compare these associations and determine statistically significant differences (Table 2). Finally, the assumption of stationarity, which is the consistency in the strength and direction of synchronous correlations between primary variables across time, was tested [44]. In other words, Dep₁Stress₁ and Dep₂Stress₂ were correlated and *F*-tests were conducted to ascertain whether significant changes in these correlations were observed over time (Table 2). Figure 1 graphically outlines a cross-lagged panel correlation diagram for the statistical assumptions described.

Cross-lagged panel correlations compare the synchronicity between two variables at the same time point (e.g. Dep₁Dep₂) using cross-lagged correlations, which are correlations of a variable (e.g. Dep₁) with another variable at two temporal time points (e.g. Stress₁, Stress₂). Two sources of extraneous variance are present in cross-lagged panel designs [45]. For the present data, variance in Dep₂ could be due to the correlation between Dep₁ and Dep₂ and/or variance in Dep₂, attributed to the correlation between Stress₂ and Dep₂. Given that the PIP

has both frequency (Stress-F) and difficulty (Stress-D) subscales, two *F*-tests were conducted to test the strength and direction of the cross-lagged correlations separately for each PIP subscale (Table 2). If a cross-lagged correlation was statistically significant, hierarchical linear regressions were conducted to determine whether the cross-lagged effect (i.e. determining temporal precedence) upheld after controlling for any significant sociodemographic or medical covariates and extraneous variance (e.g. controlling for Stress₂ to determine whether Stress₁ remains a significant predictor of Dep₂) [45].

Results

Preliminary analyses

Table 1 outlines baseline sociodemographic characteristics, along with descriptive statistics and psychometric properties of the medical and psychological measures. The mean depression scores were within typical range and participants were primarily managing Crohn's disease (CD), which was largely inactive to mildly severe. Parents were primarily mothers (90%). Correlation analyses revealed no significant associations between adolescent age, sex, ethnicity, or family income and parenting stress or depressive symptoms at either time point. Disease severity measured at baseline was significantly associated with baseline depressive symptoms ($r = 0.34$, $P < 0.01$) and parenting stress (difficulty, $r = 0.32$, $P < 0.01$; frequency, $r = 0.24$, $P < 0.05$). These associations were also detected at the 6-month follow-up (T₂), as disease severity was significantly associated with depressive symptoms ($r = 0.31$, $P < 0.01$) and parenting stress frequency ($r = 0.30$, $P < 0.05$), but not difficulty. Adolescents with Crohn's disease were more likely to have elevated depressive symptoms at both baseline ($r = -0.24$, $P < 0.05$) and 6 months later ($r = -0.28$, $P < 0.05$).

Cross-lagged panel analysis

The assumption of reliability was tested using internal consistencies over time and autocorrelations (i.e. test-retest reliabilities) to determine acceptability. Table 1 outlines internal consistency values for both parenting stress and depressive symptoms for both time points along with significant autocorrelations (Fig. 1, paths 1 and 2). Reliability statistics outline adequate internal consistencies and significant autocorrelations.

The assumption of synchronicity was examined to compare the correlations between parenting stress and depressive symptoms at baseline (Fig. 1, path 5) and then the correlations at the 6-month follow-up (Fig. 1, path 6). As outlined in Table 2, the assumption of synchronicity was met for parenting stress frequency (i.e. *F*-test was not significant for parenting stress frequency, but *F*-test was significant for parenting stress difficulty).

The final assumption, stationarity, was tested by comparing the correlation between baseline parenting stress and depressive symptoms 6 months later (Fig. 1, path 3), and baseline depressive symptoms and parenting stress 6 months later (Fig. 1, path 4). These comparisons were tested twice, once for parenting stress frequency and the second time for parenting stress difficulty. As outlined in Table 2, the *F*-test was significant for parenting stress frequency, in that the correlation between baseline parenting stress and depressive symptoms 6 months later (Fig. 1, path 3) was significantly stronger than the correlation

between baseline depressive symptoms and parenting stress 6 months later (Fig. 1, path 4). The F -test was not significant for parenting stress difficulty. Thus, the three statistical assumptions were met for parenting stress frequency, but assumptions of synchronicity and stationarity were not met for parenting stress difficulty.

