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Dysthymic Disorder and Double Depression: Prediction of 10-Year Course Trajectories and Outcomes

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Abstract

We sought to identify baseline predictors of 10-year course trajectories and outcomes in patients with dysthymic disorder and double depression. Eighty-seven outpatients with early-onset (< 21 years) dysthymic disorder, with or without superimposed major depression, were assessed five times at 30-month intervals for 10 years. Baseline evaluations included semi-structured diagnostic interviews for Axis I and II psychopathology and childhood adversity. Direct interview and family history data were collected on first-degree relatives. Follow-up assessments included the Longitudinal Follow-up Evaluation and Hamilton Depression Rating Scale. Using mixed effects growth curve models, univariate predictors of depression severity and functional impairment at 10-year outcome included older age, less education, concurrent anxiety disorder, greater familial loading for chronic depression, a history of a poorer maternal relationship in childhood, and a history of childhood sexual abuse. In addition, longer duration of dysthymic disorder also predicted greater impairment 10 years later. Predictors of a poorer trajectory of depressive symptoms over time included ethnicity and personality disorders; predictors of a poorer trajectory of social functioning included familial loading of chronic depression and quality of the childhood maternal relationship. Thus, demographic, clinical, family history, and early adversity variables all contribute to predicting the long term trajectory and outcome of DD. These variables should be routinely assessed in clinical evaluations and can provide clinicians with valuable prognostic information.

Keywords

Dysthymic Disorder; Double Depression; Prognosis; Predictors of Course and Outcome

Dysthymic disorder (DD) is defined as a mild, chronic depressive condition. In a 10-year naturalistic follow-up of an outpatient sample, we recently reported that the estimated rate of recovery from DD was 73.9%, however the median time to recovery was 52 months, and the estimated relapse rate was 71.4% (Klein et al., 2006). Although DD is typically characterized by a mild-moderate level of depressive symptoms, almost all individuals with DD experience

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exacerbations that meet criteria for a major depressive episode (MDE), or “double depression”, at some point (Keller et al., 1995; Klein et al., 2006).

Given the chronicity of DD, it is important to identify factors that predict its long-term course and outcome in order to supply clinicians and patients with information about prognosis and provide clues regarding the development and maintenance of the disorder. Unfortunately, the literature on prognostic factors in DD is sparse, and generally limited to brief follow-up periods. A two-year follow-up of double depression reported that age, marital status, and number and duration of MDEs did not predict recovery from DD (Keller et al., 1983). A combined retrospective/prospective follow-up of depressed children found that earlier age of onset (Kovacs et al., 1997) and comorbid externalizing disorders (Kovacs et al., 1984) predicted longer duration of DD, while sex, superimposed MDE, and comorbid anxiety disorders failed to predict recovery from DD (Kovacs et al., 1984, 1989, 1997). Finally, in a report based on the first 5 years of our 10-year naturalistic follow-up study, we found that comorbid anxiety disorders, cluster C personality disorder traits, and family history of bipolar disorder predicted a lower rate of recovery from DD (Hayden & Klein, 2001). In addition, after controlling for initial level of depression, anxiety disorders, cluster C traits, family history of DD, poor childhood relationships with parents, and a history of sexual abuse predicted a higher level of depressive symptoms at the five-year follow-up. Demographic variables, age of onset of DD, and history MDEs and substance use disorders did not predict recovery or level of depression at follow-up (Hayden & Klein, 2001).

This paper examines the baseline predictors of 10-year course trajectories and outcomes in DD and double depression. It extends our previous report (Hayden & Klein, 2001) in several ways. First, the length of the follow-up period has been doubled from 5 to 10 years, allowing us to examine predictors of long-term course and outcome. Second, as functional impairment may be at least as important an outcome as symptomatology, we examined predictors of the course of social functioning as well as depressive symptoms. Finally, our previous report used survival analysis to examine predictors of recovery and multiple regression analysis to examine change in symptoms between baseline and five-year follow-up. In this paper, we use mixed effects growth curve models as an alternative data analytic strategy. Mixed effects growth curve models have a number of advantages over survival analysis and multiple regression analysis. Unlike survival analysis, mixed effects models do not require the creation of somewhat arbitrary categories such as “recovery” and “relapse”, and instead allows for the full continuum of symptom variation; and they utilize all observations for each individual, rather than censoring observations after the first “event” (e.g., recovery) (Singer & Willett, 2003). Mixed effects models also have several advantages compared to traditional multiple regression approaches. They can (1) include more than two waves of assessments; (2) incorporate information about mean level change over time, rather than being based entirely on the covariance between assessment points; (3) distinguish predictors of the trajectory of change from predictors of the level of the dependent variable at outcome; and (4) model individual differences in trajectories over time (random effects) rather than assuming that the pattern of change is the same for all participants (fixed effects) (Bryk & Raudenbush, 1992; Curran, 2000; Singer & Willett, 2003).

