Stress Burden and the Lifetime Incidence of Psychiatric Disorder in Young Adults

Racial and Ethnic Contrasts

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Background: With the exception of studies of individual traumatic events, the significance of stress exposure in psychiatric disorder previously has not been effectively examined.

Objective: To address the hypothesis that accumulated adversity represents an important risk factor for the subsequent onset of depressive and anxiety disorders.

Design: A community-based study of psychiatric and substance use disorders among a large, ethnically diverse cohort representative of young adults in South Florida. Adversity was estimated with a count of major and potentially traumatic events experienced during one's lifetime and prior to the onset of disorder.

Setting: Most interviews took place in the homes of participants, with 30% conducted by telephone.

Participants: We obtained a random sample of individuals aged 18 to 23 years from a previously studied representative sample of young adolescents. Because participants in the prior study were predominantly boys, a supplementary sample of girls was randomly obtained from the early-adolescence school class rosters. A total of 1803 interviews were completed, representing a success rate of 70.1%.

Main Outcome Measures: Depressive and anxiety disorders were assessed through computer-assisted personal interviews using the DSM-IV version of the Michigan Composite International Diagnostic Interview.

Results: Level of lifetime exposure to adversity was found to be associated with an increased risk of subsequent onset of depressive and/or anxiety disorder. This association remained clearly observable when childhood conduct disorder, attention-deficit/hyperactivity disorder, prior substance dependence, and posttraumatic stress disorder were held constant and when the possibility of state dependence effects was considered.

Conclusion: Evidence suggests that high levels of lifetime exposure to adversity are causally implicated in the onset of depressive and anxiety disorders.

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go from psychiatric disorder to stress exposure, and reported stressful events and circumstances may include both contributors to and consequences of psychiatric disorder.

Thus, meaningful evaluation of the stress hypothesis requires that consideration be limited to stressful experiences that are temporally prior to the first onset of disorder. This article assesses the role and significance of lifetime exposure to major and potentially traumatic events (cumulative adversity) in depressive and anxiety disorders. For these analyses, depressive and anxiety disorders are defined in terms of DSM-IV criteria as estimated by the Michigan Composite International Diagnostic Interview (CIDI), and cumulative adversity is based on a count of the reported lifetime experience of 33 distinct events.

MAJOR EVENTS AND MENTAL HEALTH

It is hardly novel to suggest that traumatic experiences can have significant adverse mental health consequences. Such experiences have represented prominent explanatory factors since the early days of psychoanalytic theory. Moreover, a considerable body of research has accumulated, suggesting the relevance of specific forms of early trauma for adult mental health. Principal among these are sexual abuse, physical violence and abuse, parental deaths, and parental psychopathologic characteristics and substance abuse. The hypothesis that major adverse experiences have long-term psychiatric significance is clearly tenable. However, studies that have gone beyond the consideration of individual and particular events to assess the significance of an array of major events, either singly or cumulatively, have been extremely rare.

MEASURING LIFETIME EXPOSURE TO MAJOR EVENTS

Our approach to assessing lifetime exposure to major events treats such experiences as different from typically assessed life events primarily in terms of their severity and presumably the duration of their consequences. The widespread practice of limiting consideration of stressful events to a 1-year time frame has been based largely on evidence that the effects of the events considered tend to be limited to less than a year, as well as the wish to avoid the falloff observed in the ability of respondents to recall many events beyond a 1-year time frame. However, the evidence regarding childhood victimization and parental death and divorce suggests that some events can and do have significant mental health consequences despite occurring years or even decades earlier. Moreover, the problem of reliability of reporting or recall does not apply equally to all events. For example, it would seem rare for subjects to forget or fail to respond to specific questions asking if their parents had divorced, if they had been in an accident in which someone was killed or badly injured, or if they had been shot with a gun or badly injured with another weapon.

It appears that a range of severe events can be measured with reasonable accuracy and, singly or in combination, may constitute important mental health risk factors. These considerations argue for the inclusion of lifetime experience of such events within efforts to understand variations in the occurrence of mental health problems. Our article reports the individual and cumulative significance of the lifetime experience of what is, to our knowledge, the widest range of major and potentially traumatic experiences studied thus far for the occurrence of depressive and anxiety disorders. Measures of exposure to recent life events, chronic stressors, and discrimination stress, all of which assess conditions relatively proximate to the time of the interview, are not considered in this study because of the associated time-ordering problems described previously. However, racial and ethnic differences in stress exposure, estimated in terms of these dimensions, and the significance of such exposure for psychological distress have been reported elsewhere.