Regressions were conducted separately to control for synchronous and autocorrelation effects (Table 3). Given that disease severity was significantly correlated with main study variables, it was included as a covariate. For the first regression, which controlled for synchronous effects, the dependent variable was depressive symptoms at follow-up, and subsequent incremental steps included disease severity, parenting stress frequency at follow-up, and baseline parenting stress frequency. Results indicated that baseline parenting stress accounted for a significant amount of the variance in depressive symptoms at follow-up [R -change = 0.08, $F(1,61) = 4.47$, $P < 0.01$]. For the second regression, which controlled for autocorrelation effects, depressive symptoms at follow-up was the dependent variable and subsequent incremental steps included disease severity, baseline depressive symptoms, and parenting stress frequency at baseline. Baseline depressive symptoms were included to control for autocorrelational effects. Results indicated that baseline parenting stress accounted for a significant amount of the variance in depressive symptoms at follow-up [R -change = 0.03, $F(1,58) = 35.6$, $P < 0.05$]. Thus, even after controlling for synchronous and autocorrelational effects, baseline parenting stress remained a significant predictor of depressive symptoms at follow-up.

Discussion

The current study used a cross-lagged panel correlation analysis to examine the longitudinal relationship between illness-specific parenting stress and adolescent depressive symptoms in pediatric IBD. A temporal effect was identified: self-reported parenting stress contributed to elevated adolescent-reported depressive symptoms 6 months later. Multiple factors impact the onset and severity of depressive symptoms in adolescents with IBD, but this is the first study to illustrate that parenting stress directly impacts depressive symptoms in adolescents with IBD. Pediatric parenting stress and its impact on depressive symptoms is critical to consider given the recent data linking adolescent depressive symptoms with compromised disease management and health-related quality of life [14,15]. The diathesis-stress model conceptualizes that an individual may have a genetic predisposition to depression, but environmental or illness-related stressors may exacerbate genetic influences due to chronic illness, increasing the vulnerability to additional stressors (for review see Burke and Elliott [20]). Application of the diathesis stress paradigm in the context of pediatric IBD considers IBD as a specific stressor that increases the patient's vulnerability to additional stressors that can precipitate depressive symptoms. This model has particular relevance to pediatric IBD given its unpredictable, fluctuating course, associated debilitating symptoms, and the increased risk for depressive symptoms among adolescents [4,9,46]. IBD is often not a constant stressor, but peaks and valleys of symptom recurrence may lead patients to experience a perpetuated level of elevated stress. Such factors impact both the adolescent and their parent(s).

Parents of youths with IBD balance typical familial responsibilities (i.e. employment, finances) with disease-related tasks, which can cause elevated parental stress. Multiple factors can influence the occurrence of stressful events, particularly when IBD is active and causing functional impairments. Our data show that the frequency of medical events had a more direct link to elevated adolescent depressive symptoms than their perceived difficulty. In essence, if the stressful events were more chronic, they tended to have a greater negative effect on adolescent mood. One can speculate that stressful events can include frequent medical visits, hospitalizations, increased treatment costs, time away from work, increased medical decision making (i.e. changing medications to better address symptoms), and coping with an adolescent who is experiencing significant physical symptoms (i.e. bloody stool, diarrhea, pain), disruptions to their daily functioning (i.e. missing school, avoiding social activities), and increased irritability. When parenting stressors are difficult, but intermittent, their subsequent impact on adolescent functioning may be minimized.

It is likely that the association between parental stress and youth depressive symptoms is an interactive process in which parenting stress can be exacerbated by increased depressive symptoms in adolescents. However, the current data explicitly indicate that parenting stress predicts adolescent depressive symptoms 6 months later. Of critical importance is the ability to detect symptoms of parenting stress and adolescent depressive symptoms in a proactive manner to ensure that symptoms do not detrimentally impact patient well-being.

Although the prospective study design and independent self-reports of functioning by both the parent and the adolescent are notable study strengths, study limitations are important and have consequent directions for future research. The mean scores for depression were used in analyses and, as a group, the mean depression scores were not clinically elevated. Future studies with larger samples to better detect effect size may want to consider studying these relationships with only the subsample of youth at risk for depression or with clinically elevated depressive symptoms, and whether participants received depression treatment through administration of psychotropic medication or delivery of psychological care. In addition, specific aspects of parenting stress could be examined to better understand what constructs of parenting stress are most compromised (i.e. medical care, communication) during periods of active disease. For example, when IBD is inactive, the emotional distress domain could be most concerning given that subclinical symptoms and medication side effects can impact day-to-day family interactions (e.g. an adolescent experiencing facial bloating secondary to medication and consequently experiencing peer teasing and school avoidance). In addition, the use of mean scores as main study variables may compromise the ability to detect individualized differences, which are critical to consider for intervention. Classifying symptoms by disease status into active versus inactive, or by disease group, could provide insight into the impact of parenting stress on adolescent depressive symptoms. For example, the cumulative mean score to capture IBD severity may minimize variation in symptom severity, which may have important implications in IBD management.