Method

The sample and methods have been described previously (Klein et al., 1995; 2006). The sample included 97 outpatients with DSM-III-R early-onset DD (with or without superimposed MDEs). We focused on the early-onset (< 21 years) subtype as it is the prototypical form of DD (Kocsis & Frances, 1987). Patients were 18–60 years old, and were selected from consecutive admissions to the University Outpatient Psychiatry Department and Psychological

Center. Several patients were also referred from the University Counseling Center and a community mental health center.

At least one follow-up was completed with 87 (89.7%) patients. Fifty-six patients completed all four follow-up assessments, 13 completed three follow-ups, 6 patients completed two follow-ups, and 7 patients completed only one follow-up. The mean length of follow-up was 112.4 months ($SD = 24.2$). DD patients who completed, and did not complete, at least one follow-up did not differ on any of the baseline predictors examined in the study.

As this was a naturalistic study, treatment was not controlled. However, we obtained information about treatment from patients and medical records. Treatment was coded using four-point scales for the adequacy of antidepressant medication and the intensity of psychotherapy (Keller et al., 1987). On average, patients received probably or definitely adequate levels of pharmacotherapy for 26.9% of the follow-up period ($SD = 31.2$), and were in weekly or biweekly psychotherapy for 26.7% of the follow-up ($SD = 27.7$). (For more detail on the sample's treatment experience, see McFarland & Klein, 2005). The study was approved by the Stony Brook University Institutional Review Board, and written informed consent was obtained from all patients following a complete description of the study.

Baseline Evaluation

The baseline evaluation included the Structured Clinical Interview for DSM-III-R (SCID; Spitzer et al., 1990), 24-item modified Hamilton Depression Rating Scale (HAM-D; Miller et al., 1985), Personality Disorder Examination (PDE; Loranger, 1988), and Early Home Environment Interview (EHEI; Lizardi et al., 1995).

The EHEI is a semi-structured interview that assesses aspects of the early home environment prior to age 15, including physical abuse (being hit hard or often enough to leave bruises, draw blood, or require medical attention), sexual abuse (non-consenting genital contact initiated by someone at least five years older), and two scales summarizing the quality of the relationship with each parent. Physical and sexual abuse are scored present or absent. The maternal and paternal relationship scales include six items: 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 0–6, with higher scores indicating poorer relationships.

Family history information was obtained from patients for all first-degree relatives older than 14 years ($N = 446$) using an expanded version of the Family History Research Diagnostic Criteria (Andreasen et al., 1977) and direct interviews with relatives using the SCID and PDE. Direct interviews were conducted with 40% of relatives (70% of living relatives we had permission to contact). When unable to interview a family member, we attempted to obtain family history information from at least one additional informant about that relative. Lifetime best estimate diagnoses were derived using all available information (Klein et al., 1994). As described elsewhere (Durbin et al., 2000), family history scores were created using mixed effects models adjusting for differences between families in number and gender of relatives and availability of a direct interview. A family history score for bipolar disorder could not be estimated due to its low prevalence. Instead, family history of bipolar disorder was coded as positive if any relative had a history of bipolar I or II disorder.

Interrater reliability was generally good-to-excellent (Klein et al., 1994; Klein et al., 1995; Lizardi et al., 1995). Kappas for SCID diagnoses of current DD and major depressive disorder (MDD) were 0.90 and 0.78, respectively. Kappas for a PDE diagnosis of any personality disorder was 0.80. Kappas for EHEI childhood sexual and physical abuse were both 0.68, and intraclass correlations for the maternal and paternal relationship scales were .83 and .69,

respectively. Kappas for best-estimate diagnoses of mood disorders in relatives ranged from .75 – .90.

