

A Growth Curve Analysis of the Course of Dysthymic Disorder: The Effects of Chronic Stress and Moderation by Adverse Parent–Child Relationships and Family History

Lea R. Dougherty, Daniel N. Klein, and Joanne Davila
State University of New York at Stony Brook

Using mixed effects models, the authors examined the effects of chronic stress, adverse parent–child relationships, and family history on the 7.5-year course of dysthymic disorder. Participants included 97 outpatients with early-onset dysthymia who were assessed with semistructured interviews at baseline and 3 additional times at 30-month intervals for 7.5 years. Results indicated that higher levels of chronic stress 6 months prior to each follow-up predicted greater depression severity at follow-up, controlling for depression severity at the start of the chronic stress assessment. In addition, adverse parent–child relationships and family history of dysthymic disorder moderated this association. For patients with poorer parent–child relationships, chronic stress was associated with increased depression severity at follow-up, whereas patients with a higher familial loading for dysthymic disorder were less responsive to chronic stress over time.

Dysthymic disorder, a chronic, low-grade depression of at least 2 years in duration, affects a considerable number of individuals, with prevalence rates of approximately 6.0% in a nationally representative sample (Kessler et al., 1994) and 22.0% in outpatient mental health settings (Klein, Dickstein, Taylor, & Harding, 1989). Major depressive episodes are often superimposed on the mild chronic depression (referred to as double depression; Keller & Shapiro, 1982), which heightens the functional impairment of individuals with dysthymic disorder (Leader & Klein, 1996). Even though dysthymic disorder is typically characterized by milder symptomatology than episodic major depressive disorder, over time individuals with dysthymia experience greater cumulative symptoms and have more suicide attempts, hospitalizations, and social impairment than individuals with episodic major depression (Klein, Schwartz, Rose, & Leader, 2000). The course of dysthymic disorder tends to be chronic, with periodic exacerbations of double depression. A naturalistic follow-up study indicated that only 52.9% of patients with dysthymic disorder recovered within 5 years of prospective observation (Klein et al., 2000). Thus, it is important to understand what factors maintain the chronicity of dysthymic disorder–double depression over time.

Environmental adversities of various kinds are often presumed to play an important role in the course of depression (Mazure, 1998; Riso, Miyatake, & Thase, 2002). Research has demonstrated a consistent relationship between life stress and depression onset and recurrence (Kessler, 1997; Mazure, 1998; Monroe & Hadjiyannakis, 2002). However, the studies on life stress in the course of depression have generally focused on acute events and onset–

recurrence. In contrast, chronic stressors appear more likely to play a role in the maintenance of chronic conditions (Brown, Bifulco, Harris, & Bridge, 1986; Depue & Monroe, 1986; Moerk & Klein, 2000). For example, patients with dysthymia have reported greater chronic strain (Klein, Taylor, Dickstein, & Harding, 1988; Ravindran, Griffiths, Waddell, & Anisman, 1995) and more interpersonal difficulties (Leader & Klein, 1996) than individuals with episodic major depression. In addition, in a longitudinal study of the course of dysthymic disorder, we found that chronic stress was one of the strongest predictors of both failure to recover and depressive symptoms at 5-year follow-up (Hayden & Klein, 2001). Nevertheless, to our knowledge, the interplay between chronic stress and depressive symptomatology over time has not been examined. Thus, the first aim of this study was to investigate whether chronic stress is involved in the maintenance of depressive symptomatology in individuals with dysthymia.

The second aim was to investigate factors that might moderate the association between chronic stress and depression over time. We chose to focus on two distal variables, adverse parent–child relationships and family history of dysthymic disorder, as both distinguish dysthymia–double depression from episodic major depression and predict a more chronic course among patients with dysthymia–double depression. In addition, childhood adversity and family history of depression have been shown to moderate the effects of stress on the onset of depression.

Patients with dysthymia have reported more adverse parent–child relationships than patients with episodic major depression and normal controls (Lizardi et al., 1995), and adverse parent–child relationships have had greater predictive utility for the 2.5-year course of dysthymia than demographic or clinical variables (Durbin, Klein, & Schwartz, 2000). Similarly, recent studies have demonstrated higher rates of chronic depression (Klein, Clark, Dansky, & Margolis, 1988) and specifically higher rates of dysthymic disorder in the relatives of probands with dysthymia compared with the relatives of probands with episodic major depression (Klein et al., 1995). Moreover, familial loading for dysthymic disorder, but not for other forms of familial psychopathology,

Lea R. Dougherty, Daniel N. Klein, and Joanne Davila, Department of Psychology, State University of New York at Stony Brook.

This research was supported by National Institute of Mental Health Grant RO1-MH45757. We thank Suzanne Rose for help with interviewing.

Correspondence concerning this article should be addressed to Daniel N. Klein, Department of Psychology, State University of New York at Stony Brook, Stony Brook, NY 11794-2500. E-mail: dklein@notes.cc.sunysb.edu

predicted a poorer course after 5 years in outpatients with dysthymia (Hayden & Klein, 2001).

Research has also provided preliminary evidence that childhood adversity and family history moderate the association between stress and the onset of depressive episodes. In a 2-year follow-up of a nonclinical sample of young women, childhood adversity moderated the relation between stress and depression onset (Hammen, Henry, & Daley, 2000). Under low-stress conditions, women with childhood adversity were significantly more likely to become depressed than women without childhood adversity, whereas both groups had an increased likelihood of depression under high-stress conditions. However, in a large community sample of female twins, women at high genetic risk for major depression more frequently experienced depressive episodes without major environmental stressors than women at low genetic risk (Kendler, Thornton, & Gardner, 2001). In contrast, women at low genetic risk for major depression were more likely to experience a depressive episode in the presence of major environmental stressors, particularly early in their course of illness, than women at high genetic risk.

