A Longitudinal Investigation of Social Causation and Social Selection Processes Involved in the Association Between Socioeconomic Status and Psychiatric Disorders

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Social causation theory and social selection theory have been put forth to explain the finding that low socioeconomic status (SES) is associated with risk for psychiatric disorders. The predictions of both theories were investigated using data from a community-based longitudinal study. Psychosocial interviews were administered to 736 families from 2 counties in New York State in 1975, 1983, 1985–1986, and 1991–1993. Results indicated that (a) low family SES was associated with risk for offspring anxiety, depressive, disruptive, and personality disorders after offspring IQ and parental psychopathology were controlled, and (b) offspring disruptive and substance use disorders were associated with risk for poor educational attainment after offspring IQ and parental psychopathology were controlled. These findings indicate that social causation and social selection processes vary in importance among different categories of psychiatric disorders.

Considerable evidence has established that low socioeconomic status (SES), assessed using measures of educational attainment, income, and occupational status, tends to be associated with a high prevalence of psychiatric disorders among children, adolescents, and adults (e.g., Costello et al., 1996; Kessler, Foster, Saunders, & Stang, 1995; Robins & Regier, 1991). Two major theories have been advanced to explain this association. Social causation theory hypothesizes that environmental adversity, disadvantage, and stress associated with low SES contribute to the onset of psychiatric disorders (e.g., Hollingshead & Redlich, 1958). Social selection theory hypothesizes that constitutional and environmental factors contribute to the onset of psychiatric disorders, which in turn cause individuals to experience downward drift in SES or to fail to rise out of low SES (e.g., Wender, Rosenthal, Kety, Schul-singer, & Welner, 1973).

Research has provided support for both theories. Support for social causation theory has been provided by longitudinal studies indicating that loss of income and low SES predict adverse mental health outcomes (e.g., Catalano, Dooley, Wilson, & Hough, 1993; Dodge, Pettit, & Bates, 1994; Fenwick & Tausig, 1994; Hamilton, Broman, Hoffman, & Renner, 1990; Link & Phelan, 1995; Loebner, Green, Keenan, & Lahey, 1995; Shaw, Winslow, Owens, & Hood, 1998) and by quasi-experimental findings that are consistent with the predictions of social causation theory for some psychiatric disorders (Dohrenwend et al., 1992). Support for social selection theory has been provided by evidence indicating that genetic factors are associated with risk for some psychiatric disorders that are inversely associated with SES (e.g., Wender et al., 1986), by findings of cross-sectional and quasi-experimental research that are consistent with the predictions of social selection theory for some psychiatric disorders (e.g., Dohrenwend et al., 1992; Kessler et al., 1995), and by social mobility research indicating that some psychiatric disorders predict poor educational and occupational outcomes (e.g., Miech, Caspi, Moffitt, Wright, & Silva, 1999).

Such findings have indicated that a combination of social causation and social selection processes may account for the association between SES and psychiatric disorders (Dohrenwend & Dohrenwend, 1969; Plomin, 1990). However, only one previous study, a large cross-sectional investigation, has investigated differences in the relative effects of causation and selection processes across diagnostic categories (Dohrenwend et al., 1992). We report findings from a community-based prospective longitudinal study to investigate the predictions of social causation and social selection theories regarding the association between parental SES and offspring psychiatric disorders and the association between offspring psychiatric disorders and subsequent educational attainment.

Because both social causation and social selection theories concern sequences of events occurring over an extended period of time, one method for testing the theories’ predictions uses longitudinal data to investigate whether low parental SES predicts onset of offspring psychiatric disorders during childhood and adolescence and whether these disorders predict subsequent declines in offspring SES. Although it is problematic to make causal inferences without using an experimental or quasi-experimental design, use of a prospective longitudinal design to investigate these pre-
dictions can be optimized with (a) a large community sample, (b) assessment of parental psychopathology and parental SES during the childhood and adolescence of the offspring, (c) assessment of offspring IQ and psychiatric disorders during childhood and adolescence, and (d) assessment of offspring SES during adulthood (Dohrenwend & Dohrenwend, 1969).