Follow-up Evaluations

Follow-ups were conducted at 30, 60, 90, and 120 months after study entry. They included the Longitudinal Interval Follow-up Evaluation (LIFE; Keller et al., 1987) and HAM-D (Miller et al., 1985). The LIFE is a semi-structured interview that assesses the course of Axis I disorders, social functioning, and treatment.

We administered a modified version of the LIFE social functioning module, which assessed impairment in work, school, as a homemaker, as a parent, and in relationships with significant others, family members, and friends, in the month before each assessment (Leader & Klein, 1996). For this paper, we used the interviewer-rated global social functioning scale, which was scored on a six-point scale ranging from “no impairment/very good adjustment” to “incapacitation in major role”.

Follow-ups were conducted by masters- and doctoral-level clinicians who were unaware of patients’ baseline data. To assess interrater reliability, the two main study raters conducted separate HAM-D interviews ($N = 13$). The intraclass correlation for the HAM-D was .96.

Data Analysis

Five (5.7%) of the 87 DD patients developed manic or hypomanic episodes during follow-up, and were excluded from all analyses (Klein et al., 2006). In order to explore the predictors of HAM-D and LIFE global social functioning over time, we first examined the univariate effects of a selected group of baseline variables and then combined the significant predictors in a multivariate model.

Predictors (with their prevalence or mean and standard deviation in the sample) included five demographic variables: age ($M = 32.0$; $SD = 9.8$), education ($M = 13.3$ years; $SD = 2.2$), sex (74.4% female), ethnicity (91.5% Caucasian), and marital status (28.0% married; 20.7% separated/divorced; 2.4% widowed; 48.8% never married); eight clinical variables: concurrent MDE (56.1%), lifetime MDD (76.8%), age of onset of DD ($M = 10.4$; $SD = 5.0$), years duration of DD ($M = 21.6$; $SD = 12.4$); concurrent anxiety disorder (36.6%), lifetime substance use disorder (51.2%), any personality disorder (58.5%), and lifetime history of attempted suicide (31.7%); three family history variables: family history of bipolar disorder (7.3%), and familial loadings of MDD ($M = .10$; $SD = .01$) and chronic depression ($M = .01$; $SD = .04$) in first-degree relatives; and four early adversity variables: poor childhood relationships with mother ($M = 1.9$; $SD = 1.8$) and father ($M = 2.4$; $SD = 1.8$), sexual abuse (28.0%), and physical abuse (29.6%).

Predictors of depressive symptoms and functional impairment were examined using mixed effects growth curve models (Bryk & Raudenbush, 1992; Curran, 2000; Singer & Willett, 2003). Predictors were treated as fixed effects, and time (in months) was a random effect. Time was coded so that the intercepts of patients’ growth curves reflected their estimated scores at 10-year follow-up.

In unconditional growth curve models (i.e., models without any predictors), there was a significant quadratic effect for time on the HAM-D, but not LIFE social functioning. The quadratic effect for the HAM-D reflected a greater decrease in depression between baseline and the 30-month follow-up than between subsequent assessments. This is probably because the baseline evaluation was conducted when patients entered treatment and were at a peak level of depression, whereas the timing of subsequent assessments was independent of patients’ treatment and clinical status. As the quadratic effect was limited to the initial portion of the

follow-up, we report only the linear effects. Nonetheless, we controlled for quadratic effects in the HAM-D growth models. As the models failed to converge when both the linear and quadratic trends were treated as random variables, the quadratic parameter was fixed.

Results

Means for HAM-D and LIFE global social functioning at each assessment appear in Table 1.

Effects of treatment

Although this was a naturalistic study, we first examined the association between treatment and HAM-D and LIFE global social functioning scores over time. Mean level of pharmacotherapy and psychotherapy in the three months prior to each assessment were treated as random level 1 (within-person) variables. Greater pharmacotherapy in the three months before each follow-up assessment predicted significantly greater depressive symptoms (coefficient = .902; SE = .336, t [81] = 2.69, p = .008). Although this could indicate that pharmacotherapy had an adverse effect on course, it is more likely that patients with poorer prognoses received more pharmacotherapy. Psychotherapy in the three months before each follow-up assessment did not predict depressive symptoms (coefficient = .421; SE = .374, t [81] = 1.13, p = .26). Finally, neither pharmacotherapy (coefficient = .072; SE = .045, t [81] = 1.58, p = .11) nor psychotherapy (coefficient = .056; SE = .038, t [81] = 1.49, p = .14) predicted the course of social functioning. In light of the limited associations between treatment and course, treatment was not included as a covariate in the analyses.

