

# Psychosocial Functioning of Young Adults Who Have Experienced and Recovered From Major Depressive Disorder During Adolescence

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The authors examined whether adolescent major depressive disorder (MDD) was associated with difficulties in young adult functioning and whether differences would remain significant after accounting for nonmood disorder, MDD recurrence, functioning in adolescence, or current mood state. A total of 941 participants were assessed twice during adolescence and at age 24. In unadjusted analyses, adolescent MDD was associated with most young adult functioning measures. Associations were not due to interactions with adolescent comorbidity, but differences in global functioning and mental health treatment appeared as a result of MDD recurrence. Accounting for levels of functioning in adolescence or for current depression at age 24 eliminated the remaining associations. The implications of these findings for efforts to prevent MDD in adolescence are discussed.

Epidemiological studies indicate that the risk for developing major depressive disorder (MDD) begins to rise in adolescence and approach adult levels (e.g., Burke, Burke, Regier, & Rae, 1990; Sorenson, Rutter, & Aneshensel, 1991). Thus, the question of what happens to depressed adolescents after they become adults is a significant concern. Recent follow-up studies have indicated that many depressed adolescents experience recurrences in adulthood (Lewinsohn, Rohde, Klein, & Seeley, 1999; Rao, Hammen, & Daley, 1999; Weissman et al., 1999). In addition, there is a growing number of studies indicating that depressed adolescents exhibit significant psychosocial deficits in adulthood, including impaired academic and occupational functioning, early childbearing, social difficulties and poor peer relationships, lowered life satisfaction, increased adversity, increased treatment utilization, criminal arrests, and reduced global functioning (e.g., Aronen & Soininen, 2000; Bardone, Moffitt, Caspi, & Dickson, 1996; Devine, Kempton, & Forehand, 1994; Fergusson & Woodward, 2002; Fleming, Boyle, & Offord, 1993; Gotlib & Hammen, 1992; Ham-

men, 1991; Kandel & Davies, 1986; Kovacs, Akiskal, Gatsonis, & Parrone, 1994; Kovacs & Goldston, 1991; Puig-Antich et al., 1993; Rao et al., 1995; Rao, Weissman, Martin, & Hammond, 1993; Reinherz, Giaconia, Carmola, Wasserman, & Silverman, 1999; Winokur & Tsuang, 1996). However, a number of important questions remain concerning the nature and determinants of the adult psychosocial difficulties exhibited by depressed adolescents. These include the specificity of these deficits to adolescent depression, as opposed to other forms of adolescent psychopathology, and the possibility that a number of third variables might account for the association between adolescent depression and psychosocial difficulties in adulthood.

Childhood externalizing disorders, especially conduct disorder, have been repeatedly shown to be associated with longstanding negative consequences, including adult antisocial behavior, arrests and legal contacts, early pregnancy, spouse abuse, physical aggression, and early mortality (e.g., Bardone et al., 1998; Huesmann, Eron, Lefkowitz, & Walder, 1984; Kovacs, Krol, & Voti, 1994; Laub & Vaillant, 2000; Quinton, Pickles, Maughan, & Rutter, 1993; Zoccolillo & Rogers, 1991). In general, descriptive studies linking psychopathology to psychosocial functioning find that externalizing symptoms have greater long-term predictive significance than internalizing symptoms (e.g., Bardone et al., 1996; Capaldi & Stoolmiller, 1999; Kohlberg, Ricks, & Snarey, 1984; Renouf, Kovacs, & Mukerji, 1997), although more recent research on internalizing disorders has documented that internalizing problems in adolescence are also predictive for risk for problems in young adulthood (including our own research, Lewinsohn et al., 1999). Thus, it is unlikely that difficulties in adult psychosocial functioning are specific to adolescent MDD, although it is possible that the association between adolescent depression and future problems with interpersonal relationships may be particularly strong (e.g., Capaldi & Stoolmiller, 1999; Rao et

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al., 1999). To the extent that meaningful differences in the psychosocial outcomes related to specific disorders are detected, the findings would have important clinical implications, suggesting specific domains that should be targeted for intervention and assessed when evaluating treatment efficacy.

In addition, there are at least four sets of substantive factors that could potentially account for an association between adolescent MDD and deficits in psychosocial functioning in adulthood. The first, following from the issue of specificity indicated previously, is the impact of comorbid nonaffective psychopathology in adolescence. That is, the association may be evident only among depressed adolescents with comorbid disorders. As is now well known, many depressed adolescents have comorbid mental disorders (e.g., Angold & Costello, 1992; Rohde, Lewinsohn, & Seeley, 1991). However, less is known regarding the impact of comorbidity on future functioning. Fombonne, Wostear, Cooper, Harrington, and Rutter (2001) recently reported that among individuals with childhood depression, the subgroup with comorbid conduct disorder had higher suicide attempt rates, criminal offenses, and more pervasive social dysfunction in adulthood. Unfortunately, a nonpsychiatric control group was not included, hence it is unclear if the noncomorbid subgroup also exhibited elevated, albeit less extreme, dysfunction as adults. It is likely that comorbid nonaffective disorders moderate the impact of adolescent depression on adult psychosocial functioning, with comorbidity predicting poorer outcomes (but see Capaldi & Stoolmiller, 1999, for negative findings). However, it is also likely that depressed adolescents without comorbid nonaffective psychopathology experience greater difficulties in adult psychosocial functioning than individuals with no history of psychopathology in adolescence.

A second alternative explanation for an association between adolescent MDD and young adult functioning is that many formerly depressed adolescents experience a recurrence of their depression or the occurrence of another mental disorder as adults (e.g., Lewinsohn et al., 1999; Weissman et al., 1999). It is possible that experiencing the recurrent MDD or other psychopathology during adulthood accounts for the problems in adult psychosocial functioning associated with adolescent MDD (e.g., Geller, Zimmerman, Williams, Bolhofner, & Craney, 2001). However, we have found that formerly depressed adolescents and adults developed residual effects, or "scars," which were not detected prior to the onset of their first depressive episode (Rohde, Lewinsohn, & Seeley, 1990, 1994). Therefore, even though participants with recurrent MDD and other mental disorders during adulthood may account for much of the association between adolescent depression and difficulties in adult social functioning, formerly depressed participants who do not experience MDD or additional psychopathology in adulthood may also show some evidence of impaired functioning.

