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**Low Socioeconomic Status and Mental Disorders:  
A Longitudinal Study of Selection and Causation  
During Young Adulthood**

**Richard A. Miech**

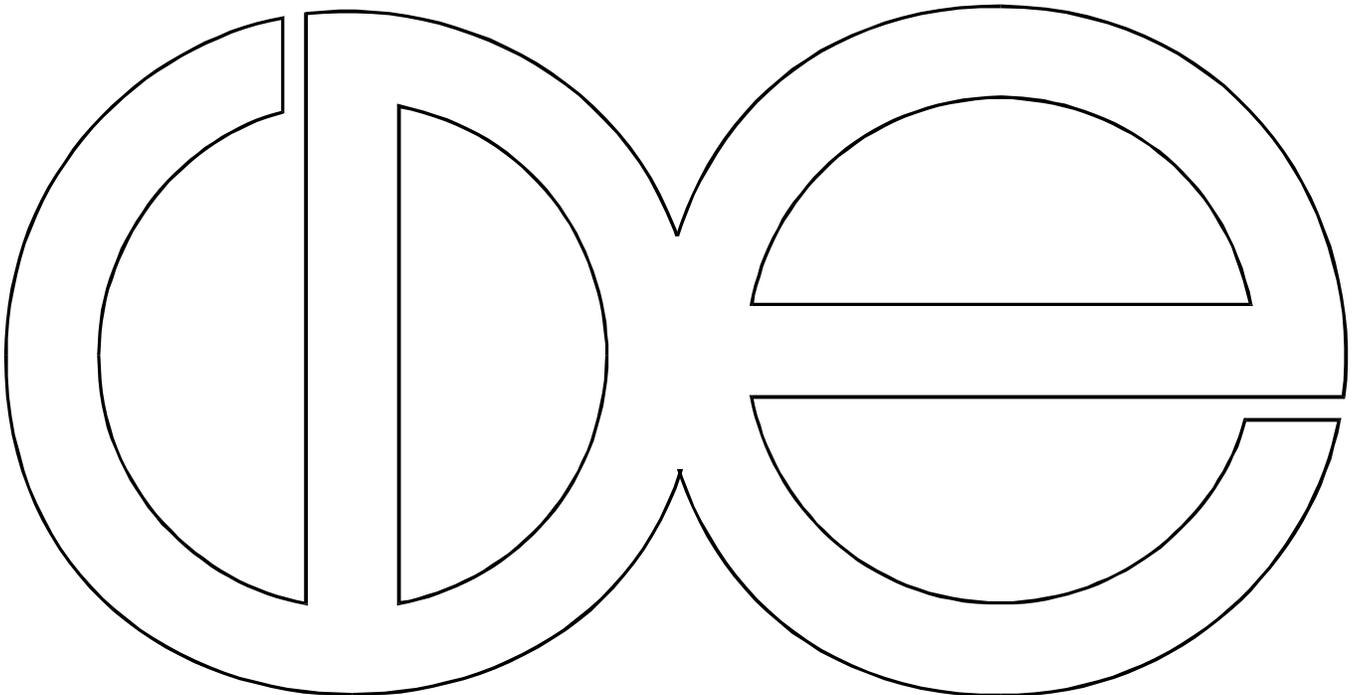
**Avshalom Caspi**

**Terrie E. Moffitt**

**Bradley E. Wright**

**Phil A. Silva**

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Richard A. Miech

*University of Wisconsin, Madison*

Avshalom Caspi, Terrie E. Moffitt

*Institute of Psychiatry, University of London and University of Wisconsin, Madison*

Bradley E. Wright

*University of Wisconsin, Madison*

Phil A. Silva

*University of Otago Medical School*

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## Abstract

Over the past half century two competing hypotheses in sociological inquiry have provided interpretations of the well-documented association between low socioeconomic status and mental disorders. The selection hypothesis asserts that mental disorders impair status attainment, whereas the causation hypothesis states that conditions of life associated with low socioeconomic status markedly increase the risk of mental disorders. Using data from the longitudinal Dunedin Multidisciplinary Health and Development Study (n=1037), we examine selection and causation processes during the transition to young adulthood by investigating the mutual influence of mental disorders and educational attainment, a core element of socioeconomic status. The Dunedin Study follows a cohort from birth to age 21, and includes psychiatric diagnoses for study members at ages 15 and 21 using DSM criteria. We focus on the four disorders of anxiety, depression, anti-social disorder, and attention deficit disorder and find a unique relationship with socioeconomic status for each one. These findings highlight the need for (a) greater consideration of antisocial disorders in the status attainment process and (b) more theoretical development in the sociology of mental disorders to account for disorder-specific relations with socioeconomic status.

Mental disorders are overrepresented in the lower social strata (Kessler et al. 1994; Dohrenwend et al. 1992; Link and Dohrenwend 1989; Neugebauer, Dohrenwend, and Dohrenwend 1980; Wheaton 1978; Holzer et al. 1986). Efforts to distinguish socioeconomic status (SES) as a cause or consequence of mental disorder address some of the most vexing problems in social demography and medical sociology. On the one hand, mental disorders may play an important role in determining who gets ahead in society, a topic pursued by sociological research in the “selection” tradition that examines the extent to which disorders impair status attainment (Eaton 1980; Eaton 1985; Dohrenwend 1975). On the other hand, adversities linked to low SES may damage the psychological functioning of individuals and play a role in the etiology of mental disorders, a topic pursued by sociological research in the “causation” tradition (Turner, Wheaton, and Lloyd 1995; Link, Lennon, and Dohrenwend 1993; Kohn 1981; Wheaton 1978). Despite fifty years of research, key theoretical issues regarding the causal direction between low socioeconomic status and mental disorders still remain unsettled (Dohrenwend et al. 1992; Fox 1990; Ortega and Corzine 1990).

In this paper we examine the selection and causation hypotheses by using a panel study to investigate the association between mental disorders and educational attainment, a key component of social status (Sewell, Hauser, and Featherman 1976). Our prospective study of adolescents as they make the transition to adulthood allows us to evaluate the temporal ordering of mental disorders and educational attainment more directly than past studies. As a consequence, we are able to test whether the relation between educational attainment and non-psychotic disorders results from selection, social causation, or a combination of both processes – three alternative interpretations that have not yet been disentangled with the research designs used in the current

literature. The data come from the Dunedin Multidisciplinary Health and Development Study (n=1037), which has followed a group of children from birth to age 21, and which includes psychiatric diagnoses for all study members at ages 15 and 21 using criteria from the Diagnostic and Statistical Manual of Mental Disorders (the DSM-III and DSM-III-R; American Psychiatric Association 1980 and American Psychiatric Association 1987).

To the extent that we find evidence for selection effects, our results speak to mental disorders as an important, yet neglected factor in the process of status attainment. In general, social demographers have not been interested in studying mental disorders. This is surprising, however, because mental disorders are not uncommon in the general population and they may play an important role in maintaining social inequalities. Although some disorders, such as schizophrenia, are indeed rare, affecting only 1% of the population, epidemiological studies reveal that the prevalence rate of mental disorders in young adulthood is alarmingly high and that 25% to 40% of young adults in industrial countries meet diagnostic criteria for a psychiatric disorder. These high rates are of special concern for at least three reasons. First, afflicted young adults are not trivially afflicted but are indeed functioning more poorly than their nondisordered peers (Newman et al. 1996). Second, rates of psychiatric disorder far exceed the rates of service use; it is estimated that only 10%-30% of cases with disorder receive any treatment (Institute of Medicine 1994). Third, the onset of most disorders peaks between adolescence and young adulthood (Institute of Medicine 1994), a developmental period when disorders have the potential to disrupt educational transitions and thereby compromise this initial and consequential first step in the status attainment process. If mental disorders are as common as psychiatric epidemiological studies suggest, and if they are as nontrivial as the data also show, they may well

be an important source of variation in determining social inequalities.

By testing whether causation acts alone or jointly with selection effects our results are also important for specifying how SES influences mental health, a central issue in the social stress literature. If social causation acts alone it suggests that people from lower-SES environments are ‘battered and bruised’ by SES-linked social stressors and end up with higher levels of mental disorders. If we find joint effects, in contrast, this would suggest a more complex process whereby SES-linked mental disorders lead afflicted individuals to fall further into the lower social strata and, over time, become further exposed to social stressors that foster disorders, a cycle of disadvantage that can accumulate over the life course (Caspi, Elder, and Bem 1987). Current research investigating the SES-based social stressors that induce disorders such as anxiety and depression operates under the assumption of exclusive social causation (Turner, Wheaton, and Lloyd 1995; Aneshensel 1992), but the need to revisit this assumption – which is based on only scarce longitudinal evidence – is suggested by recent research that contradicts it (Kessler et al. 1995). Further, it is not known if this assumption of exclusive social causation applies to other disorders. In this study we address the call to expand stress research into disorders other than anxiety and depression (Aneshensel, Rutter, and Lachenbruch 1991) by investigating selection and causation in regard to conduct disorder and attention deficit disorder, illnesses that are associated with troubled relations with peers, parents, schools, and other institutions during the transition to adulthood (Robins 1966, Weiss and Hechtman 1986; Elliott 1974).