Depression severity

In the univariate analyses, six variables significantly predicted higher estimated HAM-D scores at 10-year follow-up (intercept): older age, t (80) = 1.96, p = .05; less education, t (80) = -3.68, p < .001; concurrent anxiety disorder, t (80) = 3.26, p = .002; greater familial loading for chronic depression, t (80) = 2.63, p = .009; poorer childhood relationship with mother, t (80) = 4.00, p < .001; and history of sexual abuse, t (80) = 2.15, p = .03 (see Table 2). There were two significant univariate predictors of the slope: ethnicity, t (80) = 2.25, p = .025, and any personality disorder, t (80) = 2.69, p = .008. This reflected a significant linear decrease in depressive symptoms over time among non-whites and patients without baseline personality disorders, but no significant change in the linear component of the HAM-D slope among whites and patients with personality disorders.

Significant univariate predictors of the HAM-D intercept and slope were combined in a multivariate model. As it is advisable to limit the number of level 2 variables included in a model, we did not include age due to its marginal significance in the univariate model. In the multivariate model, significant unique predictors of higher estimated HAM-D scores at 10-year follow-up (intercept) included: concurrent anxiety disorder, t (74) = 2.61, p = .009, and familial loading for chronic depression, t (74) = 2.08, p = .038 (Table 2). Significant predictors of the HAM-D slope included ethnicity, t (74) = 2.36, p = .018, education, t (74) = 2.41, p = .016, personality disorder, t (74) = 2.98, p = .003, and childhood relationship with mother, t (74) = 1.99, p = .046. The effects of ethnicity and personality disorders on slope were similar to the univariate analyses. In addition, two new predictors of slope, education and maternal relationship, emerged in the multivariate model. Patients who had both less education and better maternal relationships improved over time, whereas patients with more education and/or poor maternal relationships exhibited minimal change.

Functional Impairment

Seven variables significantly predicted greater estimated functional impairment at 10-year follow-up (intercept) in the univariate analyses (see Table 3): older age, t (80) = 2.62, p = .009,

less education, $t(80) = -3.04$, $p = .003$, longer duration of DD, $t(80) = 2.51$, $p = .01$, concurrent anxiety disorder, $t(80) = 2.78$, $p = .006$, greater familial loading for chronic depression, $t(80) = 2.67$, $p = .008$, poorer childhood relationship with mother, $t(80) = 5.40$, $p < .001$, and history of sexual abuse, $t(80) = 2.69$, $p = .008$. There were two significant univariate predictors of the slope: familial loading for chronic depression, $t(80) = -2.15$, $p = .032$, and childhood relationship with mother, $t(80) = -3.51$, $p = .001$. Patients with greater familial loadings of chronic depression, and those with poorer maternal relationships exhibited high, but stable, levels of impairment throughout the follow-up. In contrast, patients with lower familial loadings of chronic depression and those with better maternal relationships exhibited significant linear increases in impairment, but still remained less impaired at 10 years than patients with higher familial loadings of chronic depression and poorer maternal relationships.

Significant univariate predictors of the functional impairment intercept and slope were combined in a multivariate model. In the multivariate model, significant unique predictors of greater estimated social impairment at 10-year follow-up (intercept) included: concurrent anxiety disorder, $t(74) = 1.96$, $p = .05$, greater familial loading of chronic depression, $t(74) = 2.12$, $p = .03$, and poorer childhood relationship with mother, $t(74) = 3.14$, $p = .002$ (Table 3). There were three significant predictors of the linear slope of functional impairment: age, $t(74) = -1.95$, $p = .05$, familial loading of chronic depression, $t(74) = -1.97$, $p = .049$, and quality of maternal relationship, $t(74) = -2.40$, $p = .017$. Older patients had high, but stable, levels of impairment throughout the follow-up. In contrast, younger patients exhibited increasingly greater impairment over time, but remained less impaired at 10 years than older participants. The effects for familial loading of chronic depression and quality of maternal relationship were similar to those described above for the univariate analyses.