A third explanation is the possibility that the reduced psychosocial functioning observed in adulthood was actually already present in adolescence. Thus, the adult psychosocial deficits observed in depressed adolescents may reflect continuities in social functioning, some of which may have preceded, and perhaps even contributed to, adolescent MDD.

A fourth potential explanation is the presence of depressive symptoms at the time that adult social functioning is assessed. Previous research (e.g., Judd, Akiskal, et al., 1998, 2000; Lewin-

sohn, Solomon, Seeley, & Zeiss, 2000) has indicated that sub-threshold depressive symptoms are associated with significant levels of psychosocial impairment. Given that adults who were depressed as adolescents are likely to experience periods of sub-threshold symptoms outside of, or without ever experiencing, recurrences of MDD, this may contribute to their deficits in functioning as adults.

In this article, we use data from the Oregon Adolescent Depression Project (OADP; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Lewinsohn et al., 1999), in which a large cohort of individuals was assessed twice in adolescence and again as young adults, to examine the extent to which the psychosocial functioning of young adults who experienced MDD during adolescence is impaired relative to young adults who did not experience adolescent MDD. This article is the third in a series of reports on the young adult outcomes associated with an episode of MDD during adolescence. In the first article (Lewinsohn et al., 1999), we examined the psychiatric consequences of experiencing an episode of MDD prior to age 19. Significantly more participants who were depressed as adolescents developed an episode of MDD between 19 and 23 years of age (45.0%), compared with adolescents with either no disorder or a nonmood disorder (18.5% and 28.2%, respectively). In addition, compared with those with no diagnosable psychopathology during adolescence, formerly depressed adolescents were more likely to experience a nonmood disorder during young adulthood (33.2% vs. 19.5%). In the second article (Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2000), we identified factors from adolescence that predicted recurrence of MDD by young adulthood. Significant predictors of MDD recurrence included characteristics of the adolescent (e.g., elevated depressive symptoms, excessive emotional reliance on others, lower social competence, daily hassles), characteristics of the adolescent MDD episode (e.g., longer duration, multiple episodes, severity, history of suicide attempt), and having a higher proportion of family members with MDD. In the present article, our focus shifts from predicting psychopathology in young adulthood to examining psychosocial functioning during this age period. After describing the unadjusted associations between adolescent MDD and young adult psychosocial functioning, we control for demographic differences and four variables that could potentially account for the impact of adolescent MDD: (a) psychiatric comorbidity in adolescence (i.e., Are differences in young adult functioning evident only in depressed adolescents with comorbid nonmood disorders?), (b) MDD recurrence and the occurrence of nonmood disorders in young adulthood (i.e., Are differences in young adult functioning evident only in formerly depressed adolescents who experience MDD recurrence or other disorders in young adulthood?), (c) psychosocial functioning levels in adolescence (i.e., Are differences in young adult functioning already evident in adolescence?), and (d) subthreshold depression symptoms in young adulthood (i.e., Are differences in young adult functioning evident after controlling for current depression level at the time of the young adult assessment?). In addition, we examine the specificity of the association between adolescent depression and difficulties in young adult psychosocial functioning by comparing depressed adolescents to adolescents with nonaffective psychopathology. Finally, we explore whether comorbid, nonaffective disorders in adolescence moderate the effects of adolescent depression on young adult functioning.

The term *psychosocial functioning* is similar in many respects to the concept of “competence,” which includes a variety of constructs related to a person’s success in reaching the developmental tasks expected of individuals at a given age and cultural context (Masten & Coatsworth, 1995). Masten and Curtis (2000) described several models to account for an association between competence and psychopathology (e.g., the association is a methodological artifact, competence problems lead to psychopathology, psychopathology undermines an individual’s competence and adaptive behavior, shared risk factors). Although the present study was not designed to test these models, it is grounded in the premise that adolescent MDD has an adverse impact on developing competencies, leading to difficulties in young adult psychosocial functioning.

Young adult psychosocial functioning (YAPF) in the present article is broadly defined to include measures of academic and occupational performance, marital and parenting status, income level, quality of relations with family and peers, adversity, life satisfaction, mental health utilization, and general physical health (including cigarette smoking). The measure of adversity consists of major and minor stressors. While stress generally has been examined as a risk factor for onset of psychopathology, Hammen (1991) expanded the role of stress in depression, proposing processes by which depressed individuals play a role in generating higher levels of interpersonal stressful life events, which in turn contribute to depression recurrences; this effect may be particularly salient for depressed individuals with comorbid psychopathology (Daley et al., 1997). While we do not know how much these measures of major and minor adversity are the result of the person’s functioning (i.e., the extent to which they are dependent or independent of the person’s behavior), the measures of adversity clearly represent an important negative aspect of the person’s environment.

Throughout the analyses, we address the impact of demographic factors. Relative to adolescent comparison groups, OADP participants with adolescent MDD were more likely to be female adolescents, had parents with less education, and were less likely to reside with both biological parents in high school (Lewinsohn et al., 1999). Consequently, we control for these demographic variables in the analyses. Given the important association between depression and female gender (e.g., Nolen-Hoeksema, 2002), and the fact that depression levels may be more strongly associated with future psychological maladjustment for young women compared with young men (Gjerde & Westenberg, 1998), we also examine gender as a potential moderator of the relations between depression during adolescence and YAPF.

## Method

### *Participants and Procedures*

A concerted effort was made to assemble a representative sample of community-residing adolescents for the T<sub>1</sub> OADP sample. The population for this sample was the total enrollment (approximately 10,200) of nine high schools in two urban and three rural communities in west central Oregon. Sampling within each school was proportional to size of school, size of grade within school, and sex within grade. Three cohorts were recruited between 1987 and 1989 for a total T<sub>1</sub> sample size of 1,709, with participation rates of 52%, 62%, and 68% (61% overall). Primary reasons for nonparticipation were adolescent disinterest (72%), consideration of the

assessment as too personal (12%), and parent refusal (12%). For the first cohort of T<sub>1</sub> assessments, we insisted on scheduling adolescents and parents for the T<sub>1</sub> diagnostic interview at the same time. This proved to be difficult for the family and resulted in an unacceptably low participation rate. On the basis of findings from our study, as well as others indicating that parents of depressed adolescents were not well informed of the depression symptoms of their adolescent children (e.g., Cantwell, Lewinsohn, Rohde, & Seeley, 1997), we dropped the requirement of simultaneous parental report for the remainder of T<sub>1</sub> assessments. This substantially increased our participation rate for the second and third T<sub>1</sub> cohorts. Approximately 1 year later (T<sub>2</sub>), 1,507 (88%) of the T<sub>1</sub> participants returned for a re-administration of the interview and questionnaire (mean T<sub>1</sub>–T<sub>2</sub> interval = 13.8 months, *SD* = 2.3).