#### *Selection and Causation: The Hypotheses*

Research in the “selection” perspective suggests that mental disorders are overrepresented in the lower socioeconomic strata as a consequence of impaired social mobility. Selection

processes operate both within and across generations. Within a generation mental disorders may cause downward mobility among adults and lead them to “drift” into the lower socioeconomic strata (Jarvis 1971, pp. 55-56; Eaton 1980). Across generations, a disorder may be transmitted to offspring, as suggested by recent twin and adoption studies that indicate a role for both genetic and environmental factors in mental disorders (Kendler et al. 1995). A transmitted disorder that impairs status attainment may have cumulative effects across successive generations of a lineage, ultimately leading to the creation of a “residue” of people with mental disorders in the lower socioeconomic strata (Gruenberg 1961; Dohrenwend et al. 1992). All studies that investigate selection processes are characterized by a causal arrow that points from mental disorders to socioeconomic status, unlike research in the causation perspective, which points the arrow the other way.

Research on the “causation” hypothesis suggests a wide array of mechanisms through which socioeconomic status may affect psychopathology. These mechanisms may either cause illnesses or serve as catalysts to people with genetic predispositions for disorders, and they include stress (Turner, Wheaton, and Lloyd 1995; Kessler 1979; Pearlin and Johnson 1977; Link, Dohrenwend, and Skodol 1986), poor social or psychological coping resources (Kohn 1981; Kessler and Cleary 1980; Dohrenwend and Dohrenwend 1970; Liem and Liem 1978), and lack of occupational direction, control, and planning (Link, Lennon, and Dohrenwend 1993). While the very different processes of selection and causation might at first appear easily distinguishable, disentangling their effects has proven an elusive goal.

Selection and causation research to date has faced two main obstacles that have prevented a clear test between the interpretations of causation, selection, and combined effects. First, the

temporal ordering of mental disorders and low socioeconomic status has been difficult to establish without longitudinal research designs. Second, the absence of a standardized psychiatric nosology has hindered the ability of researchers to compare findings about social status and mental disorders across different studies that use different measures. In the present study we address these two limitations by conducting a longitudinal analysis of selection and causation and by examining these processes in relation to multiple, specific mental disorders diagnosed in accordance with a standardized nosology.

*Selection and Causation: The Research Strategies*

The study of selection and causation centers on temporal ordering and whether the onset of mental disorders occurs before or after low socioeconomic experience: evidence that it occurs before provides strong support for selection, while evidence that it occurs after is consistent with a social causation interpretation. With temporal ordering in mind, an initial research strategy focused on whether adults with mental disorders came disproportionately from lower-SES family backgrounds. If they did, this evidence suggests that low socioeconomic experience preceded the onset of mental disorder and, consequently, favors a causation interpretation. Yet while many studies find that the parents of people with mental disorders are concentrated in the lower social strata (see, for example, Kessler et al. 1994), the interpretation of this finding is more complex than originally expected. For example, an illness such as conduct disorder may impair a person's status attainment and then be passed on to offspring, either through parental socialization or through increased exposure to socioeconomic-based stressors that foster the illness. In this case, the finding that children with conduct disorder are overrepresented among parents with low socioeconomic status stems both from causation processes that operated on the child, and, in

addition, selection processes that operated on the parents (Dohrenwend 1975). In short, this initial research strategy can suggest preliminary evidence for causation effects, but it is limited because it does not evaluate their size relative to selection effects, nor does it determine whether causation acts to the exclusion of selection.

Subsequent research has pursued two main research strategies to determine the temporal order between mental disorders and SES. The first relies on retrospective reports of study members. Kessler et al. (1995), for example, sought to determine the temporal order between mental disorders and educational dropout by relying on the respondents' ability to recall their mental state before major educational transitions, such as high school completion and college entrance. On average, study members were asked to recall their mental state fifteen years before the date of interview. Using these retrospective reports they find support for the selection perspective to the extent that study members who failed to make major educational transitions reported an overrepresentation of anxiety disorders, mood disorders (including depression), and conduct disorder that predated their educational dropout. Conceivably this research design could be extended to simultaneously measure the influence of causation processes. It is important to note, however, that conclusions based on analyses using this research design rest on the assumption that retrospective reports of the occurrence and timing of childhood and adolescent mental disorders are valid and reliable, a matter of considerable debate (see Aneshensel, Estrada, Hansell, and Clark 1987; Henry, Moffitt, Caspi, Langley, and Silva 1994; Rogler, Malgady, and Tryon 1992; Holmshaw and Simonoff 1996).

The questionable validity of retrospective reports has led some researchers to forgo them and pursue a second research strategy instead. This approach does not measure temporal order

directly, but rather infers it from the insight that ethnic discrimination has distinctly different consequences for selection and causation outcomes (for a detailed discussion see Dohrenwend 1975; Dohrenwend et al. 1992). In brief, the selection perspective leads to the expectation that disorders will have smaller, diluted prevalence rates across all SES levels of disadvantaged groups because ethnic discrimination hinders social mobility and keeps greater numbers of healthy members at lower SES levels. In contrast, the causation perspective leads to the competing expectation that disorders will have higher prevalence rates among disadvantaged groups, who presumably experience higher stress levels. Analysis employing this research design supports the presence of causation processes for non-psychotic disorders such as depression and anti-social personality disorder (Dohrenwend et al. 1992), but provides little information regarding the possibility that smaller selection effects are operating at the same time. The design succeeds in evaluating the relative strength of selection and causation, but it fails to provide a test to determine if either process is operating exclusively. This limitation is critical because the assumption of exclusive social causation underpins much of the social stress literature.

A longitudinal research design offers the opportunity to test between selection, causation, and combined-effects interpretations without relying on retrospective information. However, such prospective studies are rare due to the cost and logistics involved in repeated evaluations of large samples. Wheaton (1978) conducted one of the few panel studies of psychological disorder and social status, using occupational prestige to index social status in the adult years. Relying on a general measure of mental illness that combines symptoms of both anxiety and depression (the Langner index, see Langner 1962), he finds evidence for causation effects and no evidence for selection effects among adults. More recent longitudinal research supporting causation in regard

to clinical levels of depression and anxiety comes from the study of Bruce, Takeuchi, and Leaf (1991), based on a six month follow-up of 5000 adults aged 18 and over. The existing field of longitudinal research leaves room for advances in at least two ways, however, as we discuss below.

*Selection and Causation During the Transition to Young Adulthood*

The transition from adolescence to adulthood has not yet been examined with a longitudinal research design, although adolescence is a strategic time period for the study of both selection and causation. In terms of selection, adolescence is a period when individuals make decisions about their educational attainment, and, consequently, their social status. Educational attainment is both itself a primary component of SES indices and also a major predictor of subsequent income and occupational prestige over the life course (Blau and Duncan 1967; Sewell, Hauser, and Featherman 1976; Hauser 1994; Jencks et al. 1979). Indeed, as an initial and powerful step, education is the “key” factor in the status attainment process (Sewell and Hauser 1976, p. 13). Through limited educational attainment adolescents may have already selected themselves into the lower social strata before adulthood, a process that represents selection effects to the extent that it is influenced by adolescent mental illness.<sup>1</sup> In the present study we investigate this potential process in the framework of a longitudinal study designed to capture the transition from adolescence to adulthood.

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<sup>1</sup>Wheaton (1978) presents one finding regarding mental illness and educational attainment using a longitudinal design, but it is evidence that he highly qualifies. One of the cohorts he studied was initially enrolled at age 17 and presented, in theory, the opportunity to measure the effects of psychological disorder on later educational attainment. Unfortunately, it was necessary for Wheaton to produce an *ad hoc* psychological scale from the base-line survey of this cohort. While analysis using the scale suggests that psychological disorder impairs educational attainment, as Wheaton points out (1978, p. 394) there is little certainty about what psychological domain the scale measures.