In light of these findings, we expected that both adverse parent-child relationships and family history of dysthymic disorder would moderate the relation between chronic stress and depression over time in dysthymic disorder. However, we hypothesized that the form of moderation would differ. We predicted that adverse parent-child relationships would sensitize individuals with dysthymia to stress, in the sense that persons with a history of adversity would exhibit greater stress reactivity or responsivity. In contrast, we hypothesized that a familial loading for dysthymic disorder would be a marker of different etiological and maintenance processes that would be more consistent with a stable biological and temperamental predisposition, such that individuals with a greater familial loading for dysthymia would be less responsive to stress.

To address these issues, we used hierarchical linear modeling (HLM; Bryk & Raudenbush, 1992), which is a specific type of mixed effects model (also referred to as multilevel random coefficient models, mixed linear models, multilevel linear models, and general variance component models), to examine longitudinal change. Mixed effects models have several advantages for studying longitudinal change. First, they examine within-individual change by examining each individual's trajectory over time. Second, mixed effects models provide estimates of the relationships between constructs at the lower level (within persons) and constructs at the upper level (between persons) simultaneously. Third, they provide a means to test whether individual-level variables moderate associations between two within-person variables over time. Finally, mixed effects models are able to handle irregularities in data collection, such as nonsynchronous data with an unequal number of observations for each participant, variable spacing of observations over participants, and missing data for some participants.

The goals of this article were to (a) determine the effects of chronic stress on the 7.5-year course of dysthymic disorder and (b) test the possible moderating roles of adverse parent-child relationships and family history of dysthymic disorder on the longitudinal relation between chronic stress and depression.

Method

Participants

The sample for this report comes from a larger family and follow-up study of early-onset (before 21 years) dysthymic disorder, which is de-

scribed in detail elsewhere (Klein et al., 2000). Participants were 97 outpatients with a *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; *DSM-III-R*; American Psychiatric Association, 1987) diagnosis of dysthymic disorder, primary and early-onset types. At entry into the study, 57.7% were experiencing a superimposed major depressive episode, and 77.9% had a lifetime history of major depressive disorder. Participants were between the ages of 18 and 60 years, spoke English, and had knowledge of at least one first-degree relative. Most patients were recruited from the Outpatient Psychiatry Department and the Psychological Center at the State University of New York at Stony Brook. We obtained detailed information about treatment during follow-up from patients and medical records. However, treatment was not controlled, as this is a naturalistic study. In an earlier article, we reported that treatment was not a significant predictor of the course or outcome of dysthymic disorder in this sample (Klein et al., 2000). After giving a complete description of the study, we obtained written informed consent from all patients.

We attempted to conduct follow-up evaluations at 30, 60, and 90 months after the baseline assessment. At least one follow-up was completed for 86 patients (88.7% of the sample), at least two follow-ups were completed for 77 patients (79.4% of the sample), and all three follow-ups were completed for 65 patients (67.0% of the sample).

Family history information was obtained from patients for all first-degree relatives older than 14 years ($N = 446$). Direct interviews were conducted with 40% of these relatives (70% of the living relatives we had permission to contact). When we were unable to interview a family member, we attempted to obtain information about this relative from at least one additional informant.

Baseline Evaluation

The baseline evaluation included assessment with the Structured Clinical Interview for *DSM-III-R* (SCID; Spitzer, Williams, Gibbon, & First, 1990) and the 24-item modified Hamilton Depression Rating Scale (HAM-D; Miller, Bishop, Norman, & Maddever, 1985), which focused on the worst week of the patient's current major depressive episode or the worst week in the past month if the patient was not currently experiencing a major depressive episode. Assessments were conducted by doctoral- and master's-level clinicians and advanced graduate students in clinical psychology. The interrater reliability of the baseline SCID was assessed with both paired-rater and test-retest designs. Kappas for current dysthymia were .90 and .61 with the paired-rater and test-retest designs, respectively. The intraclass correlation (ICC) for interrater reliability of the HAM-D was .95.

Adverse Parent-Child Relationships

The Early Home Environment Interview (Lizardi et al., 1995) was also administered at baseline. It is a semistructured interview that assesses five aspects of the early home environment before the age of 15 years: separation-loss, neglect, physical abuse, sexual abuse, and the quality of the relationship with each parent. Only the parental relationship quality scales were used in this study because of their wider range. The scales consist of the sum of six items rated by the interviewer: rarely spent time or engaged in activities with parent, lack of parental supervision, rarely confided in parent, constantly criticized by parent, often rejected by parent, and rarely felt loved by parent. Scores range from 0 to 6, with higher scores indicating poorer relationships. Coefficient alphas for the quality of the maternal and paternal relationship scales were .79 and .76, respectively. Interrater reliability was assessed by conducting two independent interviews with 50 patients. ICCs for the quality of the maternal and paternal relationship scales were .82 and .69, respectively. The maternal and paternal relationship quality scales were averaged to create one measure of adverse parent-child relationships.

Family History of Dysthymic Disorder

Direct interviews were conducted with the relatives using the SCID—Nonpatient Version (Spitzer et al., 1990). Family history data were collected from probands and informants using an expanded version of the Family History Research Diagnostic Criteria Interview (Andreasen, Endicott, Spitzer, & Winokur, 1977). Lifetime best estimate diagnoses were derived for each relative with all available information (Klein, Ouimette, Kelly, Ferro, & Riso, 1994).

As described elsewhere (Durbin et al., 2000), family history scores were estimated with mixed effects models to adjust for differences among families in the number of relatives, gender of relatives, and type of diagnostic data available. A two-level model was used, which treated relatives as repeated observations (Level 1) nested within families (Level 2), with gender and type of diagnostic data treated as covariates. For each proband, a standardized family history score was derived for dysthymic disorder that described each family's deviation from the overall intercept after adjusting for gender and interview status. Standardized family history scores ranged from $-.03$ to $.19$, with higher scores indicating a greater familial loading for dysthymic disorder adjusting for the covariates.