STUDY POPULATION

This article is based on a study of the prevalence and social distributions of psychiatric and substance use disorders and of factors that increase and decrease risk of such disorders among a representative South Florida community cohort of 1803 young adults. Most (93%) were aged 19 to 21 years when interviewed between 1998 and 2000. The study possesses unique potential for contribution in several respects. First, these data are from perhaps the largest sample within this age range studied thus far in the United States. Second, this is one of the first large-scale community studies to estimate the occurrence of disorders based on DSM-IV criteria. Third, our study population is ethnically diverse, allowing consideration of ethnic variations in both stress exposure and the consequences of exposure. Specifically, approximately 25% of the sample were of Cuban origin, 25% were other Hispanic individuals from the Caribbean Basin, 25% were African American, and 25% were non-Hispanic white.

Our approach in obtaining this sample was in accord with a growing consensus in the field that race is more a social than a biological categorization akin to ethnic status and that there are important cultural variations within ethnic status. In an effort to minimize the effects of such variations on results, we have distinguished Cuban from other Hispanic individuals and have limited inclusion within this latter category to Hispanic people from countries in the Caribbean Basin. For the same reason, Haitian and other Caribbean black individuals were not studied and are not included in the African American subsample.

SAMPLE

This study was based on data from a representative sample of young adults, most of whom had been studied 3 to 7 years earlier. Between 1998 and 2000, we interviewed 1803 respondents aged 18 to 23 years (92% were aged 19 to 21 years), and all analyses presented in this article are based on data from these interviews. Overall, 70.1% of those sampled were successfully recruited to the study. Most interviews were carried out face to face in the homes of respondents, with 30% conducted by telephone and aided by mailed response booklets. Consistent with the bulk of evidence that in-person and telephone interviews yield comparable data, our analyses revealed no association between interviewing mode and the presence vs ab-
sence of an affective or anxiety disorder diagnosis (prevalence, 0.25 and 0.27, respectively, for in-person and telephone interviews). Although a slight difference in number of reported adversities was observed across interviewing mode (8.4 vs 7.8, respectively, for in-person and telephone interviews), the fact that the higher stress exposure corresponds with a lower prevalence of disorder suggests an absence of bias associated with interviewing mode. A more detailed description of the sample and study field procedures has been published previously.32

Informed consent was obtained from all study participants. The institutional review board of Florida State University, Tallahassee, approved the procedures for obtaining consent and protecting the rights and welfare of participants.

DIAGNOSTIC ASSESSMENT

Data on the lifetime and 1-year occurrence of psychiatric disorders were obtained through computer-assisted personal interviews that allowed the estimation of DSM-IV diagnoses. Our basic instrument was the Michigan CIDI, which was used in the National Comorbidity Survey (NCS).4 The CIDI is a fully structured interview based substantially on the Diagnostic Interview Schedule4 and designed to be administered by lay interviewers trained in its use.34-36 Using the Michigan CIDI as updated by NCS researchers to cover DSM-IV criteria, we assessed major depression, dysthymia, generalized anxiety disorder, social phobia, panic disorder, alcohol abuse and dependence, drug abuse and dependence, posttraumatic stress disorder, and antisocial personality disorder. These latter 2 modules had been borrowed from the Diagnostic Interview Schedule for the NCS. Evidence for the validity of Michigan CIDI diagnostic estimates, evaluated against the Structured Clinical Interview for DSM-III-R,36 has been reported for most NCS disorders including affective disorders,37 anxiety disorders,38,39 addictive disorders,40,41 and posttraumatic stress disorder.32

Along with the Michigan CIDI, our assessment instrument52 included a reliable module obtained from the revised Diagnostic Interview Schedule51 to assess attention-deficit/hyperactivity disorder and contained items to allow the assessment of childhood conduct disorder. The dependent variable for the analyses to be presented was the lifetime occurrence of major depressive disorder, dysthymia, social phobia, generalized anxiety disorder, and/or panic disorder.