In terms of causation, the peak risk period for the emergence of new cases of DSM mental disorders is during the transition from adolescence to young adulthood (Burke, Burke, Regier, and Rae 1990; Institute of Medicine 1994), suggesting that the factors that cause mental disorders are especially influential during this developmental period of the life course. Young adult cohorts have higher DSM period-prevalence rates than older cohorts in national cross-sectional studies (Kessler et al. 1994; Robins and Regier 1991), and the Dunedin cohort analyzed in this study shows a large increase (almost two-fold) in the rates of mental disorder between ages 15 and 21 (Newman et al. 1996). Family SES origins appear to play an influential role during this formative period, as the literature on child and adolescent psychopathology documents a significant association between SES and mental disorders (Costello et al. 1997; Velez, Johnson, and Cohen 1989; Rutter et al. 1974). However, this literature has yet to examine the association in detail and to separate causation from selection effects, and as such relatively little is known about the SES-linked factors that are associated with increased risk of mental disorder from adolescence to young adulthood (Institute of Medicine 1994).

*Are Selection and Causation Effects Disorder-Specific?*

We examine the ways in which selection and causation processes vary across different mental disorders in the transition to adulthood, a topic that has not yet been examined with a longitudinal research design. Current research suggests that different non-psychotic disorders are related to social status in different ways (Dohrenwend et al. 1992). Detailing differences across disorders can better inform selection and causation theories, many of which are not disorder-specific but have only been evaluated with regard to depression or anxiety (Aneshensel, Rutter, and Lachenbruch 1991). The standardized DSM classification system that we use in this study is

well-suited for an investigation of disorder-specific relations with socioeconomic status because it provides formal guidelines to distinguish between different mental disorders.

It is important to note, as well, some disadvantages of the DSM system. Two issues in the diagnosis and classification of psychopathology are especially relevant to the study of selection and causation processes. First, an increasing number of investigators are questioning the adequacy of a categorical system to classify psychopathology (Kirmayer, Robbins, and Paris 1994; Clarkin and Kendall 1992; Widiger and Shea 1991), and some advocate the development of an alternative system based on dimensions (Clark, Watson, and Reynolds 1995). Categorical and dimensional approaches are not mutually exclusive, however, and for this study we use both; we present one set of analyses employing DSM diagnoses as categorical measures, and a parallel set of analyses using DSM symptom scales as quantitative, dimensional measures (a strategy recommended by Ollendick and King 1994, and Aneshensel, Rutter, and Lachenbruch 1991).

Second, epidemiological studies using the DSM classification system have revealed high rates of comorbidity: the simultaneous presence of two or more mental disorders. Approximately half of all individuals with a DSM disorder meet criteria for at least one additional comorbid disorder (Kessler et al. 1994; Robins and Regier 1991; Newman et al. 1996), an issue that has implications for clinical practice and research design (Clark, Watson, and Reynolds 1995). If comorbid cases are present in a research sample (as they will be in unselected samples such as the one studied here), it is important to control for the potentially confounding effects of multiple disorders before interpreting the unique influence of a particular target disorder on later outcomes (Sher and Trull 1996). In our multivariate analyses we include statistical controls for the spectrum of symptoms (and diagnoses) before evaluating findings about associations between a

specific mental illness and educational attainment.

We focus on the internalizing and externalizing disorders, the major mental disorders that afflict adolescents (Achenbach and Edelbrock 1983). Internalizing disorders involve emotional distress that is turned inward, and include anxiety and depression. These are the disorders that have received the most sociological attention in studies of adults, and our study of adolescents serves to complement this literature. The applicability of adult anxiety and depression symptoms to adolescents was once a matter of controversy (Rutter 1986), but is now generally accepted in the field (Reynolds 1992).

Externalizing disorders, in contrast, are characterized by “acting out” behavior, such as poorly controlled, impulsive behavior as well as attention problems and hyperactivity. In adolescence they are comprised mainly of conduct disorder and attention deficit disorder. In adulthood, the anti-social behavior that characterizes conduct disorder may manifest itself as anti-social personality disorder (Lynam 1996; Moffitt 1993). We extend our knowledge of selection and causation processes by examining these disorders, which have been neglected in the sociological literature to date.<sup>2</sup>

In sum, the longitudinal Dunedin study enables us to avoid retrospective reports, extend the selection and causation literature by focusing on the formative transition from adolescence to adulthood, and examine disorder-specific relations with socioeconomic status. This study both complements the literature on adults and, more generally, adds to the underdeveloped field of

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<sup>2</sup>Our mental health measures do not include schizophrenia, which has been extensively studied in previous research on social status and mental illness. Adolescence is too early in the life course to study selection and causation effects in regard to psychoses such as schizophrenia, which tend to onset after age 20 (Robins and Regier 1991).

adolescent mental health (Institute of Medicine Committee for the Study of Research on Child and Adolescent Mental Disorders 1995).

## SAMPLE AND METHOD

### *The Dunedin Study*

Subjects for this follow-up study were members of a complete birth cohort that has been studied extensively since birth in the Dunedin Multidisciplinary Health and Development Study. The sample and the history of the study have been described elsewhere (Silva 1990; Silva and Stanton 1996). Briefly, the study is a longitudinal investigation of the health, development, and behavior of children born between April 1, 1972, and March 31, 1973 in Dunedin, New Zealand, a city of approximately 120,000. Perinatal data were obtained at delivery. When the children were later traced for follow-up at age three, 1037 (52% males and 48% females, 91% of the eligible births) participated in the assessment, forming the base sample for the longitudinal study. Prevalence rates of psychiatric disorders such as major depression and conduct disorder in the Dunedin sample match rates from national U.S. surveys (Costello 1989; Kessler et al. 1994; Newman et al. 1996).

The Dunedin sample has been assessed with a diverse battery of psychological, medical, and sociological measures at ages 3, 5, 7, 9, 11, 13, 15, 18, and 21. The basic procedure for data collection in the Dunedin study involves bringing each sample member into the research unit within 60 days of his or her birthday for a full day of data collection in which various research topics are presented as standardized modules (e.g., mental health interview, Life History Calendar, physical examination) by different trained examiners in counterbalanced order. Auxiliary data are also collected from parents, teachers, peers, and official records.

The present study uses data collected at ages 15 and 21. At the age-15 follow-up in 1987-88, data were missing for 8 study members who had died since age three, 9 who could not be located, 33 who did not participate, and 11 living overseas who were unable to come to the research unit. Thirty three subjects participated in the assessment but have missing data on a DSM-III diagnostic category at age 15 and are not included in the analysis pool. Our final sample for the analysis of selection effects consists of the 939 for whom we obtained complete longitudinal data about mental disorders and educational attainment, a response rate of 91%. Of these 939 study members, 96% were reevaluated for mental disorders at age 21 using DSM-III-R criteria for mental disorders and are included in the analysis of causation effects.

#### *Mental Health Measures*

Mental health data were collected at ages 15 and 21 using the most current version of the DSM available at the time of interview. All interviews were privately conducted by trained staff with university degrees in social work, nursing, or clinical psychology. Age-15 interviews were conducted using the Diagnostic Interview Schedule for Children (DISC-C; Costello et al. 1982) an instrument designed to reliably assess the criteria of the DSM-III (American Psychiatric Association 1980). Age-21 interviews were conducted using the Diagnostic Interview Schedule (DIS; Robins, Helzer, Cottler, and Goldring 1989), an instrument designed to reliably assess the criteria of the DSM-III-R (American Psychiatric Association 1987). The DISC-C and DIS were used to obtain diagnoses of mental disorders in the 12 months prior to the study member's 15th and 21st birthday interview. The modifications, psychometric properties, and descriptive epidemiology of the DISC-C and DIS in this sample have been described in detail by McGee et al. (1990) and Newman et al. (1996), respectively. Twenty two percent of the sample met the

requisite DSM-III criteria for disorder at age 15, and forty percent met DSM-III-R criteria for disorder at age 21, point-prevalence rates that are consistent with other epidemiological studies throughout the world (Costello 1989; Kessler et al. 1994; Robins and Regier 1991).

For the current study, we investigated the internalizing disorders of anxiety and depression, and the externalizing disorders of conduct disorder, attention deficit disorder, and anti-social personality disorder. At age 15 the anxiety disorder group (n=100) consisted of study members who met criteria for the DSM-III anxiety disorders of childhood: overanxious disorder, separation anxiety, simple phobia, social phobia, or any combination of these disorders. At age 21 the anxiety disorder group (n=185) consisted of study members who met the criteria for the DSM-III-R anxiety disorders of adulthood: generalized anxiety disorder, obsessive-compulsive disorder, panic disorder, agoraphobia, social phobia, simple phobia, or any combination of these disorders.<sup>3</sup> The depression disorder group (n=37 at age 15, n=163 at age 21) consisted of study members who met DSM-III criteria for a major depressive episode, dysthymia, or both. The conduct disorder group consisted of study members who met DSM-III criteria for conduct disorder and/or oppositional disorder at age 15 (n=81), and our anti-social disorder group (n=50) consisted of study members who met DSM-III-R criteria for either anti-social personality disorder or conduct disorder at age 21.<sup>4</sup> Finally, the attention-deficit disorder (ADD) group consisted of

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<sup>3</sup>DSM anxiety disorders of adulthood that do not overlap with the DSM anxiety disorders of childhood were not assessed at the age 15 follow-up, due to the low expected prevalence rates among subjects not yet adult.