Three checks were made to determine representativeness of the OADP T<sub>1</sub> sample: (a) Demographic characteristics of the sample were compared with 1980 census data, (b) participants were compared on demographic information with adolescents who declined, and (c) 100 participants who initially refused were assessed by offering them an increased monetary inducement of \$100. Differences were minimal: Decliners were less likely to be from two-parent families (74% vs. 66%) and had a lower average socioeconomic status, although both groups represented the middle class. Nonparticipants did not differ from participants on type or number of current and lifetime diagnoses, number or extent of clinical symptoms, race, current employment status of parents, and questionnaire variables. Overall, the analyses indicated that the participants in our sample may be considered to be representative of high school students in western Oregon.

Small but statistically significant differences were noted between adolescents who did not participate at T<sub>2</sub> (*n* = 202) and those who did (*n* = 1,507) on T<sub>1</sub> variables. Attrition was associated with lower parental socioeconomic status, smaller household size, being male adolescents (54% of attriters vs. 46% of T<sub>1</sub>–T<sub>2</sub> panel), lower educational level of the parents (whether one or both parents had completed college), history of disruptive behavior disorders (17% vs. 11%), and (for male adolescents only) history of substance use disorders (26% vs. 14%). Importantly, the two groups did not differ on most measures of psychopathology; that is, episodes of current and past disorders including depression and anxiety, self-report depression measures, number of suicide attempts, race, and grade level.

A third wave of questionnaire and diagnostic interview assessments (T<sub>3</sub>) was conducted between 1994 and 1999, with a selected subset of T<sub>2</sub> participants after individuals reached their 24th birthday. On the basis of T<sub>1</sub>–T<sub>2</sub> diagnostic information, three groups were selected for a T<sub>3</sub> diagnostic interview: (a) 351 participants with a T<sub>2</sub> lifetime history of MDD, (b) 293 participants with a T<sub>2</sub> history of nonaffective disorder, and (c) 457 participants with no history of mental disorder at T<sub>2</sub>. The participants with no history of mental disorder were randomly selected from the 863 T<sub>2</sub> participants with no disorder (although all non-White T<sub>2</sub> participants were retained in the sample to enhance diversity of the T<sub>3</sub> group). Of the 1,101 participants who were eligible for the T<sub>3</sub> interviews, 1,025 (93%) returned their mailer questionnaire, which was collected prior to the diagnostic interview, and 941 (85%) completed their T<sub>3</sub> telephone diagnostic interview. While the T<sub>1</sub> and T<sub>2</sub> interviews were conducted in a face-to-face format, we shifted to a telephone assessment format for the T<sub>3</sub> diagnostic assessments because a significant proportion of the participants no longer resided in the area and telephone interviewing was seen as less demanding on the participants. As part of the T<sub>3</sub> assessment, 60 participants were interviewed face-to-face and over the telephone regarding Axis I disorders, and an additional 60 participants were interviewed twice regarding all Axis II disorders (Rohde, Lewinsohn, & Seeley, 1997). Overall, the assessment formats were found to be quite comparable, with excellent agreement for anxiety disorder ( $\kappa = .84$ ) and very good agreement levels for (nonalcohol) substance use disorders, alcohol use disorders, and MDD ( $\kappa_s = .73, .70,$  and  $.67$ , respectively).



Written informed consent was obtained from OADP participants (and their guardians, if applicable) to conduct all assessments.

Of the 941  $T_3$  participants, 539 (57%) were women and 402 (43%) were men. Average age at  $T_3$  was 24.2 years ( $SD = 0.6$ ). Most participants were White (89%), with 1% Black, 3% Hispanic, 3% American Indian, 3% Asian, and 2% "other." The majority (61%) were single, with 34% married, 2% separated, and 3% divorced. Almost all (97%) had graduated from high school or received their general educational development diploma, and 31% had received a bachelors degree or higher. The mean time between the  $T_2$  and  $T_3$  assessments was 6.8 years ( $SD = 1.4$ ). Although women were more likely than men to complete the  $T_3$  assessments (89% vs. 81%),  $\chi^2(1, N = 1,101) = 13.55, p < .001$ ,  $T_3$  participation differences as a function of other demographic variables or  $T_2$  diagnostic status were nonsignificant. The mean time interval between the mailer questionnaire assessment and the  $T_3$  interview was 6.4 months ( $SD = 8.8$ ). Given our focus on functioning near the time of the 24th birthday, participants who had not completed a mailer questionnaire during the 23–25 age interval ( $n = 24$ ) were excluded from the analyses.

### Diagnostic Interviews

Participants were interviewed at  $T_1$  with a version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS), which combined features of the Epidemiologic version (Orvaschel, Puig-Antich, Chambers, Tabrizi, & Johnson, 1982) and the Present Episode version, and included additional items to derive *Diagnostic and Statistical Manual of Mental Disorders* diagnoses (*DSM-III-R*; 3rd ed., rev.; American Psychiatric Association [APA], 1987). At  $T_2$  and  $T_3$ , participants were interviewed using the Longitudinal Interval Follow-Up Evaluation (LIFE; Keller et al., 1987), which elicited detailed information about the course of psychiatric symptoms and disorders since the previous interview.  $T_3$  diagnoses were made using *DSM-IV* (4th ed.; APA, 1994) criteria. Based on a second interviewer independently scoring the taped assessments of a randomly selected subsample at  $T_1$  and  $T_2$  ( $n = 233$ ), interrater reliability values for lifetime diagnoses, as evaluated by the kappa statistic, were moderate to excellent: MDD  $\kappa = .86$ ,  $\kappa$ s for specific nonmood disorders ranged from .76 to .89. For the  $T_2$ – $T_3$  period ( $n = 178$ ), interrater reliability was excellent for the primary categories of MDD ( $\kappa = .87$ ) and nonmood disorder ( $\kappa = .82$ ).