MEASURING LIFETIME EXPOSURE TO MAJOR EVENTS

A retrospective procedure is necessarily required to estimate the timing and sequencing during the life course of major events and psychiatric disorders. The validity of retrospective reports is uncertain, and opinions of their usefulness vary widely. However, much of the information we have on age cohort differences in substance use and psychiatric disorders,31,38 lifetime comorbidity within and across these domains,32 social consequences of early-onset psychiatric disorders,46,47 and risk significance of early traumas for psychiatric and substance use disorders46,48 is based on retrospective reports obtained in the Epidemiologic Catchment Area Study.46 NCS, and an array of other studies. The potential significance of such relationships and the need to better understand their correctness and meanings emphasizes the importance of maximizing the accuracy of these retrospective reports. Specifically, the central need is to effectively order within the life course the first onset of psychiatric disorder and the occurrence of major and potentially traumatic life events.

We used a life history calendar based on that developed by Freedman et al32 as an aid in achieving the most accurate recall of significant life course experiences. This calendar traced 5 categories of experience. The first 3 involved a process in which respondents described divisions in their lives in terms of where they lived (country, city, or street as appropriate), landmark events (eg, birth of a sibling, getting a driver’s license, or finishing school), and the teachers or best friends they had during various years. These dimensions were completed at the beginning of the interview 1 at a time, each building on the information already obtained. The calendar was used at 3 points in the interview: (1) for questions about the age of occurrence of major or traumatic lifetime events; (2) for questions about the onset and last occurrence of substance abuse and related problems; and (3) for questions regarding the age at first and last occurrence of psychiatric disorder episodes. In each of these sections, the question of temporal order was established using all information available through scanning both upward and across the life history calendar. Thus, a reported first onset of major depression, for example, would be placed on the calendar in relation to other psychiatric disorders (if any), substance abuse disorders (if any), major life events, teachers and/or best friend at the time, landmark or transition events, and place of residence. By this means, the 33 items used to assess lifetime exposure to adversity were set in time relative to the onset of disorder. This procedure and the fact that the recall period for this young adult population was relatively short argues for the reliability of the data used in these analyses.

DEMENOPHICS

Ethnicity was measured by the respondents’ self-reported ethnic group identification. Because our subjects were in the transition to adulthood, socioeconomic status (SES) was estimated in terms of parental education, income, and occupational prestige level.53 These data were primarily obtained from parent interviews. However, it was necessary to rely on information provided by the participants for 33% of the sample. Scores for these 3 status dimensions were standardized, summed, and divided by the number of status dimensions for which the parent or participant was willing and able to provide information.

STATISTICAL ANALYSES

Our analyses were multivariate and based on discrete-time event history regression.44,51 This method used only data covering the time each individual was at risk for the event of interest. That included the entire period of observation for those for whom the event had not yet occurred, which were referred to as right-censored observations. Time at risk was divided into discrete periods (eg, years). Data for the earliest 5 years were collapsed into a single period because there was inadequate variation for analysis within the earliest individual years. The remaining information was grouped into 17 one-year intervals representing ages 6 to 22 years. Survival time to the first onset of a depressive or anxiety disorder among the 354 respondents who met criteria for 1 or more of these diagnoses, as well as the entire time at risk among the 1426 right-censored subjects, was divided into 26241 person-periods.

We refer to the period for which the conditional hazard of onset is estimated as the index period. In some of the analyses, the models distinguish between distal and proximal adversities. Distal adversities are counts of events occurring during any period earlier than the year prior to the index period. Proximal adversities are counts of events that occurred in the year preceding the index year. The effect of time is modeled as a cubic function. The coefficients for distal and proximal adversities reflect their independent associations with the conditional hazard of onset in that period. This analytic approach parallels that previously used to assess the linkage between cumulative adversity and DSM-IV drug dependence.30
Table 1. Associations Between DSM-IV Depressive or Anxiety Disorder and Lifetime Prevalence of Individual Adversities* (cont)