<sup>4</sup>The DSM-III-R specifies that a diagnosis of anti-social personality disorder (ASPD) is an adult disorder and should not be given to individuals under 18 years of age. Conduct disorder may be diagnosed for an individual 18 or over if the individual does not meet criteria for ASPD.

study members who met DSM-III criteria for ADD at 15 (n=20).<sup>5</sup>

In addition to categorical measures, we created continuous scales by summing the study member's scores on interview symptom items relevant to each disorder (see Krueger, Caspi, Moffitt, Silva, and McGee 1996). The reliability (internal consistency) of the scales for all disorders is higher than 0.7, except for anti-social personality disorder, which has a scale reliability of 0.55. Because symptom counts are highly skewed with a mode of zero, they were transformed by taking the log of the count plus one. This transformation changes their interpretation when used in regression equations so that they reference relative, rather than absolute, changes in mental disorder. For example, when used as an independent variable the beta coefficient refers to the change in Y given a one percent change in the disorder scale. When used as a dependent variable, the beta coefficient refers to the percentage change in the disorder scale given a change in the independent variable (Gujarati 1988).

### *Social Status Measures*

We indexed familial socioeconomic status at age 15 with information obtained directly from parents, using New Zealand-specific measures of parents' occupational socioeconomic status, parents' education, and family income. Parents' occupational status was measured with the Elley and Irving (1976) scale, a 6-point scale based on the average income and education levels for 546 occupations of the New Zealand labor force. We assigned scores on the basis of the higher status of either caregiver, whether father or mother. Parents' education was measured with the same scale used by Elley and Irving (1976), which categorizes attainment into three

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<sup>5</sup>Attention deficit disorder was not measured at age 21 because it is not regarded as an adult disorder in the DSM-III-R (American Psychiatric Association 1987).

levels on the basis of primary, secondary, and tertiary degrees. As with occupational status, we assigned the score of the higher-ranking parent. Familial income was measured as the combined gross income of both parents from all sources. The SES index was a linear composite of occupational status, educational attainment, and familial income, using weights from confirmatory factor analysis (the loadings were 0.80, 0.68, and 0.67, respectively).

Study members' educational attainment by age 21 came from their own self-reports. We broke overall educational attainment by age 21 into three separate transitions because the factors influencing educational dropout may differ across educational levels (Mare 1980; Mare 1981). We first examined whether mental disorders influenced study members' performance on the New Zealand School Certificate Examinations. Almost all students sit for these national exams by age 16 because they determine promotion in secondary school and technical schools, and passing also helps secure better employment in the labor market (Kennedy 1981). Eighty seven percent of the sample earned a School Certificate in at least one subject, and among this subsample we then examined the influence of mental disorders on the study member's ability to earn a Sixth Form Certificate, which is comparable to a high school degree in the United States (Kennedy 1981). Seventy six percent of those who earned a School Certificate also earned a Sixth Form Certificate, and among this subsample we then examined the effects of mental disorders on continuing to tertiary education at a university (37% of the study members who earned a Sixth Form Certificate continued to a university education). The variable "Educational Attainment at 21" represents study members' highest educational transition achieved by age 21 (0=no School Certificate, 1=School Certificate, 2=Sixth Form Certificate, 3=university attendance).

Four additional measures were used in our analysis to control for confounding influences.

We included controls for gender and the study member's ability and motivation to continue education. Intelligence was assessed with the Wechsler Intelligence Scale for Children - Revised (Wechsler 1974) between ages 7 and 11 (M=108, sd=15). Academic ability was assessed with the Burt Word Reading Test (Scottish Council for Research in Education 1976) when the subjects were 15 years old (M=91, sd=15). School involvement, an important predictor of educational attainment (Kerckhoff 1993), was measured at age 15 with a visual analogue scale. Respondents were shown a card with five concentric circles, and were asked to suppose that the "circle represents the activities that go on at your school" and asked to rank their distance from "the center of things." A value of 1 indicated little involvement in school activities and a value of 5 indicates study members who considered themselves at "the center of things" (mode=3).

#### *Analytic Strategy*

Our analysis centers on mental disorders during the 12 months prior to the age-15 birthday, the birthday after which the New Zealand adolescents in our study were first legally entitled to leave school, and consequently the age at which they were first at risk of "selection" into the lower social strata. We employed three empirical tests to examine the influences of selection and causation. First, we examined the association between mental disorders at age 15 and family SES background. Second, we examined the extent to which these mental disorders impaired social mobility by evaluating their influence on subsequent educational attainment, using models that contained traditional status attainment controls such as IQ, family SES background, and gender. Third, we examined the extent to which increases in mental disorder between ages 15 and 21 were associated with early adulthood SES, as indexed by educational attainment at age 21. No test by itself provides enough information to discriminate between the three interpretations of

selection, causation, and joint effects, but taken together they lead to discerning patterns of expected results, as outlined below.

– Table 1 about here –

Table 1 provides a summary of empirical tests and expected patterns of findings.

Exclusive evidence for selection would be indicated by the pattern of findings in which, over time, mental disorders impaired the status attainment of study members, but were uninfluenced by socioeconomic standing of origin and socioeconomic standing of early adulthood. Exclusive evidence for social causation would be indicated by the contrasting pattern, in which mental disorders were influenced by both SES of origin and SES of early adulthood, but exerted no significant influence on status attainment. Evidence for the joint effects of selection and causation processes would be indicated by a pattern in which mental disorders were influenced by SES of origin, impaired status attainment, and were additionally influenced by SES of early adulthood. Finally, evidence for the lack of both causation and selection effects in early adulthood would be indicated by nonsignificant associations across all three tests.<sup>6</sup>

## RESULTS

### *Family SES Background and Adolescent Mental Disorders*

The well documented relationship between socioeconomic status and mental disorders

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<sup>6</sup>We had longitudinal data about mental disorders and educational attainment for 939 study members. Of these, no study members were missing data on parental occupational status or parental education, and 12% were missing data on familial income. For study members with missing data on family income, the linear SES composite included family income measures assigned on the basis of parents' occupational status and education. In addition, 2% of the study members were missing data on IQ, and 0.5% and 0.7% were missing data on reading ability and school involvement, respectively. In our multivariate models, we flagged missing data for income, IQ, reading ability, and school involvement with dummy indicators and assigned scores to replace the missing data so that none of the 939 study members would be ejected from our analyses (Little and Rubin 1987). Missing data indicators that were significant in our models are noted in the text.

replicated among the adolescents in our sample. We found that adolescent mental disorders were more likely to be found among youth in families with low socioeconomic status than would be expected by chance alone.

-- Tables 2 and 3 About Here --

We limited our methods in this first section to simple bivariate associations because the association between family SES and mental disorders may represent both selection and causation (Dohrenwend 1975), and we did not know the direction of causality *a priori*. The results are presented separately for diagnostic categories (Table 2) and symptom scales (Table 3). In each table we show the association between measures of mental disorders and the overall family SES index as well as its constituent parts; i.e., parents' occupation level, education level, and income. Overall, adolescents with any DSM-III diagnosis were approximately 0.18 of a standard deviation lower in family SES origins than their peers (column 4, Table 2;  $-.07 - .11 = -.18$ ). Adolescents' total psychiatric symptom scores on the DISC-C schedule were also significantly correlated with their SES background, at a magnitude of 0.16 (column 4, Table 3).

Differences in the relation between socioeconomic status and mental disorders emerged when mental disorders were considered individually. Among the internalizing disorders, anxiety and depression had different associations with family social status. Whether assessed categorically or continuously, anxiety was disproportionately found in families with lower SES as expected, but depression was not. This is consistent with evidence that SES is more strongly related to anxiety than depression across the general population ages 15-54 (Kessler et al. 1994). However, the complete lack of an association between SES and depression among adolescents differs from research indicating the presence of a modest SES/depression association among

adults (Kessler et al. 1994; Dohrenwend et al. 1992; but see also Weissman et al. 1991, who report a nonsignificant SES/depression association).