### Outcome: YAPF

YAPF was assessed either in the mailer questionnaire or as part of the  $T_3$  interview using both dichotomous and continuous measures. Dichotomous measures included *marital status* (currently married: yes/no), *divorce or separation* (assessed only among participants who had been married), *parenting* (whether participant had ever been a parent: yes/no), *mental health utilization* (whether participant had seen a mental health professional in the past 12 months: yes/no), and current *daily smoking* (yes/no).

Continuous measures consisted of *global level of functioning* (*DSM-III-R/DSM-IV*); number of *years of school completed*; number of *weeks unemployed in past year*; *annual household income*; *quality of relationship with family members* ( $\alpha = .88$ ; 10 items; Procidano & Heller, 1983); *quality of relationship with friends* ( $\alpha = .88$ ; 10 items; Procidano & Heller, 1983); *social network* ( $\alpha = .69$ ; size and frequency of social contact; 3 items; Berkman & Syme, 1979); *minor hassles* ( $\alpha = .88$ ; daily hassles during the last 4 weeks, as assessed by 20 items from the Unpleasant Events Schedule; Lewinsohn, Mermelstein, Alexander, & MacPhillamy, 1985); *major adversity* ( $\alpha = .71$ ; major life events in past 12 months, as assessed by 33 events occurring to the participant, based on the Social Readjustment Rating Scale, Holmes & Rahe, 1967, and the Psychiatric Epidemiology Research Inventory; Dohrenwend, Levav, & Shrout, 1986); *physical health* ( $\alpha = .50$ ; 4 items assessing self-rated health, number of times received treatment in past year, treatment for illness or injury in past

year, chronic medical problems distress); and *life satisfaction* ( $\alpha = .89$ ; 15 items chosen from Andrews & Withey, 1976; Campbell, Converse, & Rodgers, 1976).

Eight of these variables had also been assessed at  $T_1$ , at which we attempted to assess all of the psychological constructs deemed important for the etiology of depression. Most of the measures assessed at  $T_1$  have been previously shown to be risk factors for depression in adolescence (Lewinsohn et al., 1994). In the present study, we were able to control for  $T_1$  levels of the following variables: global level of functioning, quality of relationship with family members, quality of relationship with friends, social network, minor adversity, major adversity, physical health, and daily smoking.

Lastly, to account for the impact of depression symptoms at the time of the young adult assessment, scores on the 20-item Center for Epidemiologic Studies—Depression Scale assessed in the mailer questionnaire preceding  $T_3$  were entered into the analyses ( $\alpha = .89$ ; CES-D; Radloff, 1977).

### Participant Groups

The 917 individuals who completed a mailer questionnaire and the  $T_3$  diagnostic interview between the ages of 23 and 25 form the reference sample for the present study. Data from their  $T_1$ ,  $T_2$ , and  $T_3$  interviews were combined to create a longitudinal record of each participant's psychiatric history up to age 24. Disorders that had onset before age 19 (which occurred in 527 participants) were considered to have an adolescent onset; disorders with onset between ages 19 and 23 (which occurred in 428 participants) were considered to have occurred in young adulthood. Of these participants, 319 experienced an episode of MDD by age 19 (175 with a lifetime comorbid adolescent, nonmood disorder): 209 (66%) experienced their first MDD episode before  $T_1$ , 31 (10%) were depressed at  $T_1$ , and 79 (25%) experienced their first MDD after  $T_1$  but before age 19. Two other groups were created: An additional 208 participants had an adolescent nonmood disorder (without MDD) and the remaining 324 had experienced no mental disorder by age 19. The adolescent nonmood disorders occurring in the sample included anxiety disorders ( $n = 155$ ), alcohol abuse/dependence ( $n = 151$ ), drug abuse/dependence ( $n = 146$ ), attention deficit hyperactivity disorder/disruptive behavior disorders ( $n = 94$ ), and eating disorders ( $n = 20$ ).

To articulate more clearly differences associated with adolescent depression, participants with a history of bipolar disorder ( $n = 18$ ), dysthymia without a lifetime history of MDD ( $n = 15$ ), psychotic disorder ( $n = 4$ ), and adjustment disorder with depressed mood ( $n = 53$ ), as well as those who had not recovered from MDD by age 19 ( $n = 22$ ), were excluded from the present analyses.

To better understand various characteristics of the sample, we compared participants with pure MDD versus comorbid adolescent MDD on MDD recurrence, MDD onset age, and total duration of MDD in adolescence. Differences between the two groups on recurrence in adolescence were nonsignificant (20.1% of pure MDD vs. 27.4% of comorbid MDD). However, compared with the pure MDD group, the comorbid MDD group had a significantly earlier onset of first MDD (175.6 vs. 185.2 months),  $t(317) = 2.48, p < .05$ , and a higher total duration of MDD between ages of 0–18 (46.8 vs. 21.1 weeks),  $t(317) = 3.32, p < .001$ . Participants were also compared on the total number of mental disorders (including MDD) by age 19. The mean number of disorders for the pure and comorbid MDD groups were 1.0 ( $SD = 0.1$ ; 4 participants had comorbid dysthymia) and 3.4 ( $SD = 1.3$ ), respectively. The pure nonmood disorder group had an average of 1.9 ( $SD = 1.0$ ) disorders.

Of the 851  $T_3$  participants in the present study, 27 (3.2%) had current MDD at the time of the  $T_3$  interview and 90 (10.6%) had current nonmood disorder (7 of these individuals had both current MDD and nonmood disorder at the  $T_3$  assessment).

Data Analysis

The four adolescent diagnostic groups were compared first on demographic variables assessed at T<sub>1</sub>; subsequent analyses were adjusted for demographic characteristics that were found to be significantly associated with adolescent diagnostic group status. Bivariate unadjusted associations between the YAPF measures and a history of MDD or nonmood disorder by age 18 were examined with point biserial correlations for continuous YAPF measures and odds ratios for dichotomous YAPF measures. Hierarchical logistic and linear regression models were used to examine the primary issues described previously. For each regression model, variables were entered into four blocks. The first block included the significant demographic measures and the two independent variables (i.e., MDD before age 18 and nonmood disorder before age 18). To account for the effects of psychopathology during the 19-to-23 age period, dichotomous measures indicating the presence or absence of MDD and nonmood disorder during this age period were entered into the regression equations in the second block. For the eight measures of YAPF also assessed at T<sub>1</sub>, the third block included the T<sub>1</sub> measure of functioning in order to examine the impact of the stability of YAPF measure. Finally, the fourth block included the CES-D assessed at T<sub>3</sub> in order to examine the role of current depressive symptomatology at the time of the young adult assessment. Variables in the earlier blocks were retained in subsequent blocks (e.g., in addition to T<sub>3</sub> CES-D scores, Block 4 was adjusted for adolescent demographic and psychopathology variables, young adult psychopathology, and T<sub>1</sub> levels of

the functioning). Given the number of outcomes examined, alpha was set to  $p < .01$  to adjust for inflation of Type I error.