The interrater reliability of the SCID and best-estimate diagnoses was generally good to excellent (Klein et al., 1994, 1995). The interrater reliability of our best estimate diagnosis of dysthymia, as indexed by kappa, was $.75$ (Klein et al., 1994).

Follow-Up Evaluations

Follow-up assessments included an abbreviated version of the Interview for Recent Life Events (IRLE; Paykel, 1997), the Longitudinal Interval Follow-up Evaluation (LIFE; Keller et al., 1987), and the HAM-D.

Chronic stress. We assessed life stress for each month of the follow-up with Paykel's (1997) IRLE and used a life calendar approach in which significant dates and events were used to facilitate recall (Caspi et al., 1996). The IRLE was administered retrospectively at each 30-month follow-up but was not used at baseline. The IRLE is an investigator-rated measure that assesses the date and objective negative impact of a list of 63 specific life events. To make our lengthy assessment battery more manageable, we reduced the list of specific events to 20 by combining similar events into single items and eliminating minor events that were less likely to be remembered over long recall periods (Moerk & Klein, 2000). Events were rated as either acute or chronic, with events occurring within a discreet time period deemed acute and events lasting 6 or more months classified as chronic. For this report, we focused only on chronic stressors (e.g., ongoing interpersonal difficulties, financial hardship, and chronic illnesses). Each identified chronic stressor was then rated by the interviewer for objective negative impact, which was a contextual rating of the impact that the event would have for most people given the participant's life circumstances. Ratings for each chronic stressor were made on a 6-point scale based on the *DSM-III-R* Axis IV scale for severity of stressors and summed to yield a total score for each month of the follow-up. For the present analyses, we examined only the chronic stress ratings during the 6 months prior to each follow-up assessment to minimize errors in recall and dating. The monthly stress ratings within each 6-month period were averaged to yield the mean chronic stress score prior to each follow-up. We computed mean values because some chronic stressors that lasted more than 6 months may have ended prior to the 6 months examined in our analysis. Thus, the overall rating before each follow-up was weighted by the amount of time in the prior 6 months that the stressors were present. The ICC for interrater reliability of chronic stress ratings was $.56$ (Hayden & Klein, 2001).

LIFE. The LIFE is a semistructured interview used to assess the longitudinal course of Axis I disorders and treatment throughout the follow-up period. Although the LIFE was originally developed for follow-up periods of 6 months, its authors have adapted it for follow-ups of any length, and it has been used successfully for follow-ups of up to 12-year intervals (Surtees & Barkley, 1994). The LIFE uses psychiatric

status ratings (PSRs), which consist of ordinal symptom-based scales reflecting levels of symptoms of *DSM-III-R* major depressive disorder and dysthymic disorder. PSR data were collected retrospectively at each 30-month follow-up. The PSRs are based on a 6-point rating scale for major depressive disorder and on a 3-point rating scale for dysthymic disorder. Both scales were combined to form a 4-point rating scale for this study: 4 = meets definite criteria for major depressive disorder; 3 = meets criteria for dysthymic disorder but not major depressive disorder; 2 = has sub-threshold major depressive disorder or subthreshold dysthymic disorder; and 1 = has minimal or no depressive symptomatology. Good interrater reliability of our LIFE ratings has been reported elsewhere (Klein et al., 1998, 2000). For the present study, only the PSR data for the 6th month prior to each 30-month assessment were used to control for depression at the start of each chronic stress assessment.

Depression severity. The HAM-D was administered at each 30-month assessment. To assess the interrater reliability of the follow-up evaluations, a different rater independently re-interviewed a random sample of 12 patients with the HAM-D within 3 days of the index evaluation. The ICC for the HAM-D was $.96$.

Follow-up assessments were conducted by doctoral- and master's-level clinicians and advanced graduate students in clinical psychology with prior experience in diagnostic interviewing. Patients were generally interviewed by different raters at each evaluation, and interviewers were unaware of the results of previous assessments.

Data analysis strategy. The analyses examined within-subjects variation of depression over 7.5 years and predictors of the course of depression using mixed effects models (Bryk & Raudenbush, 1992). Mixed effects models allow an assessment of individual-level change (Level 1) and prediction of individual-level differences in change (Level 2), if they exist. This approach for analyzing longitudinal change has been described in detail elsewhere and is not presented here (see Nezlek, 2001; Singer & Willet, 2003). For the present purposes, the multilevel approach to longitudinal data allows for the analysis of covariation over time between repeated measures of multiple variables (depression severity and chronic stress). For all analyses, HAM-D depression severity was treated as the dependent variable. First, baseline trajectory models were estimated to examine any systematic change over time. Linear and quadratic growth models were used to examine a within-subjects regression of an individual's depression score onto the time of each assessment. Second, to examine the temporal association between depression severity and chronic stress, we predicted HAM-D depression severity from chronic stress during the 6 months prior to each assessment and controlled for PSR depression at the start of each 6-month chronic-stress assessment. Unfortunately, the HAM-D was only administered every 30 months, so we were forced to use retrospective measures of PSR depression severity to control for initial level of depression at each 6-month period of chronic stress.

Following this analysis, the between-subjects Level-2 independent variables were examined as predictors of the course of depression. In this analysis, within-subjects intercepts and slopes were treated as outcomes to be predicted by the between-subjects variables measured at baseline. As such, change in HAM-D depression severity was predicted from two Level-2 variables: adverse parent-child relationships and familial loading for dysthymic disorder.

Next, we examined whether adverse parent-child relationships and/or familial loading for dysthymic disorder moderated the relation between chronic stress and depression over time. In this analysis, change in depression severity was predicted from chronic stress over time, controlling for prior depression, and the two Level-2 predictors were then examined as predictors of the longitudinal relation between chronic stress and depression.