Discussion

To our knowledge, this is the first prospective study of predictors of long-term course and outcome in adults with DD and double depression. This paper extends our previous report (Hayden & Klein, 2001) on predictors of five-year outcome by doubling the length of the follow-up period, examining predictors of functional impairment as well as depressive symptoms, and using mixed effects growth curve analyses.

In the univariate models, six variables predicted both greater depression and poorer functioning at 10 year outcome: older age, less education, concurrent anxiety disorder, greater familial loading for chronic depression, and a poorer maternal relationship and history of sexual abuse in childhood. In addition, a longer duration of DD predicted greater functional impairment 10 years later. The similarity in predictors of depressive symptoms and psychosocial impairment is not surprising, as measures of depression and social functioning are moderately correlated (Leader & Klein, 1996). However, the predictors of the trajectories of depression and social functioning over time differed. In the univariate models, non-white ethnicity and absence of personality disorder predicted greater improvement in depression, while lower familial loadings of chronic depression, and better maternal relationships predicted increasing impairment in functioning. The direction of the effect of ethnicity on the slope of depressive symptoms, and the effects of familial loading of chronic depression and maternal relationship on functional impairment were somewhat surprising. Both whites and non-whites experienced reductions in depressive symptoms over time, however the rate of improvement was steeper among non-whites and achieved statistical significance only in this subgroup. This finding should be regarded cautiously, as there were only seven ethnic minority patients in the sample. The associations between a lower familial loading of chronic depression and a better maternal relationship in childhood with a slower rate of improvement in social functioning probably reflect ceiling effects, as patients with higher loadings of chronic depression and poorer maternal relationships continued to exhibit greater impairment at 10-year outcome than patients

with lower familial loadings of chronic depression and better maternal relationships, despite the latter group's increasing impairment over time. Most predictors in the univariate models continued to be significant in the multivariate models.

Some predictors in our analyses of the first five years of follow-up were not significant in the 10-year follow-up (e.g., family history of bipolar disorder), and some new predictors emerged (e.g., age, education, and ethnicity). However, many of the same variables predicting 5-year outcome continued to predict course and/or outcome after ten years, including comorbid anxiety and personality disorders, familial loading of chronic depression, poor childhood maternal relationship, and sexual abuse history.

From a broader perspective, the results indicate that demographic, clinical, family history, and early adversity variables all contribute to predicting the long-term course and outcome of DD. The most consistent predictors across outcome domains included comorbid anxiety and personality disorders, familial loading of chronic depression, poor childhood maternal relationship, and sexual abuse history. These prognostic variables are probably not specific to DD, as most also predict outcome in MDD (Daley et al., 2000; Grilo et al., 2005; Lara et al., 2000). Hence, they appear to predict the course of a spectrum of depressive, and probably non-depressive, conditions. Importantly, the variables examined in this report should be routinely assessed in clinical evaluations and can provide clinicians with potentially valuable prognostic information.

An important question is how the baseline predictors in this study influenced course over a decade or longer. Anxiety and personality disorders tend to be chronic conditions, hence they may have persisting contemporaneous influences on the long-term course of DD. In addition, there is evidence that the effect of personality disorders on the course of depression is mediated by interpersonal difficulties (Daley et al., 1998). The impact of anxiety disorders on the course of depression may be mediated by avoidance of potentially ameliorative behaviors (Jacobson et al., 2001). The influence of early adversity on the course of depression may be mediated by long-term effects of adversity on developing stress response systems, as adults with histories of childhood trauma exhibit increased autonomic and neuroendocrine reactivity to laboratory stressors (Heim et al., 2000), and greater depression in response to life events (Dougherty et al., 2004; Kendler et al., 2004). Finally, familial loading of chronic depression may reflect a temperamental predisposition to persistent depressive states (Durbin et al., 2005).

These findings suggest that it may be useful to tailor treatment to take these poor prognostic factors into account. For example, there is evidence that psychotherapy may be more effective than pharmacotherapy for chronically depressed patients with a history of early adversity (Nemeroff et al., 2003). In addition, cognitive-behavioral psychotherapies that target anxiety disorders or avoidance may be useful, either alone or in combination with antidepressant pharmacotherapy, for DD patients with comorbid anxiety disorders. Finally, patients with comorbid personality disorders may require a modified and more intensive course of treatment.