<table>
<thead>
<tr>
<th>Item</th>
<th>Adjusted Odds Ratio</th>
<th>Lifetime Prevalence, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Witnessed violence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have you ever witnessed someone else being hurt very badly or killed?</td>
<td>1.4†</td>
<td>47.0</td>
</tr>
<tr>
<td>Have you witnessed you mother or another close female relative being physically or emotionally abused?</td>
<td>1.9†</td>
<td>21.4</td>
</tr>
<tr>
<td>Have you seen someone chased but not caught or threatened with serious harm?</td>
<td>2.0†</td>
<td>38.0</td>
</tr>
<tr>
<td>Have you seen someone get shot at or attacked with another weapon?</td>
<td>2.0†</td>
<td>36.5</td>
</tr>
<tr>
<td>Have you ever seen someone seriously injured by a gunshot or another weapon?</td>
<td>1.7†</td>
<td>31.8</td>
</tr>
<tr>
<td>Have you ever actually seen someone get killed by being shot, stabbed, or beaten?</td>
<td>0.7</td>
<td>13.7</td>
</tr>
<tr>
<td>Traumatic news</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have you ever been told that someone you knew had been shot but not killed?</td>
<td>1.7†</td>
<td>39.5</td>
</tr>
<tr>
<td>Have you ever been told that someone you knew had been killed with a gun or other weapon?</td>
<td>1.4†</td>
<td>39.4</td>
</tr>
<tr>
<td>Has anyone else you knew died suddenly or been seriously hurt?</td>
<td>1.5†</td>
<td>36.1</td>
</tr>
<tr>
<td>Have you ever been told that someone you knew killed himself or herself?</td>
<td>2.1†</td>
<td>24.5</td>
</tr>
<tr>
<td>Have you ever been told that someone you knew had been raped?</td>
<td>1.9</td>
<td>35.5</td>
</tr>
</tbody>
</table>

*Odds ratios were derived from event history analysis of 1 adversity at a time controlling for time, sex, ethnicity, and socioeconomic status. The sample size was 1783 for adversity prevalence rates. †Indicates significance at \( P < .05 \).

Table 1 presents the 33 adversity items with rates of lifetime occurrence and the adjusted odds ratios for a depressive or anxiety disorder given the previous experience of each event. The odds ratios were derived from event history analyses of individual adversities, controlling for time, sex, ethnicity, and SES. This method ensures that the association does not reflect adversities occurring after the onset of disorder and that any causal connection in reported associations goes from the event to the depressive or anxiety disorder.

Of the 9 items listed as major events, 6 were associated with a significantly elevated risk of subsequent disorder onset. Rates of exposure to all 9 items varied by ethnicity, with 7 of the 9 reported more frequently by African Americans. Six of the 9 occurred significantly more often among women. Ten of the 13 items listed as life traumas significantly predicted the subsequent onset of a depressive or anxiety disorder, 5 of which differed in prevalence across ethnicity. White non-Hispanic individuals reported the highest rates of sexual molestation and physical abuse by someone other than a partner or parent. The rates of having been shot or shot at (or otherwise attacked with a deadly weapon) were highest among African Americans and higher among men in general. Five events were more frequently reported by women: sexual molestation, rape, physical abuse by a parent, emotional abuse by a parent, and physical abuse by a spouse or partner.
The substantially higher lifetime prevalence of depressive and anxiety disorders among women and the significantly lower prevalence of depressive disorders among African Americans correspond with findings from the NCS.7 Despite the clear link between SES of origin and lifetime risk of exposure to major stressors, these results offer no indication that those who grew up in poorer economic circumstances are any more or less likely to have experienced a depressive or anxiety disorder.

The results of hierarchical event history analyses that distinguish between distal and proximal occurrences are presented in Table 3. As previously noted, events are categorized as distal if they occurred earlier than the year prior to the index year (the year for which hazard is estimated). Proximal events are those occurring in the year prior to the index year.

The upper section of Table 3 shows the associations previously presented between sex, ethnicity, and psychiatric disorder and demonstrates their independent significance. We trimmed SES from these models on the basis of preliminary analyses that revealed no independent relationship with disorder. Model 2 indicates that distal adversities are significantly associated with disorder. When proximal adversities are added (model 3), distal events remain significant, and the magnitude of the distal events association is little diminished. The nonutility of stress exposure differences for explaining ethnic variations in risk is clearly demonstrated in these results. When both distal and proximal adversities are held constant, the coefficients for each ethnic group contrasted with African Americans increase rather than decrease. Computing the change in these coefficients from model 1 to model 3 reveals that if other groups were as exposed to adversity as are African Americans, the occurrence of depressive and anxiety disorders would increase by 32% to 35% rather than decrease.

