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Predictors of First Lifetime Onset of Major Depressive Disorder in Young Adulthood

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Abstract

The first onset of major depressive disorder (MDD) most frequently occurs in young adulthood. However, few studies have examined predictors of first lifetime MDD during this high-risk period. The present study examined a broad range of demographic, clinical, and psychosocial variables as prospective predictors of first onset of MDD in a large community sample of young adults (N = 502) from the Oregon Adolescent Depression Project. Between ages 19-31, 35.3% of the sample had a first lifetime MDD episode. Female gender, familial loading of mood disorders, history of childhood sexual abuse, prior history of anxiety disorder, poor self-reported physical health, and subthreshold depressive symptoms significantly predicted MDD onset. In a multivariate model, female gender, familial loading of mood disorders, and subthreshold depression each contributed unique variance in predicting first lifetime MDD. This model had a moderate-to-large effect in predicting MDD onset. Gender did not moderate the other predictors, and the magnitude of the effects did not diminish over the course of the follow-up. These findings indicate that a number of risk factors significantly predict first lifetime MDD in young adulthood, and that simple multivariate risk models may be useful for identifying individuals at high risk for MDD.

Keywords

major depressive disorder; onset; risk; young adulthood

Major depressive disorder (MDD) is among the most prevalent mental disorders, and is associated with substantial disability and mortality (Kessler & Wang, 2009). Although MDD can appear at any age between early childhood and older adulthood, its incidence peaks in young adulthood (Kessler et al., 2005). National (Kessler et al., 2005) and cross-national

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(Weissman et al., 1996) epidemiological studies report that the first onset of MDD most frequently occurs in the 20s to early 30s.

It is important to identify variables that predict MDD to elucidate its etiopathogenesis and identify high-risk groups for targeted prevention. However, most work in this area has combined first-onset and recurrence cases, which is problematic given that the processes involved in first and subsequent episodes of MDD may differ (Monroe & Harkness, 2011). There is a small literature on predictors of first lifetime onset of MDD, however most studies have focused on only a small number of variables (e.g., Carter & Garber, 2011; Eaton et al., 2008; Horwath, Johnson, Klerman, & Weissman, 1992; Lewinsohn, Allen, Seeley, & Gotlib, 1999; Zimmermann et al., 2008). Few studies have examined a broad range of predictors of first MDD incidence in representative community samples, most of which used samples with a very wide age range (18 and above; De Graaf, Bijl, Smit, & Vollebergh, 2002; Grant et al., 2009). To our knowledge, only two studies have specifically examined even a portion of the period of peak risk for MDD—age 17–25 in Jaffee et al. (2002) and age 19–21 in Shanahan, Copeland, Costello, and Angold (2011). As etiological influences on MDD may vary as a function of age (Jaffee et al., 2002), it is important to examine predictors of incident MDD specifically during the period of peak risk in early adulthood.

Two other important issues concern whether the effects of predictors vary as a function of gender and time. First, although females are at higher risk for MDD than males, only a few prospective studies have examined whether the predictors of first lifetime MDD differ as a function of gender (Carter & Garber, 2011; De Graaf et al., 2002; Lewinsohn et al., 1999). These studies did not find gender interactions, but most examined only a few risk factors. Second, with rare exceptions (Shanahan et al., 2011), few studies have explored whether the effects of predictors diminish as the duration of follow-up increases. This has practical, as well theoretical, significance, as some prognostic variables may be useful for only a limited period of time.

The present study examined a broad range of demographic, clinical, and psychosocial variables to identify predictors of first onset of MDD from age 19–31 in a large community sample. Secondary questions included whether the effects of predictors varied as a function of gender and time. The sample was evaluated on three occasions using semistructured diagnostic interviews, and first-degree relatives were interviewed to assess familial psychopathology. Predictors were selected from past research on MDD onset in adolescents and adults (e.g., Coryell, Endicott, & Keller, 1992; Jaffee et al., 2002; Smit, Beekman, Cuijpers, de Graaf, & Vollegergh, 2004).

Method

Participants

The Oregon Adolescent Depression Project (OADP; see Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993) is a longitudinal study of a large sample of high school students who were assessed on four occasions from ages 17–31. Participants were randomly selected from nine senior high schools representative of western Oregon. At this time, 1,709 adolescents (mean age = 17) completed the initial (T_1) assessment (61% participation). One year later, 1,507 adolescents (88%) returned for a second evaluation (T_2) [mean age = 18]. Differences between the initial sample and the population from which it was selected, and between T_1 participants who did and did not participate at T_2, were small (Lewinsohn et al., 1993).