Results

Group Differences on Adolescent Demographic Variables

For descriptive purposes, the means or percentages for the T<sub>1</sub> demographic characteristics and the T<sub>3</sub> YAPF measures are presented in Table 1 for the four adolescent diagnostic groups. To examine main effects of adolescent MDD or interactions between adolescent MDD and nonmood disorder, a series of 2 × 2 analyses of variance or multiple logistic regressions was computed. Significant T<sub>1</sub> demographic differences as a function of adolescent MDD were obtained for female sex, Wald(1) = 54.55,  $p < .001$ , not residing with both biological parents, Wald(1) = 17.25,  $p < .001$ , and lower parental education, Wald(1) = 9.49,  $p < .01$ .

Main effects of adolescent MDD were nonsignificant for age,  $F(1, 847) = 1.78$ , and for race/ethnicity, Wald(1) = .03, both  $ps > .05$ . All interactions between adolescent MDD and adolescent nonmood disorder were nonsignificant. Given the significant associations with sex, residing with both biological parents, and parental education, subsequent analyses included these three variables as covariates.

Table 1  
Psychosocial Functioning During Young Adulthood (Age 24) by Adolescent Diagnostic Group

Variable	Diagnostic group (before age 19)			
	ND (N = 324)	NMD (N = 208)	MDD (N = 144)	MDD+ (N = 175)
Time 1 demographic characteristics				
Female (%)	49.4	46.2	78.5	70.3
White (%)	88.0	89.9	88.9	89.7
Mean age in years (SD)	16.7 (1.3)	16.5 (1.2)	16.8 (1.1)	16.5 (1.1)
Parental education (% with bachelor's degree)	44.4	48.1	36.8	33.7
Reside with both biological parents (%)	64.5	54.8	48.6	41.1
Continuous-type outcomes				
Mean global functioning (SD)	77.3 (9.7)	71.4 (11.6)	72.5 (10.8)	68.0 (12.8)
Mean years of education (SD)	14.5 (1.8)	13.7 (1.9)	14.1 (1.8)	13.3 (1.7)
Unemployment past year (in weeks)	5.7 (11.9)	7.7 (12.9)	8.5 (14.6)	9.7 (14.7)
Household income (per 10,000)	2.8 (1.5)	2.6 (1.5)	2.6 (1.5)	2.4 (1.4)
Quality relationship				
Family	0.13 (0.95)	0.05 (1.00)	0.04 (0.89)	0.24 (1.15)
Friends	0.04 (1.01)	-0.01 (0.93)	0.04 (1.03)	0.06 (1.05)
Small social network	0.20 (0.99)	0.06 (0.85)	0.09 (1.01)	0.28 (1.10)
Minor hassles	-0.20 (0.97)	0.08 (1.03)	0.02 (0.94)	0.30 (1.00)
Major adversities	-0.22 (0.86)	0.09 (1.09)	0.17 (1.07)	0.21 (1.00)
Poor physical health	-0.23 (0.91)	0.07 (1.03)	0.10 (0.95)	0.31 (1.09)
Low life satisfaction	-0.21 (1.00)	-0.02 (0.95)	0.27 (1.00)	0.22 (0.96)
Dichotomous outcome				
Married (%)	37.0	31.4	36.1	45.1
Divorced (of % those married)	0.8	6.6	4.2	4.3
Parenting (%)	19.0	18.2	23.7	36.8
Mental health utilization (%)	12.3	18.7	19.5	28.9
Daily smoking (%)	13.3	25.8	18.0	28.8

Note. ND = no disorder; NMD = nonmood disorder; MDD = noncomorbid major depressive disorder; MDD+ = comorbid major depression.

*YAPF as a Function of a History of Adolescent Depression*

The unadjusted associations (correlations or odds ratios) between the YAPF measures and adolescent MDD and nonmood disorder are presented in the first two columns of Table 2. As can be seen, all of the YAPF measures were associated with adolescent MDD, with the exception of household income, quality of relationships with friends, marital status, and divorce. After accounting for associations with adolescent nonmood disorder and the three significant demographic characteristics (i.e., Block 1), the following seven YAPF measures remained significantly associated with adolescent MDD: low global functioning, low quality of relations with family, small social network, minor hassles, major

adversity, low life satisfaction, and mental health treatment utilization.

*YAPF in Formerly Depressed Adolescents Who Remain Well Between 19 and 23 Years of Age*

To determine whether the associations between adolescent depression and the YAPF measures were significant after accounting for psychopathology during young adulthood, MDD and nonmood disorder during the 19-to-23 age period were entered as the second block of variables in the regression equations (see Block 2 in Table 2). As can be seen, of the seven significant YAPF measures identified in Block 1, low global functioning and mental health utilization became nonsignificant. Global level of functioning

Table 2  
Summary of Unadjusted and Adjusted Associations Between Major Depression and Nonmood Disorders by Age 18 and Measures of Functioning at Age 24