Among the externalizing disorders, support for an association between family SES and mental disorders was more robust in analyses of attention deficit disorder than conduct disorder. Based on symptom scales, both disorders showed a significant association with socioeconomic status (Table 3), but only attention deficit disorder showed the expected association when measured categorically (Table 2). The DSM-III categorical measure of conduct disorder was not significantly related to overall family SES, but it was negatively associated with parents' occupational status (column 1, Table 2), providing some evidence for an association with socioeconomic status.<sup>7</sup>

In general, the relation between mental disorders and social status was more robust using continuous symptom scales rather than categorical classifications of mental disorders. For example, Table 3 shows that the symptom scales for disorders related to the overall SES measure were associated with all three individual components of the SES measure. In contrast, the diagnostic categories in Table 2 were usually associated with only one or two SES components. We suspect that this difference stems more from methodological than substantive reasons, and reflects the fact that categorical classifications lose information and statistical power by compressing information on mental disorders into a dichotomous measure (see Mirowsky and Ross 1989).

#### *Mental Disorders and Subsequent Educational Attainment*

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<sup>7</sup>Our finding of only a weak association between SES and externalizing behavior problems at mid-adolescence is consistent with other literature reviewed by Tittle and Meier (1991) and Tittle, Villemeay, and Smith (1978).

We next examined the influence of adolescent mental disorders on educational attainment. We found effects that varied by mental illness: internalizing disorders had no effect on educational attainment, while externalizing disorders exerted a strong negative influence.

-- Tables 4 and 5 About Here --

We used mental disorders at age 15 as predictor variables in logistic regression equations modeling educational transitions. We present the results separately for the two different measures of mental disorders: Table 4 presents results using categorical diagnoses, and Table 5 presents results using symptom scales. In each table we analyzed the effects of adolescent mental disorders on three educational transitions: the acquisition of at least one School Certificate, completion of the Sixth Form Certificate, and continuation to university training. For each transition we present two models. The first includes only measures of mental disorders and family SES as predictors of educational achievement. The second includes all the predictors included in the first model, and adds status attainment controls to isolate the unique effects of adolescent mental disorders on educational attainment from other factors implicated in the status attainment process.

The internalizing disorders of anxiety and depression did not significantly affect educational attainment in any of the models, whether using DSM diagnoses or DSM symptom scales. These results suggest that adolescents with internalizing disorders are not “selected” into the lower social strata through truncated education.

In contrast, we found strong evidence for selection processes among adolescents with externalizing disorders. Conduct disorder impaired achievement at every educational transition in this study. Adolescents who met the DSM criteria for a diagnosis of conduct disorder were less

likely to earn a School Certificate by an odds ratio of 4.53, even after controlling for family socioeconomic background and the presence of other comorbid mental disorders (Table 4, Model 1 of the first transition;  $e^{1.51}=4.53$ ).<sup>8</sup> This effect was still strong after status attainment controls were entered into the equation (Table 4, Model 2 of the first transition). Adolescents meeting DSM criteria for a diagnosis of conduct disorder who earned a School Certificate were then significantly less likely to earn a Sixth Form Certificate by an odds ratio of 2.51, after controlling for family socioeconomic background and the presence of other mental disorders (Table 4, Model 1 of the second transition;  $e^{.92}=2.51$ ). Again, this effect was still significant after introducing status attainment controls into the equation (Table 4, Model 2 of the second transition). In the final educational stage of the analysis, we found evidence that adolescents with conduct disorder who overcame the odds against them and received both a School Certificate and a Sixth Form Certificate were later less likely to continue to a university education. Using a DSM diagnosis this effect is significant only in the full model, at the .10 level (Table 4, Model 2 of the third transition). When substituting DSM symptom counts for diagnoses, however, the effect of conduct disorder on educational attainment is significant for all educational transitions, in both the abbreviated and full models. In sum, by the time adolescents with conduct disorder reach adulthood they appear to be “selected” into the lower socioeconomic strata through restricted educational attainment.

Attention deficit disorder also impaired educational attainment, although its effects in our

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<sup>8</sup>Study members who were missing family income data were significantly less likely ( $p<.01$ ) to earn any School Certificate in Model 1 of the first transition in both Tables 3 and 4 ( $\beta=0.89, 0.85$ , respectively). Study members missing data on family income did not significantly differ in their educational attainment in any of the other models.

models were contingent, in part, on its measurement. Using a DSM diagnosis, we found that adolescents with attention deficit disorder (ADD) were less likely to earn a School Certificate by an odds ratio of 5.58 (Table 4, Model 1 of the first transition;  $e^{1.72} = 5.58$ ), although this effect lost statistical significance after introducing the status attainment controls into the equation (Table 4, Model 2 of the first transition). Those adolescents who met DSM criteria for ADD that did earn a School Certificate were then significantly less likely to earn a Sixth Form Certificate (Table 4, Model 1 of the second transition), although, again, this effect was not significant after status attainment controls were introduced into the equation (Table 4, Model 2 of the second transition). We found that adding only IQ and reading ability to Model 1 of the first and second transition was enough to make the effects of ADD lose statistical significance (analyses not shown). The effect of ADD on educational attainment past the sixth form was difficult to measure because the sample size became extremely small. Only three (15%) adolescents with ADD earned both a School Certificate and Sixth Form Certificate, a number too small to enter into our models predicting university attendance.

The results differed in one important way when attention deficit disorder was measured with a symptom count instead of a dichotomous diagnosis. Namely, the symptom count continued to predict failure at educational transitions, even after the status attainment controls were added to the equation (Table 5, Model 2 in transitions 1 and 2). The only models in which ADD did not exert a significant influence were in the equations predicting university attendance (Table 5, Models 1 and 2 of the third transition), when the number of Sixth Form recipients displaying serious ADD symptoms was so small that we believe it precluded us from finding significant effects. In sum, our analysis of ADD indicates strong support for selection effects

early in the educational career of adolescents.

Overall, the controls acted in their predicted directions. Lower socioeconomic background was significantly associated with impaired educational attainment in all models. Higher academic ability, as measured by IQ and reading ability, was associated with greater educational attainment at all educational stages. School involvement at age 15 significantly predicted successful completion of a School Certificate, although its effects were not significant at later educational stages. Finally, we found that women were more likely to earn a School Certificate than men, a finding consistent with government statistics (Department of Education in New Zealand 1987, Table 39).

In models not shown we examined whether the effects of mental disorder on educational attainment differed significantly between men and women. We included multiplicative interaction terms between gender and each disorder in the full models for the three educational transitions.<sup>9</sup> Only one out of 22 interaction terms reached statistical significance at the .05 level, an interaction we do not interpret here because it may have occurred by chance alone. These results suggest that the effects of mental disorder on educational attainment are similar for men and women.

Finally, we also ran models to examine the extent to which comorbidity influenced our results. We found that with and without the comorbid controls in the models, all disorders acted in the same direction and at the same significance levels.

### *Early Adulthood SES and Mental Disorders*

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<sup>9</sup>Only one woman met criteria for attention deficit disorder in our model predicting 6th Form completion and no women met it in our model predicting university attendance. These small ns precluded us from including these two gender interactions in our models using DSM diagnoses as measures of mental illness. We were, however, able to include all gender interactions in our models using DSM symptom scales.

In the third part of the analysis we focused on early adulthood SES, as indexed by educational attainment at age 21, and examined its association with increased disorder between ages 15 and 21. We found respondents with lower educational attainment were more likely to experience increases in anxiety and anti-social disorders, but not depression.

-- Table 6 About Here --

We used logistic and ordinary least squares (OLS) regressions to predict age-21 disorders from educational attainment, statistically controlling the influence of age-15 disorders, gender, and parental SES. For all disorders, model 1 in Table 6 presents results using symptom scales and OLS regression, while model 2 in Table 6 presents results using dichotomous diagnoses and logistic regression.

We found that study members with low educational attainment at age 21 reported significantly higher levels of anxiety, after statistically controlling for age-15 levels of anxiety, parental SES, and gender (Table 6, Anxiety Models 1 and 2). Upon further inspection (not shown) we found that the relationship between educational attainment and anxiety was monotonic: increases in anxiety between ages 15 and 21 were highest among study members with the least educational credentials, and declined monotonically with higher educational attainment. These findings are consistent with the prediction of the causation hypothesis that greater exposure to lower socioeconomic experience increases anxiety disorders.

While slightly higher levels of depression were also found among study members with low educational attainment at age 21, this effect was not significant using either DSM diagnoses or symptom scales (Table 6, Depression Models 1 and 2).