This study had many strengths, including a prospective, longitudinal design with five evaluations over the course of 10 years, an extensive collection of baseline predictors, and semi-structured interview assessments of all prognostic and outcome measures. However, the study also had several limitations. We conducted a large number of statistical tests without adjusting significance levels, raising the possibility of some chance findings. The sample size was modest, limiting our ability to detect small effects. Patients reported on lengthy time intervals, possibly introducing error into dating recovery and relapse. Finally, the majority of patients had a history of MDD. However, superimposed MDEs appear to be part of the natural

history of DD, rather than constituting a distinct syndrome (Akiskal, 1983; Keller et al., 1995; Klein et al., 2006).

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Mean 24-Item Hamilton Rating Scale for Depression (HAM-D) and Longitudinal Interval Follow-up Evaluation (LIFE) Global Social Functioning Scores at each Assessment

Assessment	N	HAM-D		Social Functioning	
		M	(SD)	M	(SD)
Baseline	92	25.3	(10.6)	4.0	1.1
30 Months	71	17.7	(10.4)	3.5	1.3
60 Months	73	19.5	(12.2)	3.7	1.4
90 Months	72	18.7	(11.5)	3.5	1.4
120 Months	66	16.8	(10.4)	3.3	1.2

Note. The LIFE Global Social Functioning Scale was scored as follows: 1) no impairment; very good adjustment; 2) no impairment, good adjustment; 3) mild impairment; 4) moderate impairment; 5) marked impairment; 6) complete incapacitation in major role.

Table 2

Univariate and Multivariate Mixed Effects Growth Curve Models of Predictors of 10-Year Course and Outcome of Depressive Symptoms in Patients with Dysthymic Disorder (DD)

Variable	Univariate Model		Multivariate Model	
	10-year outcome b (SE)	Change over time b (SE)	10-year outcome b (SE)	Change over time b (SE)
Demographic				
Age	0.192 (0.098)*	−0.041 (0.029)		
Education	−1.610 (0.438)***	0.017 (0.015)	−0.901 (0.497) ⁺	0.034 (0.014)*
Sex	3.295 (2.888)	0.016 (0.061)		
Ethnicity	0.626 (3.645)	0.167 (0.074)*	−2.663 (2.931)	0.212 (0.090)*
Marital Status				
Married	−0.080 (0.070)	0.001 (0.001)		
Separated/Divorced	−0.070 (0.078)	0.001 (0.001)		
Widowed	−0.301 (0.186)	0.003 (0.001) ⁺		
Clinical				
Concurrent MDD	4.143 (2.261) ⁺	−0.077 (0.058)		
Lifetime MDD	4.721 (2.663) ⁺	−0.103 (0.058) ⁺		
% Time Depressed				
Past 2 Years	2.000 (1.292)	0.022 (0.041)		
Age of Onset of DD	−0.184 (0.217)	−0.004 (0.006)		
Duration of DD	0.148 (0.079) ⁺	−0.001 (0.002)		
Anxiety Disorder	8.125 (2.493)**	−0.024 (0.064)	5.386 (2.061)**	−0.048 (0.060)
Substance Use Disorder	2.144 (2.343)	−0.077 (0.057)		
Personality Disorder	1.162 (2.320)	0.153 (0.057)**	−0.777 (2.026)	0.157 (0.053)**
Cluster A PD	0.538 (5.134)	0.097 (0.084)		
Cluster B PD	3.892 (2.692)	0.099 (0.066)		
Cluster C PD	2.958 (2.757)	0.121 (0.067) ⁺		
Hx suicide attempt	4.459 (2.711) ⁺	−0.014 (0.063)		
Family History/Familial Loading				
Bipolar Disorder	−0.528 (2.859)	−0.009 (0.120)		
MDD	30.253 (20.232)	−0.681 (0.491)		
Chronic Depression	75.731 (28.850)**	0.008 (0.660)	47.458 (22.862)*	0.020 (0.627)
Early Adversity				
Poor Maternal Relation	2.422 (0.605)***	0.018 (0.015)	1.168 (0.664) ⁺	0.029 (0.015)*
Poor Paternal Relation	1.186 (0.666) ⁺	0.019 (0.017)		
Sexual Abuse	6.313 (2.941)*	0.070 (0.058)	3.267 (2.319)	0.011 (0.055)
Physical Abuse	3.124 (2.683)	−0.044 (0.065)		