Measure of functioning	Unadjusted associations (rs)		Adjusted association (rs)							
			Block 1 <sup>a</sup>		Block 2 <sup>a, b</sup>		Block 3 <sup>a, b, c</sup>		Block 4 <sup>a, b, c, d</sup>	
	MDD	NMD	MDD	NMD	MDD	NMD	MDD	NMD	MDD	NMD
Continuous-type variables										
Lower global functioning	.20**	.25**	.13**	.22**	.06	.12**	.05	.11**	.02	.10**
Less education	.14**	.22**	.06	.18**	.05	.15**			.04	.15**
Unemployed past year	.11*	.10*	.08	.07	.05	.02			.05	.02
Lower income	.08	.08	.07	.05	.05	.02			.03	.02
Low quality relations										
Family	.09*	.13**	.09*	.10*	.09*	.08	.05	.05	.03	.04
Friends	.04	.02	.08	.00	.08	.01	.05	-.01	.03	-.01
Small social network	.14**	.13**	.10*	.11*	.09*	.09*	.09*	.09*	.06	.08*
Minor hassles	.13**	.17**	.12**	.14**	.09*	.09*	.04	.04	-.02	.03
Major adversity	.16**	.16**	.13**	.12**	.09*	.04	.07	.02	.04	.01
Poor physical health	.18**	.17**	.06*	.15**	.04	.11**	.02	.10*	.01	.09*
Low life satisfaction	.16**	.08	.16**	.04	.12**	.00			.05	-.02
Dichotomous variable										
Ever married OR	1.24	0.97	1.06	0.98	1.15	1.25			1.22	1.28
99% CI	0.86-1.79	0.68-1.39	0.71-1.58	0.67-1.42	0.77-1.74	0.83-1.88			0.81-1.86	0.84-1.93
Divorced or separated OR	1.38	2.56	1.00	2.85	0.94	2.47			1.12	2.58
99% CI	0.28-6.78	0.50-13.23	0.18-5.48	0.51-16.04	0.17-5.34	0.38-15.91			0.19-6.59	0.40-16.68
Parenthood OR	1.91**	1.41	1.39	1.31	1.37	1.43			1.36	1.43
99% CI	1.26-2.88	0.94-2.12	0.89-2.17	0.85-2.01	0.87-2.16	0.90-2.29			0.86-2.16	0.89-2.29
Mental health utilization OR	2.10**	2.12**	1.78*	1.92**	1.49	1.38			1.27	1.32
99% CI	1.35-3.28	1.36-3.31	1.11-2.87	1.22-3.03	0.91-2.44	0.84-2.28			0.76-2.11	0.79-2.20
Daily smoking OR	1.72*	2.36**	1.55	2.13**	1.46	1.79*	1.40	1.56	1.36	1.55
99% CI	1.11-2.68	1.52-3.67	0.96-2.53	1.34-3.36	0.89-2.40	1.09-2.96	0.85-2.31	0.93-2.61	0.82-2.25	0.93-2.59

Note. Semipartial correlations are presented for the adjusted associations for continuous-type functioning measures. MDD = major depressive disorder by age 18; NMD = nonmood disorder; OR = odds ratio; CI = confidence interval.

<sup>a</sup> Adjusted for NMD or MDD before age 19 years, gender, parental education, and residing with both biological parents. <sup>b</sup> Adjusted for MDD and NMD during the 19-23 age period. <sup>c</sup> Adjusted for same measure of functioning at Time 1 (where available). <sup>d</sup> Adjusted for Center for Epidemiologic Studies—Depression scale at age 24.

\*  $P < .01$ . \*\*  $P < .001$ .

scores for formerly depressed adolescents with no recurrence averaged 74.5 ( $SD = 9.4$ ) versus 65.8 ( $SD = 11.7$ ) for formerly depressed adolescents with MDD recurrence between 19 and 23; recent mental health treatment utilization rates for the two groups were 17.5% versus 33.6%, respectively. Thus, after adjusting for the occurrence of young adult psychopathology, the following five YAPF measures remained significantly associated with adolescent MDD: low quality relationships with family, small social network, minor hassles, major adversity, and low life satisfaction.

### *Specificity of YAPSF With Adolescent Depression*

To examine the specificity of the associations between adolescent MDD and nonmood disorder and the YAPF measures, differences between the magnitude of the correlation coefficients were tested using (a) Fisher's  $r$ -to- $z$  transformation for continuous measures and (b) confidence intervals for the odds ratios for dichotomous measures. After adjusting for demographic differences and the presence of other adolescent and young adult psychopathology (i.e., Block 2 in Table 2), one variable—life satisfaction—was more strongly associated with adolescent MDD and adolescent nonmood disorder (.12 vs. .00;  $z = 3.65$ ,  $p < .001$ ). Two other variables had a stronger association with adolescent nonmood disorder than MDD: (a) fewer years of education (.15 vs. .05;  $z = 3.06$ ,  $p < .01$ ) and (b) poor physical health (.11 vs. .04;  $z = 2.13$ ,  $p < .05$ ).

### *Impact of Adolescent Comorbidity on YAPF*

For each of the YAPF regression models, an interaction term between adolescent MDD and nonmood disorder was considered for entry into the model after the main effects based on the change in  $F$  test for linear models and the improvement in chi-square test for logistic models. No significant interactions were obtained for any of the models. Thus, a multiplicative effect of comorbidity between adolescent MDD and nonmood disorder was not supported for any of the YAPF measures.

### *Adjusting for the Stability of Psychosocial Functioning*

The associations between adolescent depression and the YAPF measures may have resulted from the stability of psychosocial functioning from adolescence to young adulthood. Because 8 of the 16 YAPF measures had also been administered at  $T_1$ , we were able to address this issue by including the same measure of functioning assessed during adolescence as a covariate in the third block of the regression equations. As can be seen in Block 3 of Table 2, the associations between MDD and low-quality family relations, minor hassles, and major adversity became nonsignificant after adjusting for  $T_1$  level of the psychosocial measure. The association between adolescent MDD and social network at  $T_3$  remained significant after controlling for the  $T_1$  measure.

### *Adjusting for Depressive Symptomatology at the Time of the Young Adult Assessment*

The associations between adolescent depression and the YAPF measures may have also resulted from residual depressive symptomatology at the time of the last assessment. As expected, participants with a history of adolescent MDD had significantly

higher depression levels at  $T_3$  compared with participants with no history of adolescent MDD, with mean (standard deviation) CES-D scores of 16.8 (10.8) versus 11.4 (9.2), respectively,  $t(588) = 77.42$ ,  $p < .001$ . Given these differences, the CES-D score assessed at  $T_3$  was entered into each regression equation as the fourth block. As can be seen in Table 2 (Block 4), after adjusting for current depression symptoms, none of the associations with MDD remained significant. By contrast, lower global functioning, lower years of education, small social network size, and poor physical health remained associated with nonmood disorder.

## Discussion

Given the developmental demands required of adolescents as they enter young adulthood, our first objective in the present study was to document the degree to which experiencing an episode of MDD during adolescence was associated with detectable differences in young adult functioning. Our second objective was to test the specificity of associations between adolescent MDD and difficulties in young adult functioning. Finally, our third objective was to examine the extent to which four categories of salient variables (e.g., psychiatric comorbidity, MDD recurrence, stability of functioning, current mood state) might account for any differences in young adult functioning that appear to be related to adolescent MDD.