It should be noted that the residual error terms for the individual-level predictors were restricted when the random error term associated with a coefficient was not significant or in order for the model to converge. The effects of time, where appropriate, and chronic stress were specified as random, whereas the effects of prior depression were specified as fixed in order for the model to converge. When a parameter is fixed, neither the error

variances for that term nor the covariances between the error variances for the term and other error variances are estimated. Mixed effects models allow all available Level-1 data to contribute to the estimation of trajectories for each participant. We used a pairwise missing data procedure to handle any missing data at Level 1. Level 2 did not contain any missing data. Finally, all variables were grand mean centered except time, which was uncentered.

Results

The sample included 79 White (91.9%), 2 African American (2.3%), and 5 Hispanic (5.8%) patients. Of the participants, 75.6 % were women ($n = 65$), and 24.4% were men ($n = 21$). Their mean age was 32.1 years ($SD = 9.7$) at baseline. Marital status at baseline was as follows: 46.5% ($n = 40$) had never married, 31.4% ($n = 27$) were married, 19.8% ($n = 17$) were separated or divorced, and 2.3% ($n = 2$) were widowed. Their mean level of education at baseline was 13.2 years ($SD = 2.2$). The patients were moderately depressed at baseline, with a mean 24-item modified HAM-D score of 25.7 ($SD = 10.6$). The means and standard deviations for all independent and dependent variables are presented in Table 1. We also examined whether any baseline characteristics of our sample predicted attrition at each follow-up. We found that none of the baseline variables distinguished patients who had missing data on HAM-D depression and chronic stress at any of the follow-up assessments.

Course of Dysthymic Disorder Over 7.5 Years

To investigate whether there was any systematic change over time, we estimated baseline trajectory models. Both a model of linear change and a model of quadratic change were examined. These models can be understood as a within-subjects regression of an individual’s depression score onto the time of each assessment. To evaluate these models, we specified the following function to describe the data from each individual:

$$\text{Level 1: } Y_{ij} = \beta_{0j} + \beta_{1j}(\text{time}) + \beta_{2j}(\text{time}^2) + r_{ij}$$

Table 1
Means and Standard Deviations of All Variables

Variable	M	SD	n
Hamilton Depression Scale score			
Baseline	25.72	10.57	97
30 months	18.56	10.44	75
60 months	19.30	12.10	77
90 months	19.12	11.64	76
Chronic stress score ^a			
24–30 months	22.59	19.74	86
54–60 months	22.74	20.51	80
84–90 months	22.13	16.16	79
PSRs of depression ^a			
24 months	2.67	1.14	84
54 months	2.66	1.15	77
84 months	2.53	1.14	77
Adverse parent–child relationships	2.18	1.47	97
Familial loading for dysthymic disorder	0.01	0.05	97

Note. PSRs = psychiatric status ratings of depression from the Longitudinal Interval Follow-Up Evaluation.

^a Retrospective monthly ratings of chronic stress and PSR depression were not collected for the 6 months before baseline.

Level 2:

$$\text{Intercept: } \beta_{0j} = \gamma_{00} + u_{0j}$$

$$\text{Slope: } \beta_{1j} = \gamma_{10} + u_{1j}$$

$$\text{Curvature: } \beta_{2j} = \gamma_{20} + u_{2j} \tag{1}$$

In this equation, Y_{ij} is depression severity of individual j at time i ; β_{0j} is the depression severity of individual j at Time 0 (i.e., the baseline depression rating score of individual j); β_{1j} is the rate of the linear change in depression for individual j over 7.5 years; β_{2j} is the rate of the curvature in depression over 7.5 years; and r_{ij} is the residual variance in repeated measurements for individual j , which is assumed to be independent and normally distributed across participants. There was a significant linear decrease in the level of depression over 7.5 years (unstandardized coefficient = -5.39), $t(96) = -4.80$, $p < .001$. In addition, there was a significant quadratic effect (unstandardized coefficient = 1.34), $t(96) = 4.40$, $p < .001$, indicating that the decrease in depression was greater in the first 2.5 years of follow-up than at subsequent assessments. This asymptotic function reflects the fact that at baseline, our sample was seeking treatment and, hence, was at a peak in symptom severity, whereas all subsequent assessments were conducted at a standard time that was not based on the patients’ clinical state. The leveling off in depression severity at 2.5 years suggests that patients with dysthymia tend to seek treatment during exacerbations of symptomatology and return to a low-grade depression over time. The random error term associated with the quadratic effect was not significant (variance component = 1.78), $\chi^2(96, N = 97) = 89.35$, $p > .50$, which indicates that the random error term cannot be estimated reliably for this effect. However, the random error term associated with the linear component was significant (variance component = 44.73), $\chi^2(96, N = 97) = 124.60$, $p = .026$, suggesting that it might be useful to model between-persons predictors of the linear growth function. Therefore, the residual error term for the quadratic effect was restricted for all further analyses.¹

Chronic Stress Predicting Course of Depression

Building on the growth model, we added chronic stress 6 months prior to each 30-month assessment of HAM-D depression and controlled for PSR depression at the start of each assessment of chronic stress. The equation below describes the model predicting the course of depression from chronic stress. Note that the error term for PSR prior depression was fixed in order for the model to converge:

$$\text{Level 1: } Y_{ij}(\text{depression}) = \beta_{0j} + \beta_{1j}(\text{time}) + \beta_{2j}(\text{time}^2) + \beta_{3j}(\text{chronic stress}) + \beta_{4j}(\text{PSR prior depression}) + r_{ij}$$

¹ Because the chi-square test of significance for the quadratic effect was not significant, we restricted the residual error terms for the quadratic effect. However, it is also possible to allow the errors to vary freely without specifying Level-2 predictors for that term. We tested this method, and the results are consistent with, but somewhat stronger than, what is reported in the article. Nevertheless, we opted to examine the model fixing the error term for the quadratic effect because a model that does not include the Level-2 predictors in the quadratic equation but does include the Level-2 predictors in the other equations assumes that the Level-2 predictors and the quadratic effect are unrelated, which may not be the case (Nezlek, 2001).