All adolescents with a history of psychopathology by T_2 (n = 644) and a random sample of adolescents with no history of psychopathology by T_2 (n = 457) were invited to participate

in a third (T_3) evaluation. All non-White T_2 participants were retained to maximize ethnic diversity. Of the 1,101 T_2 participants selected for a T_3 interview, 941 (85%) completed the T_3 evaluation (mean age = 25). All T_3 participants were subsequently invited to participate in the T_4 assessment; it was completed by 816 (87%; mean age = 31).

At T_3, we assessed lifetime psychopathology in first-degree relatives. Of the 941 probands with T_3 data, diagnostic data for relatives were available for 803 (85%). Informed consent was obtained from all participants (or guardian for participants under 18).

The present sample includes 502 participants who completed all diagnostic interviews through T_4, had no lifetime history of mood disorder through T_2 (adolescence), and had no lifetime history of bipolar or psychotic disorder through T_4.

Diagnostic Measures

At T_1 and T_2, participants were interviewed with the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Orvaschel, Puig-Antich, Chambers, Tabrizi, & Johnson, 1982), which included items to derive Diagnostic and Statistical Manual of Mental Disorders, 3rd edition revised (*DSM–III–R*; American Psychiatric Association, 1987) diagnoses. Follow-up assessments at T_2 and T_3 were jointly administered with the Longitudinal Interval Follow-Up Evaluation (LIFE; Keller et al., 1987). The T_4 interview consisted of a joint administration of the LIFE and the Structured Clinical Interview for *DSM–IV* (SCID; First, Spitzer, Gibbon, & Williams, 1997). Diagnoses were based on *DSM–III–R* for T_1–T_3 and *DSM–IV* for T_4. Interviews at T_3 and T_4 were conducted by telephone, which yields comparable results to face-to-face interviews (Rohde, Lewinsohn, & Seeley, 1997). Data on lifetime anxiety, disruptive behavior, and substance use disorders before age 18, and first-onset MDD episodes after T_2, were used in the present analyses.

Interviewers had advanced degrees in a mental health field and completed a 70-hr course in diagnostic interviewing. Interrater reliabilities for all diagnoses used in this paper were good to excellent (see Lewinsohn et al., 1993; Rohde et al., 2007).

Self-Report Measures

Participants completed a battery of questionnaires at each assessment (Lewinsohn et al., 1994). The following variables from the T_2 assessment are included in this report.

Subthreshold depressive symptoms—The 20-item Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977) assessed subthreshold depressive symptoms ($\alpha = .90$). Items are rated from 1 (*rarely or none of the time*) to 4 (*most, or all of the time*).

Suicidal ideation—Suicidal ideation was assessed using the sum of four items, each rated on a 4-point scale adopted from the CES-D: “I thought about killing myself”; “I had thoughts about death”; “I felt my family and friends would be better off if I were dead”; and “I felt that I would kill myself if I knew a way” ($\alpha = .84$).

Self-rated physical health—Respondents rated their physical health on a 4-point scale ranging from 1 (*poor*) to 4 (*excellent*).

Self-esteem—Three items from Rosenberg’s (1965) self-esteem scale were included ($\alpha = .85$).

Major life events—Fourteen negative life events during the past year were selected from several life events inventories (Dohrenwend, Krasnoff, Askenasy, & Dohrenwend, 1978; Sandler & Block, 1979). Items that may reflect symptoms of psychopathology and events that did not have a direct impact on the participant were not included.

Daily hassles—Twenty items from the Unpleasant Events Schedule (Lewinsohn, Mermelstein, Alexander, & PacPhillamy, 1985) were administered (α= .89).

Perceived social support—Perceived social support from family and friends was assessed using 20 items adapted from the Arizona Social Support Interview Schedule (Barrera et al., 1986) [α= .78 for family and .71 for friends].

Childhood physical and sexual abuse—Childhood physical and sexual abuse were assessed retrospectively at T_3 using 12 items from the Assessing Environment II (Berger, Knutson, Mehm, & Perkins, 1988) and five items from the Childhood Trauma Questionnaire (Bernstein et al., 1994), respectively (α= .71 for physical abuse and .96 for sexual abuse).

Familial Psychopathology

Parents and siblings age 18 years or older were interviewed with the nonpatient SCID (First et al., 1997). Siblings ages 14-18 years were interviewed with the K-SADS. Interviewers were unaware of probands' diagnoses. Family history data were collected from the proband, and, for relatives who could not be directly interviewed, from another relative. Of 1,506 first-degree relatives with diagnostic information, direct interviews were obtained from 1,014 (67.3%). Two sources of data were available for 1,308 (86.9%) family members.