Clearly, there is a compelling relationship between cumulative adversity and risk of a depressive or anxiety disorder. The magnitude of this associated risk can be illustrated in terms of the odds ratio for disorder onset at high compared with low levels of exposure to adversity. At age 18 years, the mean ± SD number of preonset

### Table 2. Lifetime Prevalence of DSM-IV Depressive and Anxiety Disorders and Lifetime Exposure to Adversities Among Community-Dwelling Young Adults*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Lifetime Depressive Disorder</th>
<th>Lifetime Anxiety Disorder</th>
<th>Lifetime Disorders Combined</th>
<th>Adversity Count, Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>12.6</td>
<td>2.9</td>
<td>14.0</td>
<td>7.59 ± 4.52</td>
</tr>
<tr>
<td>Women</td>
<td>23.5</td>
<td>7.8</td>
<td>26.5</td>
<td>6.60 ± 4.61</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Non-Hispanic white</td>
<td>19.9</td>
<td>6.3</td>
<td>21.9</td>
<td>6.52 ± 4.49</td>
</tr>
<tr>
<td>Cuban</td>
<td>19.8</td>
<td>5.2</td>
<td>22.0</td>
<td>6.32 ± 4.50</td>
</tr>
<tr>
<td>Non-Cuban Hispanic</td>
<td>18.4</td>
<td>6.6</td>
<td>21.3</td>
<td>6.84 ± 4.53</td>
</tr>
<tr>
<td>African American</td>
<td>13.0</td>
<td>3.2</td>
<td>14.3</td>
<td>8.97 ± 4.38</td>
</tr>
<tr>
<td>P value</td>
<td>.03</td>
<td>.11</td>
<td>.01</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Socioeconomic level</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lowest quartile</td>
<td>17.4</td>
<td>4.8</td>
<td>19.0</td>
<td>7.67 ± 4.64</td>
</tr>
<tr>
<td>Second quartile</td>
<td>18.3</td>
<td>4.7</td>
<td>20.5</td>
<td>7.62 ± 4.91</td>
</tr>
<tr>
<td>Third quartile</td>
<td>16.6</td>
<td>5.1</td>
<td>19.6</td>
<td>7.24 ± 4.49</td>
</tr>
<tr>
<td>Highest quartile</td>
<td>19.3</td>
<td>6.4</td>
<td>21.1</td>
<td>6.07 ± 4.14</td>
</tr>
<tr>
<td>P value</td>
<td>.75</td>
<td>.64</td>
<td>.87</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Total sample</td>
<td>18.0</td>
<td>5.4</td>
<td>20.3</td>
<td>7.12 ± 4.59</td>
</tr>
</tbody>
</table>

*Data are presented as percentage unless otherwise indicated. Depressive disorders include major depression and dysthymia; anxiety disorders include generalized anxiety, panic disorder, and social phobia. Adversity score is a lifetime count. Reported significance of difference in group rates was determined using the χ² test; mean count differences were determined using 1-way analysis of variance. Results reflect the application of weight to correct socioeconomic status bias across sex.
adversities experienced was 4.21 ± 3.57 for distal events and 0.88 ± 1.19 for proximal events. Based on the effects in Table 3, the odds ratio for disorder onset among subjects with distal adversity scores 1 SD higher than the mean is double that for those with scores 1 SD lower than the mean (odds ratio = 2.0) with proximal adversities controlled. The comparable odds ratio for the effect of proximal adversity with distal adversities controlled is a lower but still substantial 1.53. Additional analyses of interaction effects (data not shown) revealed that the rate at which increases in lifetime stress are translated into increased risk for disorder is generally equivalent across ethnicity and sex.

The lower section of Table 3 presents an effort to assess the plausible alternative hypothesis that problematic individuals tend to place themselves in circumstances in which stress exposure is more likely, on the one hand, and are at elevated risk for psychiatric disorder on the other. This analysis controls for childhood conduct disorder, which reflects serious conduct problems up to age 15 years, and attention-deficit/hyperactivity disorder as well as the prior occurrence of posttraumatic stress disorder and alcohol or drug dependence. Although attention-deficit/hyperactivity disorder represents a significant independent predictor of psychiatric disorder, the inclusion of this set of previous conditions produces no substantial changes in the observed effects of cumulative adversity.