Level 2:

$$\begin{aligned} \text{Intercept: } \beta_{0j} &= \gamma_{00} + u_{0j} \\ \text{Time (slope): } \beta_{1j} &= \gamma_{10} + u_{1j} \\ \text{Time}^2 \text{ (curvature): } \beta_{2j} &= \gamma_{20} \\ \text{Chronic stress: } \beta_{3j} &= \gamma_{30} + u_{3j} \\ \text{PSR prior depression: } \beta_{4j} &= \gamma_{40} \end{aligned} \quad (2)$$

In this equation, Y_{ij} is the level of depression of individual j at time i ; β_{0j} is the depression severity of individual j at Time 0 (i.e., baseline level of depression of individual j); β_{1j} is the rate of linear change in depression for individual j ; β_{2j} is the rate of the curvature in depression for individual j ; β_{3j} is the amount of chronic stress of individual j at time i (6 months prior to each 30-month follow-up); β_{4j} is the depression rating of individual j at time i (time of chronic-stress assessment); and r_{ij} is the residual variance in repeated measurements for individual j . Results are shown in Table 2 and indicate that while controlling for prior depression, chronic stress in the 6 months prior to each follow-up predicts depression at follow-up (unstandardized coefficient = 0.51), $t(84) = 2.78, p = .006$. The effects size (ES) for the longitudinal relationship between chronic stress and depression severity is .29.² As can be seen, a greater level of chronic stress was significantly associated with a higher level of depression.³

Adverse Parent–Child Relationships and Familial Loading for Dysthymic Disorder Predicting the Course of Depression

Preliminary to the moderation analyses, we examined the main effects of adverse parent–child relationships and familial loading for dysthymic disorder on the trajectory of change in depression over time. As both Level-2 variables were moderately correlated ($r = .40, p < .001$), we entered them simultaneously to control for the effects of one variable on the other in all analyses. This analysis was conducted with the growth model as the Level-1 equation and both adverse parent–child relationships and familial loading for dysthymic disorder as Level-2 predictors of the depression intercepts and slopes:

$$\text{Level 1: } Y_{ij} = \beta_{0j} + \beta_{1j}(\text{time}) + \beta_{2j}(\text{time}^2) + r_{ij}$$

Level 2:

$$\begin{aligned} \text{Intercept: } \beta_{0j} &= \gamma_{00} + \gamma_{01}(\text{parent–child relationship}) \\ &+ \gamma_{02}(\text{familial dysthymic disorder}) + u_{0j} \\ \text{Time (slope): } \beta_{1j} &= \gamma_{10} + \gamma_{11}(\text{parent–child relationship}) \\ &+ \gamma_{12}(\text{familial dysthymic disorder}) + u_{1j} \\ \text{Time}^2 \text{ (curvature): } \beta_{2j} &= \gamma_{20} \\ &+ \gamma_{21}(\text{parent–child relationship}) \\ &+ \gamma_{22}(\text{familial dysthymic disorder}) \end{aligned} \quad (3)$$

Mean adverse parent–child relationship scores at baseline were significantly associated with the trajectory of change in depression (unstandardized coefficient = 1.46), $t(94) = 1.93, p = .05$ (ES = .20) but were

not significantly associated with the intercept. Familial loading for dysthymic disorder did not independently predict the intercept or the slope of depression over time (see Table 2). In this case, patients with higher mean adverse parent–child relationship scores exhibited less of a reduction in depression over time, suggesting that patients with dysthymia with poorer parent–child relationships are likely to have a more severe course of depression than other patients with dysthymia.

Moderation Analysis

As reported above, chronic stress 6 months prior to each 30-month assessment predicted depression at follow-up, controlling for depression at the start of the chronic stress ratings. Next, we determined whether adverse parent–child relationships and/or familial loading for dysthymic disorder moderated the longitudinal association between chronic stress and HAM-D depression. Both baseline adverse parent–child relationships and familial loading for dysthymic disorder were examined simultaneously as Level-2 predictors of the slope of the association between chronic stress and depression over time (controlling for PSR depression at the start of each chronic stress assessment).

$$\begin{aligned} \text{Level 1: } Y_{ij} \text{ (depression)} &= \beta_{0j} + \beta_{1j}(\text{time}) + \beta_{2j}(\text{time}^2) \\ &+ \beta_{3j}(\text{chronic stress}) + \beta_{4j}(\text{PSR prior depression}) + r_{ij} \end{aligned}$$

Level 2:

$$\begin{aligned} \text{Intercept: } \beta_{0j} &= \gamma_{00} + \gamma_{01}(\text{parent–child relationship}) \\ &+ \gamma_{02}(\text{familial dysthymic disorder}) + u_{0j} \\ \text{Time (slope): } \beta_{1j} &= \gamma_{10} + \gamma_{11}(\text{parent–child relationship}) \\ &+ \gamma_{12}(\text{familial dysthymic disorder}) + u_{1j} \\ \text{Time}^2 \text{ (curvature): } \beta_{2j} &= \gamma_{20} \\ &+ \gamma_{21}(\text{parent–child relationship}) \\ &+ \gamma_{22}(\text{familial dysthymic disorder}) \\ \text{Chronic stress: } \beta_{3j} &= \gamma_{30} + \gamma_{31}(\text{parent–child relationship}) \\ &+ \gamma_{32}(\text{familial dysthymic disorder}) + u_{0j} \\ \text{PSR prior depression: } \beta_{4j} &= \gamma_{40} \\ &+ \gamma_{41}(\text{parent–child relationship}) \\ &+ \gamma_{42}(\text{familial dysthymic disorder}) \end{aligned} \quad (4)$$

² Effect sizes were computed with the following formula (Rosenthal, Rosnow, & Rubin, 2000) and are presented for significant effects only: $r = \text{square root of } [t^2/(t^2 + df)]$.