Without adjusting for any of the relevant covariates, young adults who had experienced an episode of MDD during adolescence exhibited pervasive impairments across numerous domains of psychosocial functioning, including occupational performance, interpersonal functioning, quality of life, and physical well-being, although the effect sizes for each of these differences were generally small in magnitude. These findings replicate previous results indicating that young adults with a history of adolescent depression show numerous difficulties in psychosocial functioning (e.g., Brook, Whiteman, Finch, & Cohen, 1996; Fergusson & Woodward, 2002; Geller et al., 2001; Kandel & Davies, 1986; Rao et al., 1999; Reinherz et al., 1999; Weissman et al., 1999).

When we examined the specificity of these associations, however, only one measure of young adult functioning emerged as uniquely associated with adolescent MDD: reduced life satisfaction. These results suggest that experiencing depression in adolescence may be associated with subsequent and enduring reductions in life satisfaction, although the causal nature of this association remains to be elucidated (e.g., depression may lead to a longstanding decline in life satisfaction; dissatisfaction may precede, and perhaps play a causal role, in the development of depression; or both depression and low life satisfaction may be the result of a third factor, such as environmental stress, childhood abuse, or personality traits such as neuroticism).

The remaining associations of adolescent MDD with young adult functioning were not stronger than comparable associations between adolescent nonmood disorder and future functioning. Thus, many of the impairments in young adulthood are associated with the occurrence of adolescent psychopathology more broadly. A shortcoming of the present study is that we aggregated diverse psychopathologies into a single nonmood disorder category and,



thus, could not examine associations with specific nonmood disorders. Two studies using a New Zealand birth cohort (Bardone et al., 1996, 1998) examined similar issues in a longitudinal sample of young women assessed at ages 15 and 21, comparing participants with adolescent depression to those with a history of conduct disorder. The latter diagnosis tended to be associated with more difficulties in young adulthood than a history of depression, including illegal activity, welfare dependency, multiple cohabitation partners, domestic violence, acquisition of sexually transmitted diseases, and poor self-reported health. Two young adult functioning measures in our study (lower educational attainment and poorer physical health) were found to be more strongly associated with adolescent nonmood disorder than with adolescent MDD. Clearly, it would be informative to identify the psychosocial concomitants in young adulthood that may be associated with specific nonmood disorders occurring during adolescence.

Probably the most unique feature of our study is that we were able to examine the impact of alternative factors that might help explain the link between adolescent MDD and subsequent functioning. When we accounted for the associations with demographics and the presence of adolescent nonmood disorders (i.e., Block 1), adolescent MDD was still significantly associated with seven measures of young adult psychosocial functioning: poorer global functioning, lower quality of relationships with family, a smaller social network, greater major and minor adversity, lower life satisfaction, and greater mental health treatment utilization. However, five measures of young adult psychosocial functioning that had initially been associated with adolescent MDD became nonsignificant: years of education, recent unemployment, physical health, parenting status, and daily smoking. Along with the fact that four other variables (i.e., income level, quality of relations with friends, marital status, and divorce/separation) were not associated with adolescent MDD in the univariate analyses, these findings indicate that the specific impacts of adolescent depression on academic, occupational, and marital functioning as well as on parenting status, are relatively weak or nonexistent. However, as the participants were only 24 years of age at the  $T_2$  assessment, they had limited employment and marital histories. Thus, it is possible that adolescent MDD may be associated with greater differences in these areas at a later age.

A few studies have found adolescent psychopathology to be associated with early marriage (e.g., Forthofer, Kessler, Story, & Gotlib, 1996; Gotlib, Lewinsohn, & Seeley, 1998). However, we did not find differences in marital status as a function of adolescent psychopathology. Given the discrepant results, it is possible that the previously reported findings apply only to those who marry at a very early age (age at marriage was not included in our analyses).

Consistent with other studies (Bardone et al., 1996; Fergusson & Woodward, 2002), we found that being a parent by age 24 was associated with adolescent MDD. This effect became nonsignificant, however, after accounting for demographic variables and the presence of adolescent nonmood disorder. Post hoc analyses indicated that controlling for differences in the proportion of women and years of education eliminated the apparent association between adolescent MDD and parenthood.

We also explored whether the effects of adolescent MDD were moderated by comorbid, nonaffective psychopathology in adolescence. This did not appear to be the case: None of the interactions

between adolescent MDD and adolescent nonmood disorder accounted for additional variance in the examined associations.

After controlling for the second covariate of our study, MDD recurrence and the presence of nonmood disorders between the ages of 19 and 23, two of the seven previously significant associations—global functioning and mental health treatment utilization—became nonsignificant. These differences probably reflect the fact that individuals with recent episodes of psychopathology are more likely to experience impairment and seek treatment.

However, even after accounting for the effects of MDD recurrence and the presence of nonmood disorders in both adolescence and young adulthood, differences in five measures of young adult functioning continued to be detectable in formerly depressed adolescents: lower quality family relationships, smaller social networks, more minor hassles and major adversity, and lower life satisfaction. The nature of these variables suggests that residual effects of adolescent depression appear to cluster in the relatively specific domains of relationship quality and environmental adversity.

The availability of comparable functioning measures assessed at  $T_1$  made it possible for us to examine a third alternative explanation; namely, that participants who showed reduced functioning at  $T_3$  had already reduced functioning at  $T_1$ . We were able to address this issue with four of the young adult functioning variables that were still significant after adjusting for demographics, comorbid adolescent nonmood disorders, and MDD and nonaffective psychopathology between the ages of 19 and 23: major and minor adversity, quality of family relationships, and size of social network. Only one of the measures of functioning assessed at  $T_1$  appeared to worsen from adolescence to young adulthood: Formerly depressed adolescents reported smaller, less connected social networks in young adulthood.

To our knowledge, previous research has not controlled for preexisting levels of psychosocial functioning in adolescence. Our findings suggest that this is an important factor to consider, as many of the impairments found in young adulthood were already evident in adolescence. Unfortunately, we cannot determine whether these variables that preceded, and perhaps contributed to, adolescent MDD were early consequences of adolescent MDD or shared a common cause with adolescent MDD.