Note. 10 year outcome = intercept; change over time = slope; MDD = Major Depressive Disorder; PD = Personality Disorder; Hx = history; Marital status categories are compared to reference category never married; Anxiety Disorders are concurrent; Substance Use Disorders are lifetime.

⁺ p ≤ .10;

* p ≤ .05;

** p ≤ .01;

* p ≤ .001.

Table 3

Univariate and Multivariate Mixed Effects Growth Curve Models of Predictors of 10-Year Course and Outcome of Functional Impairment in Patients with Dysthymic Disorder (DD)

Variable	Univariate Model		Multivariate Model	
	10-year outcome b (SE)	Change over time b (SE)	10-year outcome b (SE)	Change over time b (SE)
Demographic				
Age	0.0373 (0.0143) **	-0.0002 (0.0001) +	0.0348 (0.0298)	-0.0006 (0.0003) *
Education	-0.1535 (0.0505) **	0.0006 (0.0004) +	-0.0042 (0.0546)	-0.0000 (0.0004)
Sex	0.1279 (0.3444)	-0.0009 (0.0023)		
Ethnicity	0.5170 (0.2848) +	-0.0030 (0.0022)		
Marital Status				
Married	0.1107 (0.3412)	-0.0045 (0.0025) +		
Separated/Divorced	0.2871 (0.3869)	-0.0024 (0.0029)		
Widowed	0.8665 (0.9316)	-0.0071 (0.0068)		
Clinical				
Concurrent MDD	-0.0597 (0.2913)	0.0029 (0.0021)		
Lifetime MDD	-0.1034 (0.3267)	0.0007 (0.0023)		
% Time Depressed				
Past 2 Years	0.2817 (0.2058)	0.0010 (0.0014)		
Age of Onset of DD	-0.0361 (0.0300)	-0.0001 (0.0002)		
Duration of DD	0.0289 (0.0115) **	-0.0001 (0.0001)	-0.0213 (0.0263)	0.0004 (0.0002)
Anxiety Disorder	0.9029 (0.3244) **	-0.0039 (0.0025)	0.4873 (0.2492) *	-0.0016 (0.0021)
Substance Disorder	-0.0765 (0.2919)	0.0007 (0.0022)		
Personality Disorder	0.2431 (0.2963)	0.0039 (0.0022) +		
Cluster A PD	0.4006 (0.4669)	-0.0038 (0.0034)		
Cluster B PD	0.4474 (0.3161)	0.0006 (0.0023)		
Cluster C PD	0.4430 (0.3599)	0.0022 (0.0025)		
Hx suicide attempt	0.5469 (0.3498)	0.0019 (0.0026)		
Family History/Familial Loading				
Bipolar Disorder	-0.2960 (0.2212)	-0.0007 (0.0018)		
MDD	3.1331 (2.922)	-0.0233 (0.0219)		
Chronic Depression	9.6759 (3.6223) **	-0.0548 (0.0255) *	6.2654 (2.9494) *	-0.0435 (0.0221) *
Early Adversity				
Maternal Relation	0.3951 (0.0732) ***	-0.0019 (0.0005) ***	0.2841 (0.0906) **	-0.0016 (0.0007) *
Paternal Relation	0.0621 (0.0825)	-0.0006 (0.0006)		
Sexual Abuse	0.9889 (0.3674) **	-0.0042 (0.0029)	0.4613 (0.3145)	-0.0023 (0.0026)
Physical Abuse	0.3152 (0.3624)	0.0002 (0.0023)		

Note. 10 year outcome = intercept; change over time = slope; MDD = Major Depressive Disorder; PD = Personality Disorder; Hx = history; Marital status categories are compared to reference category never married; Anxiety Disorders are concurrent; Substance Use Disorders are lifetime.

+ p ≤ .10;

* p ≤ .05;

** p ≤ .01;

* p ≤ .001.