³ We chose to use the 6-month interval for the main analysis to maximize patients' ability to recall chronic stressors. However, we also examined whether chronic stress over the entire previous 30 months predicted HAM-D depression while controlling for HAM-D depression at the previous follow-up. We performed this supplementary analysis to replicate our findings with a control variable that was both assessed prospectively and was the same measure as the dependent variable. Results indicate that greater levels of chronic stress were significantly associated with greater depression at each follow-up (unstandardized coefficient = 0.74), $t(80) = 4.86, p < .001$.

Table 2
Results of Hierarchical Linear Models of Depression Severity Over Time

Fixed effect	β	SE	<i>t</i>	Variance component	SD	χ^2 test of variance
Baseline trajectory model ^a						
Intercept, β_0	25.14	1.02	24.75***	63.72	7.98	262.90***
Time (slope), β_1	-5.39	1.12	-4.80***	44.73	6.69	124.60*
Time ² (curvature), β_2	1.34	0.30	4.40***	1.78	1.33	89.35
Longitudinal association between chronic stress and depression severity ^b						
Intercept, β_0	24.61	1.08	22.69***	45.33	6.73	156.39***
Time (slope), β_1	-6.21	1.28	-4.86***	7.08	2.66	168.04***
Time ² (curvature), β_2	1.62	0.34	4.74***			
Chronic stress, β_3	0.51	0.18	2.78**	0.11	0.33	105.52**
PSR prior depression severity, β_4	2.53	0.43	5.81***			
Effects of adverse parent-child relationships and familial loading for dysthymic disorder ^c						
Intercept, β_0				58.67	7.66	288.77***
Intercept 2, γ_{00}	25.14	1.01	24.82***			
Adverse parent-child relationships, γ_{01}	0.54	0.72	0.72			
Familial loading for dysthymic disorder, γ_{02}	-3.88	21.82	-0.17			
Time (slope), β_1				4.29	2.07	146.13***
Intercept 2, γ_{10}	-5.39	1.08	-5.01***			
Adverse parent-child relationships, γ_{11}	1.46	0.75	1.93*			
Familial loading for dysthymic disorder, γ_{12}	35.19	23.77	1.48			
Time ² (curvature), β_2						
Intercept 2, γ_{20}	1.34	0.30	4.52			
Adverse parent-child relationships, γ_{21}	-0.17	0.21	-0.83			
Familial loading for dysthymic disorder, γ_{22}	-11.12	6.72	-1.66			
Moderation analysis ^d						
Intercept, β_0				53.16	7.29	163.39***
Intercept 2, γ_{00}	24.87	1.19	20.97***			
Adverse parent-child relationships, γ_{01}	-0.17	0.81	-0.21			
Familial loading for dysthymic disorder, γ_{02}	-20.31	27.74	-0.73			
Time (slope), β_1				6.15	2.48	157.24***
Intercept 2, γ_{10}	-6.53	1.25	-5.21***			
Adverse parent-child relationships, γ_{11}	1.34	0.86	1.56			
Familial loading for dysthymic disorder, γ_{12}	41.37	28.66	1.44			
Time ² (curvature), β_2						
Intercept 2, γ_{20}	1.75	0.34	5.09***			
Adverse parent-child relationships, γ_{21}	-0.11	0.23	-0.49			
Familial loading for dysthymic disorder, γ_{22}	-10.76	7.39	-1.46			
Chronic stress, β_3				0.01	0.12	100.50**
Intercept 2, γ_{30}	0.49	0.16	3.00**			
Adverse parent-child relationships, γ_{31}	0.49	0.14	3.52**			
Familial loading for dysthymic disorder, γ_{32}	-12.16	4.18	-2.90**			
PSR prior depression severity, β_4						
Intercept 2, γ_{40}	2.98	0.38	7.94***			
Adverse parent-child relationships, γ_{41}	-0.51	0.21	-2.45*			
Familial loading for dysthymic disorder, γ_{42}	21.77	9.24	2.36*			

Note. Random effects are reported in variance component and chi-square test of variance. PSR = psychiatric status rating of depression from the Longitudinal Interval Follow-Up Evaluation.

^a For *t* test, *df* = 96; for chi-square tests, *df* = 96. ^b For *t* test, *df* = 84 for intercept, time, and chronic stress; for *t* test, *df* = 335 for Time² and PSR prior depression severity; for chi-square tests, *df* = 66. ^c For *t* test, *df* = 94; for *t* test, *df* = 379 for Time²; for chi-square tests, *df* = 94. ^d For *t* test, *df* = 82 for intercept, time, and chronic stress; for *t* test, *df* = 325 for Time² and PSR prior depression severity; for chi-square tests, *df* = 64. * *p* < .05. ** *p* < .01. *** *p* < .001.

Results, as shown in Table 2, indicated that adverse parent-child relationships moderated the longitudinal association between chronic stress and depression (unstandardized coefficient = 0.49), *t*(82) = 3.52, *p* = .001 (ES = .37), in that poorer parent-child relationships predicted a stronger positive association between

chronic stress and depression over time. In addition, familial loading for dysthymic disorder also moderated the longitudinal association between chronic stress and depression over time (unstandardized coefficient = -12.16), *t*(82) = -2.90, *p* = .004 (ES = .30). However, in this case a higher level of familial

dysthymic disorder predicted a negative association between chronic stress and depression over time.

To better understand these interactions, we substituted values one standard deviation above the mean and one standard deviation below the mean into the regression equation and plotted the predicted outcomes (see Aiken & West, 1991; Cohen & Cohen, 1983). The resulting plots revealed the predicted pattern of interactions. As can be seen in Figure 1, for those individuals with more adverse parent–child relationships, there was a greater positive association between chronic stress and depression severity at each follow-up ($\beta_{3j} = 2.11$) than for those with better parent–child relationships ($\beta_{3j} = 0.67$). Next, as can be seen in Figure 2, for those with a more positive familial loading for dysthymic disorder, there was a smaller positive association between chronic stress and depression severity at each follow-up ($\beta_{3j} = 0.82$) than for those with a more negative familial loading for dysthymic disorder ($\beta_{3j} = 1.98$).