Our findings are generally consistent with a recent study by Geller and colleagues (Geller et al., 2001). In their longitudinal study of children treated for prepubertal MDD (onset between the ages of 6 and 12), former patients who had experienced no mood or substance use disorders in the 5 years prior to the follow-up assessment did not differ from a normal comparison group, with the one exception of having fewer friends. The formerly depressed children who had experienced depression recurrence or substance use disorder within the last 5 years showed many more impairments, including poorer social adjustment, lower life quality, and poorer functioning across several domains.

These findings are also consistent with theories that emphasize interpersonal issues and the interactional role of stressful life events in the etiology of depression (e.g., Brown & Harris, 1978; Hammen et al., 1995; Joiner & Coyne, 1999), as well as with Hammen's (1991) stress generation hypothesis, which suggests that people prone to depression recurrence create or contribute to



stressful conditions, especially to interpersonal stresses, by their behavior. As mentioned earlier, we were not able to determine the degree to which the formerly depressed adolescents were responsible for generating these major and minor adversities in their lives. Thus, the occurrence of stressful life events may not reflect anything about the individual's level of psychosocial functioning. Nonetheless, both of these measures are clearly indices of the environmental context in which these people are living. Future research needs to assess the degree to which formerly depressed individuals contribute to the occurrence of adverse events in their lives. Ideally, we would have examined change in each functioning domain before and after onset of the first MDD episode, but that was not possible given the timing of the first assessment (approximately two thirds of the adolescent MDD episodes had occurred prior to  $T_1$ ). Additionally, some of the variables we examined were not applicable to most high-school-aged adolescents (e.g., marital status).

The last variable we considered as an explaining factor was the level of depression symptoms at the time of the  $T_3$  assessment. As expected, formerly depressed adolescents were elevated on depression symptoms in young adulthood, therefore, controlling for current depression level eliminated all remaining associations. Several studies (Judd et al., 1998; Judd, Paulus, et al., 2000; Mojtabai, 2001; Paykel et al., 1995; Rohde, Lewinsohn, & Seeley, 1990, 1994) have shown that elevated depression symptoms are one of the more salient residual differences evident in many who experienced a depressive episode, both in adolescence and adulthood, and are associated with significant psychosocial impairment. The present findings once again emphasize the pervasive effects associated with depressive symptoms, even at subthreshold levels. However, we cannot exclude the possibility that the depressive symptoms biased participants' reporting of functioning rather than resulting in actual impairment.

An intriguing aspect of this last set of analyses is that controlling for depressive symptoms did not eliminate any of the significant associations between young adult functioning and adolescent nonmood disorders. Given that the CES-D is often considered a measure of nonspecific psychological distress (e.g., Coyne & Schwenk, 1997), it is noteworthy that this effect was specific to MDD; when depression level in the nonmood disorders is controlled, all of the associations with nonmood disorders remained significant.

To summarize the findings relevant to the third goal of the study, once we accounted for the effects of adolescent comorbidity, adolescent status on the outcome measures, young adult psychopathology, and current depressive symptoms, the effects of adolescent MDD on young adult functioning disappeared. Thus, it appears that adolescent MDD, in and of itself, does not have significant effects on young adult psychosocial functioning. The pattern of findings suggests that these variables may mediate the associations between adolescent MDD and subsequent functioning. Future longitudinal studies designed to formally test mediational models would be particularly informative. The present findings are similar to a recent study by Fergusson and Woodward (2002), and taken together, these results provide some reason for optimism regarding the long-term effects of adolescent MDD. However, it is important to bear in mind that we used a very conservative data analytic strategy, in which we included a number of covariates associated with both adolescent MDD and the young

adult outcomes. Thus, all of the shared variance between adolescent MDD and the covariate in predicting young adult outcome was attributed to the covariate. At any event, an optimistic interpretation of the results is applicable only to the approximately 20% of formerly depressed adolescents whose adolescent MDD episode was nonrecurrent and noncomorbid.

Although several limitations have already been noted, a few additional caveats are necessary. First, the sample was from a single region of the country and consisted predominantly of European Americans; these factors may limit the generalizability of the findings. Second, our measures of young adult psychosocial functioning relied exclusively on participants' self-report. This is a concern given that some of our strongest effects (e.g., life satisfaction, quality of relationships, and adversity) are quite subjective in nature. Third, a relatively large number of statistical comparisons were computed. While we required that findings reach the  $p < .01$  level of significance, this requirement did not fully correct for experiment-wide Type I error. Results need to be independently replicated as some of the findings may have been significant as a result of chance. Fourth, while we reported the associations of adolescent psychopathology with mental health treatment utilization in young adulthood, we could not examine the impact of mental health treatment services on depression and psychosocial functioning. Ideally, one hopes that treatment would ameliorate the negative functioning associated with psychiatric disorder. Unfortunately, it has been repeatedly shown that most depressed adults receive either no treatment or inadequate treatment (e.g., Hirschfeld et al., 1997; Ramana, Paykel, Surtees, Melzer, & Mehta, 1999); we previously found that this statement also applies to depressed adolescents (Lewinsohn, Rohde, & Seeley, 1998). Evaluating the impact of treatment on future functioning is complicated, however, by the fact that the more severely impaired individuals are also more likely to seek and receive treatment.

Finally, we want to emphasize that the OADP is a naturalistic study. While we can say with confidence that young adults with a history of adolescent MDD showed numerous signs of functional impairment, we cannot determine the causal chain of events. With these caveats in mind, given that the adolescent MDD episode preceded the measurement of functioning in this study, the pattern of findings is consistent with the possibility that some or all of these effects are related to the depression experienced during childhood and adolescence, either directly or, more often, through psychiatric comorbidity, depression recurrence, prior functioning, or current depressive symptoms. Clinically, our findings emphasize the need for effective interventions aimed at preventing the incidence of depression in adolescence and, given the high rate of MDD recurrence, effective monitoring and intervention efforts for preventing recurrence.

Future research needs to examine whether formerly depressed adolescents continue to be distinguishable from never-depressed controls, even after longer periods of time without recurrent depressive episodes. In addition, given that the risk of recurrence increases and the interval between depressive episodes appears to decrease with each recurrent episode (Lewinsohn, Pettit, Joiner, & Seeley, 2003; Solomon et al., 2000), the hypothesis needs to be examined that the magnitude of functional impairment increases as a function of the number of depression episodes.

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