A second competing hypothesis is that these results arise from state dependence bias. State dependence bias refers to the tendency for persons with a current disorder to be more likely to remember and/or report having stressful events more than those same individuals when they are relatively free of disorder symptoms. We evaluated the plausibility of this hypothesis in 2 ways. First, we compared the total number of adversities reported by participants with a current (last 6 months) psychiatric (excluding posttraumatic stress disorder) or substance abuse disorder with the reports of those who met criteria for 1 or more of these disorders but for an earlier period in their lives. The mean scores for these 2 groups were highly similar (9.99 among those with a current disorder and 10.23 among those with a previous disorder; \( P = .82 \)). We also extended the analysis shown in model 4 of Table 3 by controlling for level of depressive symptoms at the time of the interview, as estimated by scores on the Center for Epidemiologic Studies Depression Scale. Although the coefficients for distal and proximal adversities were diminished, they remained statistically significant. These twin results make it unlikely that the observed links between cumulative adversity and psychiatric disorder can be attributed to state dependence.

As previously reported,56 these results indicate that exposure to major and potentially traumatic events is commonplace among young people, at least in South Florida. The typical African American in the sample had experienced more than 9 such events, and the remaining 3 groups averaged more than 6. A total of 26 of the 33 events examined were associated with significantly increased risk for a depressive or anxiety disorder. In some cases the experience itself may be implicated in the observed elevation in risk, whereas in others the event may represent simply a marker for the occurrence of other stressors and/or the presence of other significant risk factors.

For both sexes and for all 4 ethnic groups, increases in lifetime exposure to adversity were associated with increased risk for psychiatric disorder. Moreover, the rates at which increases in exposure were translated into increases in risk were generally equivalent for all subgroups. These latter findings make it unlikely that the relatively low prevalence of affective and anxiety disorders among African Americans is attributable to social or cultural differences in resiliency or other stress-
moderating factors. More plausible are the possibilities that response differences to diagnostic measures systematically underestimate the presence of disorder among African Americans or that stress exposure is more often translated into physical disorders that are dramatically elevated in that population.

These results also demonstrate that accumulating adversities, both distal and proximal, contribute significantly and independently to the prediction of disorder with demographic factors controlled. Moreover, the role of prior adversity remains clearly observable when child conduct disorder, attention-deficit/hyperactivity disorder, prior substance dependence, and prior posttraumatic stress disorder are held constant.

This set of findings along with evidence contrary to the state dependence hypothesis suggests that high levels of lifetime exposure to adversity may be causally implicated in the onset of depressive and anxiety disorders. Although this statement appears equally applicable across ethnic groups, it is clear that exposure differences cannot account for the relatively low prevalence of depressive and anxiety disorders among African Americans observed in this as well as previous studies. If exposure to stressors were equalized across ethnic groups, the more favorable outcome found among African Americans would be substantially more rather than less pronounced.

The finding of no SES variations in risk of depressive and anxiety disorders is of special interest in the context of the clear links between SES and stress exposure and between stress exposure and the occurrence of such disorders. Because the SES measure is based on parental education, occupation, and income and must therefore roughly index participants’ developmental social environment, the lack of any association with the disorders examined would appear to challenge social causation perspectives.

Given the well-established relationship between adult SES and risk of psychiatric disorder, it is the fact that the young adults in this study with a lifetime occurrence of depressive and anxiety disorder appear to be randomly recruited with respect to SES of origin would traditionally be held as indirect but compelling evidence for the primacy of biogenetic etiology. In contrast, the clear relationship between social stress and the occurrence of these disorders and the fact that stress exposure is significantly elevated among those with parents of low SES provide direct and persuasive support for the hypothesis that variations in the social environment represent crucial causal contingencies in psychiatric disorder. The combination of these seemingly contradictory results suggests that level of exposure to social stress during the developmental years may influence an individual’s eventual socioeconomic achievement, at least partially through affecting the risk for disorders that tend to impede such achievement. Thus, with respect to the widely documented social class–mental health associations, these findings suggest that social selection phenomena may arise substantially from prior social causation processes rather than simply reflecting the consequences of biogenetically determined disorders.

The limitations of this study, as previously noted, include those characterizing prior studies that have derived diagnoses from a single structured interview that does not involve clinical judgment. The data are cross-sectional, so estimates of the lifetime occurrence of depressive and anxiety disorders rely entirely on retrospective recall. Whereas the young age of this cohort presumably minimizes recall problems, they remain of some concern. The fact that study participants represent a narrow age range (approximately 93% were aged 19 to 21 years) advises caution in generalizing these findings to other age groups, particularly given the remaining years of high risk for the onset of the disorders considered. Finally, although we believe our sample to be representative of young adults in Miami-Dade County, Florida, the distinct nature of the resident Hispanic population suggests caution in generalizing to other areas of the country.

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