Discussion

Using mixed effects models, we investigated the effects of chronic stress, adverse parent–child relationships, and familial loading for dysthymic disorder on the 7.5-year course of dysthymic disorder in an outpatient sample. This type of longitudinal data analysis takes into account both the higher level (between-persons) and the lower level (within-persons) constructs in a hierarchically nested dataset.

First, we examined the 7.5-year course of dysthymic disorder by estimating each individual's trajectory of depression severity over time and the best overall form of change (i.e., linear or quadratic) for the group. Our results indicated that there were significant linear and quadratic growth trajectories over time. The linear effect demonstrated that depression severity tended to decrease over time. However, the quadratic effect demonstrated that the decrease was greatest in the first 2.5 years of follow-up and then began to level off to a low-grade chronic depression over time. These results are consistent with previous findings from this sample on the course of dysthymic disorder (Klein et al., 2000).

Next, we investigated whether chronic stress maintained depression over time. Our results demonstrated that a higher level of chronic stress 6 months prior to each follow-up predicted greater severity of depression at that follow-up after controlling for the

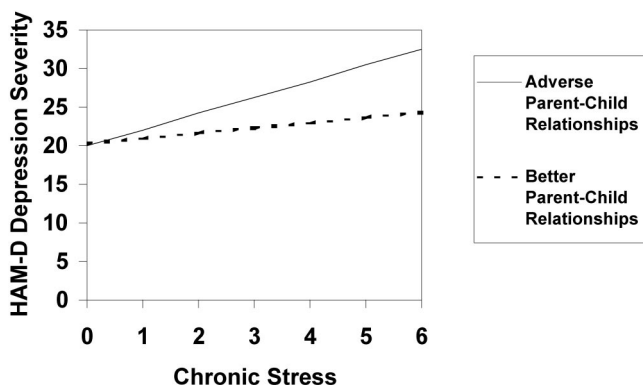


Figure 1. Moderation of adverse parent–child relationships on the relation between chronic stress and Hamilton Depression Rating Scale (HAM-D) depression severity.

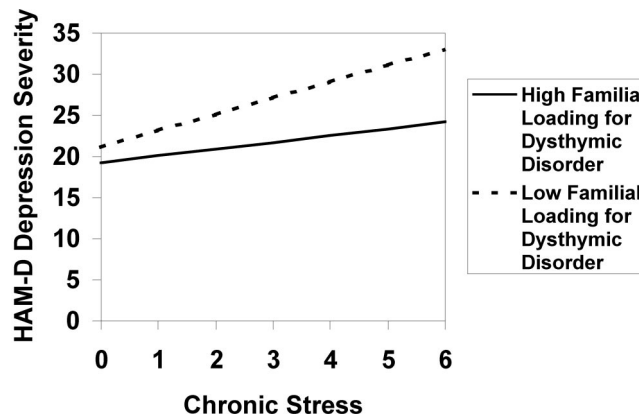


Figure 2. Moderation of family history of dysthymic disorder on the relation between chronic stress and Hamilton Depression Rating Scale (HAM-D) depression severity.

level of depression at the beginning of the period in which stress was assessed. As the focus of our study was on the maintenance of a chronic disorder rather than on the precipitation of acute episodes, we did not explore whether the association between stress and depression changed over time. However, it should be noted that when we examined this association separately at each follow-up with multiple regression analyses, there was no evidence for a decline in the association over time as there may be in the role of stress on the onset of subsequent major depressive episodes (Kendler, Thornton, & Gardner, 2000). Our data support previous findings that chronic stress may play a role in maintaining chronic forms of depression (Brown et al., 1986; Hayden & Klein, 2001). Individuals with dysthymia may be particularly vulnerable to chronic stress because they lack the necessary coping skills to manage the presence of ongoing strain (McCullough et al., 1994; Ravindran et al., 1995). Our results extend previous studies by our use of repeated measurements of chronic stress and depression and mixed effects analyses that allowed us to model the association between chronic stress and depression over time.

Preliminary to our moderation analysis, we also examined whether adverse parent–child relationships and/or family history of dysthymic disorder predicted the course of depression. We found that patients with more adverse parent–child relationships exhibited less of a reduction in depression over time, suggesting that patients with dysthymia with poorer parent–child relationships are likely to have a more severe course of depression than other patients with dysthymia. These findings lend further support for the predictive utility of adverse parent–child relationships on the course of dysthymic disorder (Durbin et al., 2000).

Finally, we examined whether adverse parent–child relationships and/or family history of dysthymic disorder moderated the longitudinal relation between chronic stress and depression. Although we found support for moderation for each vulnerability factor, the effects were in opposite directions as we hypothesized. For adverse parent–child relationships, our findings support a form of moderation in which the presence of more adverse parent–child relationships increased the level of depression in response to stress. Our findings are consistent with Hammen et al.'s (2000) study, demonstrating that young women with childhood adversity succumbed to depression in the face of lower levels of stress than

individuals without such adversity. Hammen et al.'s findings, which were interpreted as supporting a stress-sensitization model, examined depression onset in a community sample of young women. We extended their findings by demonstrating a similar form of stress reactivity or sensitization in the maintenance and course of depression in patients with dysthymia. Taken together, these findings suggest that childhood adversity decreases one's later resiliency to stress, increasing the risk of both onset and chronicity of depression. Interestingly, childhood adversity appears to affect the development of neurobiological processes of stress regulation (Francis, Caldji, Champagne, Plotsky, & Meaney, 1999; Heim et al., 2002) that may play an important role in depression (Gold, Goodwin, & Chrousos, 1988; Heim & Nemeroff, 2001). For instance, early life stress may induce chronic hyperactivity of the corticotropin-releasing factor system as well as alterations in several neurotransmitter systems, resulting in increased vulnerability to stress and depression (Heim & Nemeroff, 2001). Our findings are also consistent with evidence that childhood adversity may be a marker for a subgroup of persons with depression with a distinct pattern of neurobiological abnormalities (e.g., smaller hippocampal volume; Vythilingam et al., 2002) and a better response to psychosocial than to pharmacological treatment (Nemeroff et al., 2003).