In our final analysis we focused on anti-social disorders and found that they were

overrepresented among respondents with low educational attainment. Because conduct disorder at age 15 may continue as anti-social personality in adulthood (American Psychiatric Association 1987), we used both age-21 conduct disorder and anti-social personality disorder as measures of anti-social disorder in early adulthood. At age 21, study members with low educational attainment were more likely to meet DSM criteria for either conduct disorder or anti-social personality disorder, a finding that persists after statistically controlling the influence of conduct disorder at age 15, parental SES, and gender (Table 6, Anti-Social Disorder Models 1 and 2). Further, we examined age-21 conduct disorder and anti-social personality disorder in two separate models, and in both cases educational attainment significantly ( $p < .05$ ) predicted disorder using both DSM diagnoses and symptom scales, after controlling the influence of age-15 conduct disorder, parental SES, and gender (analysis not shown). In all anti-social disorder models the relationship between education and mental disorder was monotonic. These findings are consistent with the hypothesis that greater exposure to low socioeconomic experience fosters anti-social disorders.

Focusing on the control variables, we found that age-15 levels of disorder strongly predicted age-21 levels of disorder in all models, indicating substantial continuity of mental health problems from adolescence to adulthood (Ollendick and King 1994; McMahon 1994). Low parental SES had no significant effect on mental disorder at age 21, net of the effect of the study members' own educational attainment and past history of mental disorder at age 15. Finally, consistent with the literature we found that women reported significantly higher levels of anxiety and depression than men, and significantly lower levels of anti-social disorder. The only significant gender interaction we found (not shown) was in the model using DSM symptom scales

of anti-social disorder, and it suggests that the effects of low educational attainment on anti-social disorders may be greater for men than women.

## DISCUSSION

The aim of this study was to examine whether low socioeconomic status serves as a cause or consequence of mental illness, and we focused our inquiry on a cohort of adolescents as they made the transition to adulthood. Consistent with previous research in this area we used educational attainment as a proxy for SES in early adulthood (Dohrenwend et al. 1992), a time period when many young adults have not yet realized their potential in terms of other status measures such as income and occupational prestige. Our analysis was especially well suited to study mental disorders and educational attainment for four reasons. First, we used a prospective longitudinal study that allowed us to test between the three interpretations of selection, causation, and joint effects. Second, we evaluated subjects for a wide array of DSM-III disorders, allowing us to examine selection and causation processes across different psychiatric disorders. Third, we studied the sample during the transition from adolescence to young adulthood, a developmental period that includes both the peak onset of psychiatric disorders and the normative educational transitions that constitute a key first step in the status attainment process. Finally, the high response rate of the longitudinal Dunedin study diminished any bias introduced by selective missing data.

We found that the relation between mental disorders and SES is unique for every disorder examined in this study. The main difference centers on selection effects – the extent to which adolescents with mental disorders “select” themselves into the lower social strata through curtailed education. We found no evidence for selection effects among youth with the

internalizing disorders of anxiety and depression. In contrast, we found strong evidence for selection effects among youth with the externalizing disorders of conduct disorder and attention deficit disorder. Below we discuss these and more detailed differences in selection and causation processes across the different disorders, and point out implications for methodology, treatment, and theory.

Before turning to a discussion of the findings, it is important to note three limitations of this study. First, the sample size is too small to support analyses of specific disorders such as social phobias or dysthymia. Accordingly, we limited our analyses to families of specific disorders, i.e. anxiety, depression, anti-social disorders, and attention deficit disorder. Our sample size is also too small to provide a reliable analysis of psychoses such as schizophrenia, which generally do not onset until later in the life course (Robins and Regier 1991). Due to these sample size constraints we have limited our inquiry about selection and causation processes to the most prevalent adolescent mental health disorders.

Second, subjects who dropped out of school may return in the future and finish uncompleted degrees. This potential bias is nominal in our analyses of School Certificate and Sixth Form Certificate completion because very few people attempt to acquire these certificates in later life by returning to school (Department of Education in New Zealand 1987), or through correspondence courses (Department of Labor in New Zealand 1990). However, students who return to education later in life for university training are more common (Department of Labor in New Zealand 1990). Our analysis of university attendance thus represents conservative estimates, and the effects of adolescent mental disorders may be greater than our results suggest if they also deter people from returning to university training in adulthood.

Third, this study requires future replications to support its generalizability beyond New Zealand. To date the selection and causation literature has proceeded on the unstated assumption that cross-national differences in selection and causation processes throughout the Western world are minimal. Fox (1990), for example, presents results from both British and American samples in his analysis of selection processes without noting the possibility of cross-national biases.

Dohrenwend et al. (1992) do not explicitly consider country-specific effects in their Israeli-based research, and neither do Kessler et al. (1995) or Wheaton (1978) in their American-based research. The assumption that selection and causation effects are similar across countries has yet to be tested explicitly; such tests are, of course, contingent on the design of mental health studies with comparable measures and methods. With these qualifications in mind, we discuss our findings by disorder.

### *Anxiety*

In regard to anxiety our pattern of results conforms to the predictions of exclusive social causation (summarized in the second row of Table 1). The influence of causation processes is supported by our findings that SES of origin predicted anxiety at age 15, and that SES of destination by age 21, as measured by educational attainment, predicted increases in anxiety from ages 15 to 21. At the same time, we did not find evidence to indicate the presence of selection processes. The selection interpretation predicts that disorders will impair educational attainment, but anxiety did not hinder young adults' progress through any of the educational transitions analyzed in this study.

These results in support of exclusive social causation are particularly important to the social stress literature. Much of the theory and methodology in this tradition is based on the

assumption that social causation alone accounts for the disproportionately high prevalence rate of anxiety in the lower social strata. If our analysis had indicated that anxiety exerts selection effects it would have provided evidence for the interpretation of joint causation and selection effects, and, if true, much of the theoretical and empirical research in the stress literature would be misspecified. Our analysis provides evidence that the assumption of social causation is appropriate in early adulthood, although it is important for future research to investigate the influence of selection effects through other potential processes later in the life course.

Our research is not consistent with that of Kessler et al. (1995), who conclude that anxiety impairs status attainment through a negative effect on educational attainment. At least three differences between this study and the work of Kessler et al. (1995) may account for the discrepant outcomes. First, cross-national differences may play a role, as discussed above. Second, differences in the measurement of anxiety may also have an effect. The results of Kessler et al. (1995) may reflect the effects of specific anxiety disorders, such as post-traumatic stress disorder, that were not assessed in our study. Third, and perhaps most importantly, we used prospective disorder measures, while those of Kessler et al. (1995) are retrospective. Analyses based on retrospective disorder measures may incorrectly portray causation effects as selection effects because of “state-dependency” - the tendency for individuals to construct their past psychological states to be consistent with their present ones. Current distress may evoke reminders of past disorder, and people who are not currently distressed may forget or deny past disorder (Aneshensel et al. 1987). As a consequence, the fact that adults’ retrospective reports of anxiety are inversely related to educational attainment may represent not only selection effects as Kessler et al. (1995) argue, but also causation processes in adulthood projected into the past.

*Depression*

Our analysis of depression provides support for neither causation nor selection processes, suggesting that SES and depression have little influence on each other before age 21. Depression at age 15 was not overrepresented among lower SES families, it did not influence subsequent educational attainment, and increases in depression between ages 15 and 21 were not significantly overrepresented among study members with low educational attainment. As with anxiety, our outcomes differ from Kessler et al. (1995) in that we did not find evidence for selection effects, a discrepancy that may stem from cross-national differences, psychological measurement differences (our inventory of depressive disorders did not include bi-polar disorder, which contains psychotic symptoms such as delusions and hallucinations), and/or differences between retrospective versus prospective assessments of mental disorders.

Our findings showing no association between SES and depression in adolescence suggests that the SES/depression association found in some studies of adults (Kessler et al. 1995; Dohrenwend et al. 1992, but see also Weissman et al. 1991) may be specific to adulthood, reflecting the consequences of adult-specific processes (Macintyre and West 1991; Rosenberg and Pearlin 1978). For example, the young adults in this sample are not yet old enough to experience the full impact of two of the main factors that place lower-SES adults at increased risk for depression – divorce/separation and becoming trapped in lower status jobs (Link, Lennon, and Dohrenwend 1993; Turner, Wheaton, and Lloyd 1995; Weissman et al. 1996). We highlight these processes as only a starting point for the development of theory that explains age-specific relations between SES and depression; a more detailed treatment may benefit from life-course analysis that examines age-specific mechanisms linking social conditions and mental disorders

(e.g., Elder, George, and Shanahan 1996).