Two diagnosticians, blind to proband diagnoses, reviewed all data for each relative and independently derived best-estimate *DSM–IV* diagnoses. Disagreements were resolved by consensus. Interrater reliabilities of diagnoses from direct and family history interviews, and best estimates were good to excellent (Klein, Lewinsohn, Seeley, & Rohde, 2001).

Data Analyses

As participants with no history of psychopathology were under-sampled at T_3, participants were weighted as a function of the probability of selection at T_3 in all analyses. Predictors included four demographic variables from T_2 (gender, race, living with both biological parents, and parental education); three preexisting non-mood disorder variables (lifetime anxiety, disruptive behavior, and substance use diagnoses before age 18); three family history variables (proportions of first-degree relatives with lifetime mood, anxiety, and substance use disorders); two childhood abuse variables (physical and sexual abuse); two symptom variables (subthreshold depressive symptoms and suicidal ideation at T_2); self-rated physical health at T_2; and five psychosocial variables assessed at T_2 (self-esteem, stressful life events, daily hassles, and social support from family and friends).

After testing the proportionality assumption, bivariate Cox proportional hazards (PH) models were estimated in which age at onset of first lifetime MDD was regressed, separately, on the predictors. Benjamini and Hochberg's (1995) procedure was used to correct for multiple comparisons. Statistically significant bivariate predictors were subsequently entered into a simultaneous multivariate model to determine unique effects. Further model trimming was achieved using a backward elimination process. The retaining p value for backward elimination was .05.

The utility of the combination of unique predictors was evaluated by computing the area under the curve (AUC) in a receiver operating characteristic (ROC) analysis. The AUC

values of .56, .64, and .71 correspond to small, medium, and large effects, respectively (Rice & Harris, 2005). Positive predictive value (PPV) and negative predictive value (NPV) were derived using an optimum cutoff score that maximized sensitivity and specificity.

Results

Of the 502 participants, 177 (35.3%) had a first lifetime MDD episode between ages 19–31. First onsets were distributed across the follow-up period, but hazard rates were somewhat higher in the earlier portion of the period (e.g., the median annual hazard rate from ages 19–24 was .05, vs. .03 for ages 25–31). The cumulative hazard rate was 38%.

Descriptive data on the 20 predictors appear in Table 1.[1] Results of bivariate Cox PH models examining the associations of each predictor with first onset of MDD appear in Table 2. Female gender, familial loading of mood disorders, history of childhood sexual abuse, history of anxiety disorder before age 18, poor self-reported physical health, and subthreshold depressive symptoms were statistically significant after correction for multiple comparisons.

None of the interactions between gender and the other predictors were significant after correcting for multiple comparisons, indicating that the effects were similar for males and females. To test whether predictive utility changed as a function of time, we examined the interactions of each of the predictors with time. None of these interactions approached significance, indicating that the magnitude of the associations between the predictors and MDD onset did not change over the course of the follow-up.[2]

The significant bivariate predictors were entered into a multivariate PR model using the backward de-selection procedure to determine the most salient prognostic variables. Female gender (hazard ratio [HR] = 2.52, 95% confidence interval [CI] = 1.76–3.59, $p < .001$), familial loading of mood disorders (HR = 2.28, 95% CI = 1.36-3.82, $p = .002$), and subthreshold depressive symptoms (HR = 1.02, 95% CI = 1.01-1.04, $p < .05$) were retained as statistically significant predictors, with a medium-large effect size and moderate classification properties (AUC = .69; PPV = .51; NPV = .74; sensitivity = .62; specificity = .65).

Discussion

We examined predictors of first-onset MDD in young adulthood, the period of peak incidence for depression, which has been curiously neglected in the literature. To our knowledge, this is the first study to assess a broad set of risk factors in predicting MDD incidence across the young-adult developmental period. In bivariate analyses, we identified six predictors of first onset of MDD in young adulthood: female gender, familial loading of mood disorders, history of childhood sexual abuse, anxiety disorder in childhood/adolescence, poor self-reported physical health, and subthreshold depressive symptoms. These variables are generally recognized as risk factors for depression in the literature, and previous studies of predictors of first-onset MDD in community samples have reported similar effects for gender (De Graaf et al., 2002; Grant et al., 2009), family history of depression (Zimmerman et al., 2008), childhood maltreatment (Jaffee et al., 2002; Smit et

[1]Forty (7.9%) participants reported experiencing one or more suicidal ideation items at least occasionally or a moderate amount of the time; 202 (40.3%) reported experiencing at least one of the eight physical abuse items that would mandate reporting; and 68 (13.5%) reported having been molested or sexually abused.