We also found that familial loading for dysthymic disorder moderated the association between chronic stress and depression over time. Our results indicated that those with a higher familial loading for dysthymic disorder were less responsive to chronic stress over time. On the other hand, for those with a low familial loading for dysthymic disorder, greater levels of chronic stress predicted greater depression at each follow-up. These findings are consistent with Kendler et al.'s (2001) study, which showed that women at high genetic risk for depression were more likely to experience depressive episodes without major environmental stressors than women at low genetic risk. Consequently, a family history of dysthymic disorder and adverse parent-child relationships may be markers for different etiological and maintenance processes, with the former involving a stable genetic and temperamental predisposition and the latter reflecting greater sensitivity to life stress.

Our findings lend some support to Akiskal's (1983) typology for primary early-onset dysthymia, which differentiates between two subtypes: subaffective dysthymic disorder and character spectrum disorder. Subaffective dysthymic disorder is characterized by a family history of mood disorders and a depressive temperamental style, whereas character spectrum disorder is thought to develop from a background of childhood adversity and loss. Although previous studies have not provided much support for Akiskal's typology (Anderson et al., 1996; Murphy & Checkley, 1990), our findings are broadly consistent with it. Specifically, individuals with dysthymia with a family history of dysthymia and low stress reactivity bear some resemblance to Akiskal's concept of subaffective dysthymic disorder, whereas individuals with dysthymia, a history of adverse parent-child relationships, and a high level of stress reactivity appear to fit into Akiskal's category of character spectrum disorder. Interestingly, however, that family history of dysthymia and adverse parent-child relationships were moderately correlated. Hence, the negative findings of previous studies may be due to the overlap between these variables, making it more difficult to identify distinctive patterns of associations.

The strengths of our study include (a) its prospective design; (b) three follow-up assessments over a 90-month period; (c) semi-structured interviews of stress, depression, and early adversity with probands; and (d) assessments of psychopathology in family members from both relatives and knowledgeable informants. This design allowed us to use mixed effects models to investigate the relationships among chronic stress, parent-child relationships, family history of dysthymic disorder, and the course of dysthymic disorder. However, the study has several limitations. Our sample size was modest, which limited our power to detect small effects. In addition, the chronic stress and depression ratings were collected retrospectively at each follow-up, which increases the chances of poor or biased recall. However, we attempted to reduce this problem by limiting our retrospective data to the 6 months prior to each follow-up and by using a life-calendar approach to facilitate recall. Furthermore, the measure used to control for depression at the time of the chronic-stress rating was not the same measure of depression severity used as the dependent variable. Another limitation is that we used an abbreviated version of the IRLE that may have underestimated levels of stress, particularly mild stressors. Therefore, further work is needed to explore the relationship between mild chronic stress and the course of dysthymia. Finally, even though this is a prospective follow-up study, it is not technologically or ethically possible to manipulate the key variables; hence, we are unable to draw definitive conclusions about causal relationships.

In conclusion, our findings suggest that chronic stress plays a role in the maintenance of depression in individuals with dysthymia. However, this effect varies as a function of history of adverse parent-child relationships and family history of psychopathology. Chronic stress serves to maintain depressive symptomatology over time only among patients with adverse parent-child relationships and patients with a low familial loading for dysthymic disorder. Hence, it appears that there may be several different pathways involved in the maintenance of dysthymic disorder, with one being a familial predisposition whose effects appear to be independent of stress, and the other involving an adverse early home environment that appears to amplify the effects of stress on depressive symptoms. Future research should further seek to delineate these pathways and elucidate the processes responsible for these effects.

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Received September 30, 2003

Revision received December 23, 2003

Accepted February 18, 2004 ■

***Journal of Consulting and Clinical Psychology* Call for Papers:
Special Section on Benefit Finding or Growth Following
Highly Stressful or Traumatic Life Events**

The *Journal of Consulting and Clinical Psychology* is requesting submissions of empirical papers that focus on the positive effects of highly stressful or traumatic events—or what has been referred to in the literature as *benefit finding*, *posttraumatic growth*, or *stress-related growth*. The papers must be empirical rather than theoretical in nature and should address one or more of the following topics:

1. Antecedents to benefit finding (i.e., Who engages in benefit finding?),
2. Measurement issues (i.e., What is benefit finding? How should it be measured?),
3. Mechanisms (i.e., How is benefit finding brought about? How do responses to stress and trauma sometimes result in positive changes?),
4. Validity issues (i.e., Is benefit finding real or is it an illusion/cognitive distortion?), and
5. Links to well-being (i.e., What are effects of benefit finding on well-being?).

If the fifth topic is the focus of the paper, studies must be either longitudinal or experimental. Because children, adolescents, and families have been neglected by past research, papers that examine these populations are especially encouraged. The goal of this special section is to have a set of papers that represent the life span.

The deadline for submissions of manuscripts is June 1, 2005. Final editorial decisions will be made by early 2006, with an anticipated publication date of mid- to late 2006. All submissions should be entered through the main submission portal for the journal (www.apa.org/journals/ccp.html). Authors should indicate in their accompanying cover letter that the paper is to be considered for the special section.

Questions or inquiries regarding the special section should be directed to the section coeditors, Vicki Helgeson (vh2e@andrew.cmu.edu) and Crystal Park (clpark@uconnvm.uconn.edu).