### *Anti-Social Disorders*

The pattern of results for anti-social disorders conforms to the prediction of joint selection and causation effects (summarized in the third row of Table 1). Consistent with the selection hypothesis, we find adolescents with conduct disorder at age 15 are at a higher risk to fail at every educational transition analyzed in this study, and thereby select themselves into the lower social strata. At the same time the results are also consistent with the interpretation of strong causation effects, a finding consistent with Dohrenwend et al. (1992). We found that SES of origin predicted conduct disorder at age 15, and that low SES in early adulthood, as measured by educational attainment, predicted increases in anti-social disorders between ages 15 and 21, as measured by both age-21 conduct disorder and anti-social personality disorder.

With regard to the sociological literature, evidence for selection effects indicates that current sociological theories and methods used to study the internalizing disorders are not appropriate in the study of anti-social disorders. Unlike the internalizing disorders, our results suggest that anti-social disorders adversely affect status attainment by impairing educational attainment. They therefore violate the theoretical assumption of one-way causality between SES and mental illness. They also violate the methodological assumption of one-way causation required by traditional regression models that are currently used in stress research. Our analysis therefore highlights the need for more sophisticated theory and analysis in order for sociological research to expand its focus to encompass a wider range of psychiatric conditions beyond the internalizing disorders, an expansion necessary for a fuller understanding of stress processes (Aneshensel, Rutter, and Lachenbruch 1991).

Three issues are of particular theoretical importance for an expansion of sociological research to the anti-social disorders. First, the evidence for joint selection and causation effects indicates the need to address the reciprocal relation between SES and anti-social disorders over the life course. This will require a shift away from current conceptualizations of individual mental health as a passive consequence of social structure, and a move towards different research topics, such as the influence of anti-social disorders on interactions between individuals and their environment (e.g., Caspi, Elder, and Bem 1987). Second, evidence from the psychological literature strongly suggests that early onset of anti-social disorder in childhood identifies a more severe condition than onset during adolescence or later life stages (Moffitt 1993), suggesting that the results of this study represent an average of these two different groups. Further examination is required to determine the extent to which these groups differ both in terms of their social etiology and their life consequences. Third, antisocial disorders warrant admission to models that predict status attainment, alongside more traditional status-attainment variables such as family SES, IQ, and academic achievement.

With respect to the psychological literature, the findings provide important evidence that the effect of conduct disorder on educational attainment cannot be explained solely by its association with impaired intelligence. The relative influence of these two correlated factors has been debated heatedly (Mandel 1997). By providing evidence showing a unique, negative influence of conduct disorder on educational attainment, our findings highlight the importance of research into possible intervening mechanisms and their relative influence. Candidate mechanisms associated with conduct disorder include teenage pregnancy (Robins and Price 1991; Zoccolillo and Rogers 1991), addiction to drugs and alcohol (Elliott, Huizinga, and Menard 1989), and peer

deviance (Cairns and Cairns 1994), all of which are associated with school dropout (Crane 1991; Kaplan and Liu 1994; Cairns and Cairns 1994) and which may mediate the far-reaching effect of conduct disorder on status attainment. In addition, the effects of conduct disorder on truncated education may also be mediated by the effect that this disorder has on other persons in the social environment; for example, the disruptive behavior of conduct-disordered youth make them more likely to be “thrown out” of school (Cairns and Cairns 1994).

#### *Attention Deficit Disorder*

Our analysis also points to selection processes among youth with attention deficit disorder (ADD). We found evidence that these youth are much less likely to earn a School Certificate or a Sixth Form Certificate, which is comparable to a high school degree in the United States. As with anti-social disorder, evidence for selection effects strongly suggests that the relation between ADD and SES should not be studied in the same manner as the relation between internalizing disorders and SES.

The extent to which IQ and reading ability mediate the effects of ADD on educational underachievement is a matter of debate (Hinshaw 1992), and is sensitive, we show, to the disorder’s measurement. When ADD is measured using a DSM diagnosis its effects on educational attainment are entirely mediated by IQ and reading ability, but when measured with a DSM symptom scale, its effects are only partially mediated. There is a need for a more definitive conception and measurement of ADD before empirical research can shed light on its influence on educational achievement relative to intellectual ability.

#### *Conclusion*

Different mental health problems are differently related to social status. Each adolescent

disorder in this study (anxiety, depression, conduct disorder, and attention deficit disorder) bore a different relation to educational attainment, indicating that differences between disorders extend beyond the simple psychotic/non-psychotic dichotomy currently recognized in sociological research on social selection and social causation. In terms of methodology, our findings suggest that research on social status and mental health has much to gain by incorporating measures of specific mental illness, rather than relying on the omnibus concept and measure of “psychological distress” which combines symptoms of different disorders. Of course, available interview schedules are expensive to administer – and the methods of diagnoses recommended by different classification systems are imperfect – but the alternative to well-conceived mental-health measurement may be misspecified theories of the relations between social status and mental illness.

In terms of treatment, the finding that different psychiatric diagnoses are differently related to social status suggests that the need for interventions and therapeutic strategies varies by disorder. Our results indicate that anxiety and depression do not significantly impair educational attainment, and consequently do not require special intervention programs to counteract their effect on educational attainment. By contrast, conduct disorder and attention deficit disorder uniquely impair educational attainment, and thereby damage the future life chances of the persons they afflict. It is particularly important for treatment regimens targeted at these latter adolescent mental health problems to include interventions that improve school performance and curb the initial slide into downward drift.

In terms of theory, our findings highlight the need for disorder-specific explanations of the relations between social status and mental disorders. Our findings about anxiety and anti-social

disorders underscore the importance of social-causation processes in the life course; the elevated prevalence rate of these disorders in the lower social strata is partly a product of social inequalities and class-related social conditions. These results thus confirm a role for social structure in the etiology of anxiety and anti-social disorder and highlight the need for research to identify the mechanisms that account for these SES differentials. We also find evidence that conduct disorder and attention deficit disorder exert strong selection effects, indicating that they disrupt the status attainment process during the transition from adolescence to adulthood. To the extent that these disorders are passed to offspring, either through socialization or biological factors, they play a heretofore overlooked and powerful role in the reproduction of class structure and social inequality. These selection processes violate the assumption of social causation and point to the need for research to identify the mechanisms that lead to their disruptive intra- and inter- generational patterns of influence.

Sociological research offers a unique perspective on the social causes and consequences of mental disorders. While anxiety and depression have captured much of the sociological imagination, our results suggest that other psychiatric conditions – historically neglected in sociology – merit careful scrutiny in order to more fully understand how social conditions influence individual lives.

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**Table 1:** Expected patterns of evidence for selection and causation processes.

| Type of Evidence                               | Association between parental SES and disorder at age 15 | Effect of disorder at 15 on subsequent educational attainment | Effect of truncated education on increased disorder between 15 and 21 |
|--|---|---|---|
| Exclusive evidence for selection               | no  | yes   | no  |
| Exclusive evidence for causation               | yes   | no  | yes   |
| Evidence for joint effects                     | yes   | yes   | yes   |
| Evidence for no effects during early adulthood | no  | no  | no  |

**Table 2.** DSM-III Diagnoses At Age 15 and Social Class: Mean Level Comparisons Between Disordered and Non-Disordered Adolescents<sup>a</sup>

|                                    | Parents' Mean Occupational Status | Parents' Mean Education Level | Parents' Mean Income | Parents' Mean Composite SES <sup>b</sup> | n                |
|------------------------------------|-----------------------------------|-------------------------------|----------------------|--|------------------|
| Mean                               | 4.00                              | 0.90                          | 36,070               | 0.03                                     | 939              |
| Standard Deviation                 | 1.25                              | 0.62                          | 16,082               | 0.99                                     | 939              |
| Range                              | 1-6                               | 0-2                           | 0-80, 000            | -2.49-2.30                               |                  |
| <hr/>                              |                                   |                               |                      |  |                  |
| Any DSM-III Disorder               | 3.78**                            | 0.83                          | 35,487               | -0.11*                                   | 207 <sup>c</sup> |
| No Disorder                        | 4.06                              | 0.90                          | 36,235               | 0.07                                     | 732              |
| <b>Internalizing Disorders</b>     |                                   |                               |                      |  |                  |
| DSM-III Anxiety                    | 3.74*                             | 0.81                          | 34,338               | -0.16*                                   | 100              |
| No Anxiety                         | 4.03                              | 0.90                          | 36,276               | 0.05                                     | 839              |
| DSM-III Depression                 | 3.81                              | 0.84                          | 36,383               | -0.07                                    | 37               |
| No Depression                      | 4.01                              | 0.89                          | 36,057               | 0.04                                     | 902              |
| <b>Externalizing Disorders</b>     |                                   |                               |                      |  |                  |
| DSM-III Conduct Disorder           | 3.78+                             | 0.89                          | 37,292               | -0.07                                    | 81               |
| No Conduct Disorder                | 4.02                              | 0.84                          | 35,954               | 0.04                                     | 858              |
| DSM-III Attention Deficit Disorder | 3.45*                             | 0.60*                         | 31,275+              | -0.42*                                   | 20               |
| No Attention Deficit Disorder      | 4.01                              | 0.89                          | 36,174               | 0.04                                     | 919              |

a Differences between group means are evaluated using t-tests for all variables except for parents' education, which is categorical and evaluated using the Mantel-Haenszel Chi-Square.

b The mean composite SES measure has been transformed to z-scores.

c Due to comorbidity, the number of study members who met DSM criteria for "any disorder" is less than the sum of study members who met criteria for individual disorders.