[2]Life events, hassles, and social support were also assessed at T_3. To examine whether these variables had more proximal effects, we used logistic regression to test whether the T_2 values of these variables predicted first MDD onset from T_2–T_3, and their T_3 values predicted first onset from T_3–T_4. Of the eight models, only hassles at T_3 significantly predicted MDD onset at T_4 (OR = 1.03, 95% CI = 1.01–1.05, $p = .009$).

al., 2004), anxiety disorders (Eaton et al., 2008; Grant et al., 2009; Smit et al., 2004), health problems (Smit et al., 2004), and subthreshold depression (De Graaf et al., 2002; Horwath et al., 1992).

Surprisingly, the two previous studies focusing on onset of first-lifetime MDD in early adulthood found few prospective predictors (Jaffee et al., 2002; Shanahan et al., 2011). However, our study differed from each of them in significant ways. Both Jaffee et al. and Shanahan et al. examined only the early part of the peak risk period for MDD (up to age 25 and age 21, respectively). In addition, due to the small number of cases of MDD ($n = 16$), Shanahan et al. combined new onsets of MDD, dysthymic disorder, and depressive disorder not otherwise specified, with the latter two diagnoses comprising 76% of the group. Our findings address a critical gap in the literature by indicating that a number of risk factors identified in previous studies using less representative samples, combining first-onset and recurrent cases, and using participants with a wide range of ages, significantly predict first lifetime onset of MDD in young adulthood—the period of peak incidence for depression.

We entered significant bivariate predictors into a multivariate PR model, and found that female gender, familial loading of mood disorders, and subthreshold depressive symptoms contributed unique variance in predicting first lifetime MDD onset. Surprisingly, a model with only these three variables exhibited relatively good predictive power, with a moderate-to-large effect size (AUC = .69).

Constructing a risk index for screening was not our primary goal. However, it is informative to compare our findings with well-accepted risk indices in medicine. The Framingham Risk Score (FRS) is widely used to predict risk for coronary heart disease on the basis of age, blood pressure, high and low density lipoprotein cholesterol, smoking, and diabetes. In a recent review, the median AUC in 20 studies in which the FRS was adequately applied was . 66 (Tzoulaki, Liberopoulos, & Ioannidis, 2009). This suggests the feasibility of developing simple, clinically useful, composite risk indices for MDD.

We followed participants from ages 19–31. Developmental psychologists have argued that the earlier part of this period (up to approximately age 25) is a distinctive stage referred to as “emerging adulthood” (Arnett, 2007). In our sample, the risk of first lifetime MDD onset was slightly higher before age 25. However, despite the developmental changes between ages 19 and 31, the effects of the predictors did not change significantly over the course of the follow-up.

Although gender was a strong predictor of MDD onset, it did not moderate the effects of other predictors. This is consistent with the few previous studies addressing this issue, most of which examined only a few predictors (Carter & Garber, 2011; De Graaf et al., 2002; Lewinsohn et al., 1999). This suggests that although females are at higher risk, the processes contributing to MDD onset in young adulthood are similar for both genders.

In a 12-year follow-up, Shanahan et al. (2011) reported that predictors of MDD appeared to lose power over time. We did not observe this. The lack of significant Predictor × Time interactions indicates that, at least during the 13-year period that we examined, the power of prognostic variables did not diminish with the passage of time. This is consistent with evidence that these variables predict MDD onset in studies using participants with a broad range of ages and follow-up durations.

This study had a number of strengths. We assessed a wide range of predictors in a large community sample; conducted four waves of semistructured diagnostic assessments over a 15-year period; completed direct assessments of first-degree relatives; and focused on a relatively narrow age range that comprises the period of greatest risk for first lifetime onset

of MDD. However, the study also had limitations. The sample suffered attrition at each assessment and was relatively ethnically homogeneous. Physical and sexual abuse were evaluated retrospectively, and life events were assessed via self-report rather than a semistructured interview. Potentially important predictors, such as neuroticism and cognitive style, were not included. The interval between the assessment of predictors and the onset of MDD was lengthy. Although we found no evidence that the predictors' effects diminished over time, it is conceivable that more frequent or proximal measurement may produce stronger effects for some variables (particularly life events and hassles). Finally, and perhaps most importantly, we could not test theoretically richer and more complex etiological models involving interactions with dynamic (e.g., life stress) risk factors, which require precise information about the timing of events relative to the onset of MDD episodes.

In conclusion, female gender, familial loading of mood disorders, and subthreshold depressive symptoms made unique contributions to predicting first lifetime onset of MDD in young adulthood, the period of peak risk for depression. Collectively, these variables had a medium-to-large effect, suggesting that simple multivariate risk models may be useful in identifying high-risk groups for targeted prevention.