This is the first study conducted on adolescents with IBD that highlights the family context in IBD by examining the temporal relationship between parenting stress and adolescent self-reported depressive symptoms. Clinicians have an opportunity to systemically screen for parenting stress and patient depressive symptoms during routine medical clinic visits. For

example, during clinic appointments, parents could complete the PIP and patients could complete the CDI. Both are brief, self-report measures that can be administered, scored, and reviewed with the family to initiate discussion if elevations are identified. However, these measures should be supplemented with a clinical interview, as recent data suggest that CDI scores may be inflated because of physical illness symptoms related to IBD [47]. Early identification of symptoms at the parent and/or patient level that negatively impact IBD management is critical given that psychosocial impairment (i.e. depressive symptoms) and compromised quality of life have been documented to increase healthcare utilization [48,49]. Clinicians have the opportunity to proactively make referrals to psychologists to address psychological symptoms detrimentally affecting patients or offer parent-based support for positive strategies to manage parenting stress. This in turn may improve the quality of life of both parents and youth, along with decreasing the costs associated with IBD management.

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Fig. 1.

Cross-lagged panel correlation diagram for adolescent depression (Dep) and parenting stress (Stress) at time 1 and time 2. Correlation values for parenting stress frequency are included (r_t).

Table 1
Descriptive statistics and psychometric properties of sociodemographic, medical, and psychological measures

	<i>n</i> (%)	Mean $\pm$ SD	Internal consistency	Test–retest reliability
Adolescent				
Age (years)		15.5 $\pm$ 1.4		
Female	40 (43.0)			
Caucasian (non-Hispanic)	77 (91.7)			
Primary parent				
Age (years)		45.7 $\pm$ 5.6		
Female	84 (90.3)			
Married/two-parent	74 (88.1)			
Annual income		126 880 $\pm$ 65 351		
Education level (at least a college degree)	30 (58.3)			
Diagnosis				
Crohn's disease	74 (79.6)			
Ulcerative colitis	10 (20.4)			
PCDAI				
Inactive	36 (48.6)		0.95	
Mild	35 (47.3)		0.95	
Moderate/severe	3 (4.1)			
LCAI				
Quiescent	12 (63.2)		0.75	
Response to therapy	6 (31.6)		0.73	
No response to therapy	1 (5.3)			
IBD total severity score		8.6 $\pm$ 8.7		
Medication treatment				
6-MP/azathioprine	18 (19.8)			
5-ASA	19 (20.9)			
6-MP/azathioprine+5-ASA	54 (59.3)			
Depression (Dep)				
Total Dep raw score (T ₁)		5.8 $\pm$ 6.3	0.90	0.79***
Total Dep raw score (T ₂)		4.9 $\pm$ 5.7	0.89	
Parenting Stress (Stress)				
Total Stress, <i>F</i> -score (T ₁)		85.8 $\pm$ 26.2	0.96	0.57***
Total Stress, <i>F</i> -score (T ₂)		80.7 $\pm$ 25.4	0.96	
Total Stress, <i>D</i> -score (T ₁)		78.5 $\pm$ 24.5	0.95	0.54***
Total Stress, <i>D</i> -score (T ₂)		77.5 $\pm$ 26.9	0.96	

IBD, inflammatory bowel disease; LCAI, Lichtiger Colitis Activity Index; PCDAI, Pediatric Crohn's Disease Activity Index.

*** *P* 0.001.

Table 2
Comparisons of synchronous correlations and cross-lagged correlations between parenting stress and adolescent depression

Parenting stress	Synchronous correlations			Cross-lagged correlations		
	<i>r</i> Dep ₁ Stress ₁	<i>r</i> Dep ₂ Stress ₂	<i>F</i>	<i>r</i> Stress ₁ Dep ₂	<i>r</i> Dep ₁ Stress ₂	<i>F</i>
Frequency	0.33**	0.25*	3.33	0.41**	0.23	11.10***
Difficulty	0.43**	0.32**	6.48*	0.34**	0.30*	2.18

Dep, depression; Stress, parenting stress.

* *P* 0.05.

** *P* 0.01.

*** *P* 0.001.

Table 3
Summary of hierarchical multiple regression analyses

Dependent variables and predictors	R^2	R^2	F -change	β
Synchronous effects				
Adolescent depression (T_2)				
Disease severity (T_1)	0.06	0.06	3.83	0.11
Parenting stress frequency (T_2)	0.10	0.04	2.86	0.04
Parenting stress frequency (T_1)	0.18	0.08	6.06	0.35*
Autocorrelational effects				
Adolescent depression (T_2)				
Disease severity (T_1)	0.06	0.06	3.67	-0.06
Adolescent depression (T_1)	0.62	0.57	88.80	0.74***
Parenting stress frequency (T_1)	0.65	0.02	3.98	0.18*

* $P < 0.05$.

*** $P < 0.001$.