+ p<.10

\* p<.05

\*\* p<.01

**Table 3.** Number of DSM-III Mental Disorder Symptoms at Age 15 (Logged) and Social Class: Bivariate Correlations (n=931).

| Types of Symptom              | Parents' Occupational Status | Parents' Education | Parents' Income | Parents' Composite SES |
|-------------------------------|------------------------------|--------------------|-----------------|------------------------|
| Total Symptoms                | -.016**                      | -0.12**            | -0.10**         | -0.16**                |
| <i>Internalizing Symptoms</i> |                              |                    |                 |                        |
| Anxiety                       | -0.12**                      | -0.10**            | -0.07*          | -0.12**                |
| Depression                    | 0.02                         | 0.05               | 0.03            | 0.03                   |
| <i>Externalizing Symptoms</i> |                              |                    |                 |                        |
| Conduct Disorder              | -0.15**                      | -0.11**            | -0.11**         | -0.15**                |
| Attention Deficit Disorder    | -0.13**                      | -0.12**            | -0.08*          | -0.13**                |

\* p<.05

\*\* p<.01

**Table 4.** DSM Diagnoses at Age 15 as Predictors of Educational Attainment: Unstandardized Coefficients from Logistic Regression Equations.

| Variable                       | Transition 1:<br>Failure to Earn Any<br>School Certificate |         | Transition 2:<br>Failure to Earn Sixth Form <sup>a</sup><br>Among School Certificate<br>Recipients |         | Transition 3:<br>Failure to Enter University<br>Among Sixth Form Recipients |         |
|--------------------------------|--|---------|--|---------|---|---------|
|                                | Model 1  | Model 2 | Model 1  | Model 2 | Model 1   | Model 2 |
| <i>Internalizing Diagnoses</i> |  |         |  |         |   |         |
| Anxiety                        | -0.14  | -0.23   | 0.26   | 0.28    | 0.29  | 0.25    |
| Depression                     | 0.28   | 0.48    | -0.04  | 0.16    | -0.62   | -0.49   |
| <i>Externalizing Diagnoses</i> |  |         |  |         |   |         |
| Conduct Disorder               | 1.51**   | 1.59**  | 0.92**   | 1.21**  | 0.84  | 1.04+   |
| Attention Deficit Disorder     | 1.72**   | 0.36    | 1.98**   | 1.57    | --  | --      |
| <i>Controls</i>                |  |         |  |         |   |         |
| Family SES at 15               | -0.92**  | -0.76** | -0.57**  | -0.40** | -0.52**   | -0.32** |
| Female                         | --   | -0.77** | --   | -0.41+  | --  | -0.08   |
| IQ                             | --   | -0.04** | --   | -0.06** | --  | -0.04** |
| Reading Ability                | --   | -0.04** | --   | -0.03** | --  | -0.04** |
| School Involvement             | --   | -0.49** | --   | -0.20   | --  | -0.22   |
| Intercept                      | -2.57**  | 6.58**  | -1.37**  | 8.22**  | 0.66**  | 10.01** |
| n                              | 939  | 939     | 815  | 815     | 617   | 617     |

<sup>a</sup> The New Zealand Sixth Form certificate is comparable to a U.S. high school diploma (Kennedy, 1981)

-- Variable not included in Model 1, and included in Model 2.

+ p<.10

\* p<.05

\*\* p<.01

**Table 5.** Number of DSM-III Mental Disorder Symptoms at Age 15 (Logged) as Predictors of Educational Attainment: Unstandardized Coefficients from Logistic Regression Equations.

| Variable                       | Transition 1:<br>Failure to Earn Any<br>School Certificate |         | Transition 2:<br>Failure to Earn Sixth Form <sup>a</sup><br>Among School Certificate<br>Recipients |         | Transition 3:<br>Failure to Enter University<br>Among Sixth Form Recipients |         |
|--------------------------------|--|---------|--|---------|---|---------|
|                                | Model 1  | Model 2 | Model 1  | Model 2 | Model 1   | Model 2 |
| <i>Internalizing Diagnoses</i> |  |         |  |         |   |         |
| Anxiety                        | -0.14  | -0.24   | -0.11  | -0.25   | -0.05   | -0.16   |
| Depression                     | -0.19  | 0.03    | -0.18  | -0.07   | -0.08   | -0.00   |
| <i>Externalizing Diagnoses</i> |  |         |  |         |   |         |
| Conduct Disorder               | 0.54**   | 0.47**  | 0.35**   | 0.40**  | 0.29*   | 0.35*   |
| Attention Deficit Disorder     | 0.90**   | 0.64*   | 0.92**   | 0.74**  | 0.25  | 0.11    |
| <i>Controls</i>                |  |         |  |         |   |         |
| Family SES at 15               | -0.86**  | -0.70** | -0.52**  | -0.34*  | -0.49**   | -0.32** |
| Female                         | --   | -0.59*  | --   | -0.19   | --  | -0.03   |
| IQ                             | --   | -0.04** | --   | -0.06** | --  | -0.04** |
| Reading Ability                | --   | -0.04** | --   | -0.03** | --  | -0.04** |
| School Involvement             | --   | -0.48** | --   | -0.17   | --  | -0.24   |
| Intercept                      | -4.47**  | 5.08**  | -3.08**  | 6.63**  | 0.13  | 9.24**  |
| n                              | 931  | 931     | 806  | 806     | 613   | 613     |

<sup>a</sup> The New Zealand Sixth Form certificate is comparable to a U.S. high school diploma (Kennedy, 1981)

-- Variable not included in Model 1, and included in Model 2.

\* p<.05

\*\* p<.01

**Table 6.** Educational Attainment as a Predictor of Mental Disorder at age 21: Unstandardized Coefficients from Regression Equations<sup>a</sup>

| Variable                     | Anxiety at 21      |         | Depression at 21 |         | Anti-Social Disorder at 21 |         |
|------------------------------|--------------------|---------|------------------|---------|----------------------------|---------|
|                              | Model 1            | Model 2 | Model 1          | Model 2 | Model 1                    | Model 2 |
| Educational Attainment at 21 | -0.20 <sup>+</sup> | -0.15** | -0.08            | -0.04   | -0.72**                    | -0.08** |
| <i>Controls</i>              |                    |         |                  |         |                            |         |
| Disorder at 15 <sup>b</sup>  | 1.35**             | 0.57**  | 1.56**           | 0.29**  | 1.27**                     | 0.24**  |
| Family SES at 15             | -0.18              | 0.01    | -0.02            | -0.08   | 0.15                       | 0.01    |
| Female                       | 0.87**             | 0.28**  | 0.86**           | 0.31**  | -2.52**                    | -0.33** |
| Intercept                    | -1.70**            | 1.05**  | -1.93**          | 0.87**  | -1.31**                    | 0.92**  |
| n                            | 903                | 893     | 903              | 896     | 905                        | 916     |

+ p<.10  
 \* p<.05  
 \*\* p<.01

<sup>a</sup> Model 1 uses dichotomous DSM diagnoses as measures of mental disorder at ages 15 and 21, and coefficients are from logistic regression equations. Model 2 uses continuous DSM symptom scales (logged) as measures of mental disorders at ages 15 and 21, and coefficients are from ordinary least squares equations.

<sup>b</sup> In models predicting age-21 anxiety, depression, and anti-social disorders, control disorders are age-15 anxiety, depression, and conduct disorder, respectively.

Center for Demography and Ecology  
University of Wisconsin  
1180 Observatory Drive Rm. 4412  
Madison, WI 53706-1393  
U.S.A.  
608/262-2182  
FAX 608/262-8400  
comments to: [rmiech@ssc.wisc.edu](mailto:rmiech@ssc.wisc.edu)  
requests to: [cdepubs@ssc.wisc.edu](mailto:cdepubs@ssc.wisc.edu)