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Table 1
Descriptive Statistics for Predictors (Unweighted)

Predictor	With MDD (*N* = 183)		Without MDD (*N* = 319)	
	Valid *N*	*n*(%)	Valid *N*	*n*(%)
Female	183	123 (67.2)	319	129 (40.4)
Non-white	183	21 (11.5)	319	38 (11.9)
Lives with both biological parents	183	91 (49.7)	319	174 (54.5)
Parents' education level (bachelor's or higher)	179	83 (45.4)	304	150 (47.0)
Anxiety disorder before age 18	183	28 (15.3)	319	21 (6.6)
Disruptive behavior disorder before age 18	183	13 (7.1)	319	30 (9.4)
Substance use disorder before foreage 18	183	39 (21.3)	319	62 (19.4)
		M (*SD*)		*M* (*SD*)
Familial loading mood disorder	169	0.32 (0.31)	284	0.23 (0.27)
Familial loading anxiety disorder	169	0.13 (0.22)	284	0.10 (0.18)
Familial loading substance use disorder	169	0.35 (0.30)	284	0.31 (0.29)
Sexual abuse during childhood	164	7.26 (5.01)	275	6.40 (4.08)
Physical abuse during childhood	165	2.44 (1.74)	277	2.23 (1.68)
Subthreshold depressive symptoms	180	14.00 (9.85)	314	11.29 (8.77)
Suicidal ideation	180	4.59 (1.59)	314	4.55 (1.55)
Physical health	162	3.12 (0.67)	289	3.30 (0.66)
Self-esteem	180	3.24 (0.55)	311	3.35 (0.63)
Stressful life events	181	6.46 (4.36)	313	6.11 (4.34)
Daily hassles	180	45.34 (11.95)	314	43.41 (12.08)
Social support family	181	−0.06 (1.02)	314	0.04 (0.99)
Social support friends	181	0.04 (0.96)	314	−0.02 (1.02)

Note. MDD = major depressive disorder; Valid *N* = number of participants with valid data; *M* = mean; *SD* = standard deviation.

Table 2

Predictors of First Onset of MDD in Young Adulthood

Predictor	β	*SE*	*p*	Hazard ratio [CI_{95}]	R^2
Female	**0.94**	**0.17**	**<.001**	**2.57 [1.84, 3.59]**	.069
Non-White	0.06	0.23	.809	1.06 [0.67, 1.67]	<.001
Lives with both biological parents	−0.09	0.16	.586	0.92 [0.67, 1.25]	.001
Parents' education level	−0.03	0.16	.851	0.97 [0.71, 1.33]	<.001
Anxiety disorder before age 18	**0.63**	**0.21**	**.003**	**1.88 [1.24, 2.83]**	.011
Disruptive behavior disorder before age 18	−0.13	0.31	.684	0.88 [0.48, 1.61]	<.001
Substance use disorder before age 18	0.18	0.20	.384	1.19 [0.80, 1.77]	.001
Familial loading mood disorder	**0.86**	**0.27**	**.002**	**2.37 [1.39, 4.04]**	.023
Familial loading anxiety disorder	0.70	0.38	.069	2.02 [0.95, 4.29]	.007
Familial loading substance use disorder	0.40	0.28	.149	1.49 [0.87, 2.58]	.005
Sexual abuse during childhood	**0.04**	**0.02**	**.007**	**1.04 [1.01, 1.08]**	.014
Physical abuse during childhood	0.06	0.04	.165	1.06 [0.97, 1.16]	.004
Subthreshold depressive symptoms	**0.02**	**0.01**	**.005**	**1.02 [1.01, 1.04]**	.016
Suicidal ideation	<0.01	0.04	.954	1.00 [0.92, 1.10]	<.001
Physical health	**−0.37**	**0.11**	**.001**	**0.69 [0.56, 0.86]**	.021
Self-esteem	−0.18	0.12	.119	0.84 [0.67, 1.05]	.005
Stressful life events	0.01	0.02	.480	1.01 [0.98, 1.05]	.001
Daily hassles	0.01	0.01	.142	1.01 [1.00, 1.02]	.004
Social support family	−0.09	0.09	.271	0.91 [0.77, 1.08]	.003
Social support friends	0.03	0.08	.716	1.03 [0.88, 1.19]	<.001

Note. Statistically significant effects are bolded. *SE* = standard error; CI_{95} = 95% confidence interval; R^2 = generalized R^2 based on the likelihood-ratio statistic (Allison, 1995); MDD = major depressive disorder.