This type of design has a number of methodological advantages. First, because many of the processes hypothesized by social causation theory (e.g., poor school atmosphere, child neglect) and social selection theory (e.g., distractibility in the classroom, failure to complete homework) take place during childhood and adolescence, it is particularly revealing to investigate these processes during this developmental period. Second, if parental education and occupational status should be associated with onset of psychiatric disorders among offspring, it would be implausible to attribute this relationship to the impact of offspring psychiatric disorders on parental education and occupational status. Third, if parental SES should predict onset of psychiatric disorders among offspring, an alternative hypothesis—that this association is due to negative effects of parental psychopathology on parental SES and to genetic transmission of psychopathology between parents and their offspring—can be investigated. Fourth, another alternative explanation—that the association between parental SES and offspring psychiatric disorders is due to negative effects of common constitutional or environmental factors associated with low IQ on both parental SES and offspring psychopathology—can be investigated.

Because it includes the systematic and repeated assessment of parental SES, parental and offspring psychopathology, offspring IQ, and offspring educational attainment, the present study permits investigation of the following research questions: (a) Are the offspring of low-SES parents more likely than the offspring of higher SES parents to develop psychiatric disorders? Offspring age and gender are controlled because they are known to be associated with risk for psychiatric disorders during childhood and adolescence (Cohen et al., 1993; Costello et al., 1996). Single-parent status is controlled to investigate whether the effects of parental SES are independent of the effects of single-parent status, which is associated with low parental income and which may increase risk for psychopathology among offspring. Offspring IQ and parental psychopathology are controlled because they are potential third variables that might account for the association between parental SES and offspring psychiatric disorders. (b) Are children and adolescents with specific types of psychiatric disorders more likely to experience low educational attainment during late adolescence and young adulthood? Single-parent status, parental psychopathology, parental SES, and offspring age, gender, and IQ are controlled to investigate whether the adverse effects of offspring psychiatric disorders on educational attainment are independent of the potential association of these variables with offspring psychopathology and offspring SES.

According to social causation theory, adversities associated with low SES, such as exposure to environmental trauma (Dodge, Bates, & Pettit, 1990), should cause the offspring of low-SES parents to be more likely than those of higher SES parents to develop psychiatric disorders during childhood and adolescence, independent of the effects of single-parent status, parental psychopathology, and offspring age, gender, and IQ. According to social selection theory, constitutional and environmental factors contrib-
disorder, and conduct disorder); and substance use disorders. Because use of multiple informants has been found to increase the reliability and validity of psychiatric diagnoses (Bird, Gould, & Staghezza, 1992; Placentini, Cohen, & Cohen, 1992), mother and offspring responses to the DISC-I were combined. Each diagnostic criterion was assessed using what is commonly referred to as the “or rule”: A positive indication by either informant was accepted as valid. To minimize the number of false positive diagnoses, we added, as an additional criterion for each diagnosis, a requirement that a score of at least two standard deviations above the mean on the corresponding symptom scale must also be present. The scales used for this purpose incorporated all of the diagnosis-specific symptoms and impairment questions asked of either respondent. There was a moderate correlation between the number of diagnostic criteria identified as present from the offspring and maternal interviews ($r = .45, p < .05$). As Cantwell, Lewinsohn, Rohde, and Seeley (1997) have stated, the importance of conducting diagnostic interviews with parents and their offspring is due in part to the different perceptions that parents and their offspring often have regarding the behavior of the offspring. Previous research has supported the diagnostic reliability and validity of the DISC-I (Cohen, O'Connor, Lewis, & Malachowski, 1987) and of the diagnostic algorithms used in the present study (Cohen, Velez, Kohn, Schwab-Stone, & Johnson, 1987).

Interview items used to assess personality disorders in 1983 were drawn from the DISC-I and the Personality Diagnostic Questionnaire (Hyler et al., 1988). Supplementary items from the Structured Clinical Interview for DSM-III-R Personality Disorders (SCID-II; Spitzer & Williams, 1986) were added in 1985–1986 to cover the diagnostic criteria of the revised third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R; American Psychiatric Association, 1987). In both 1983 and 1985–1986, Cluster A (paranoid, schizoid, and schizotypal), Cluster B (antisocial, borderline, histrionic, and narcissistic), and Cluster C (avoidant, dependent, obsessive-compulsive, and passive-aggressive) personality disorders were assessed. Diagnoses were computed with diagnostic algorithms that combined information from offspring and their mothers, using the same types of diagnostic algorithms as those described in the paragraph above regarding the DISC-I. Previous research has supported the reliability and validity of personality disorder diagnoses obtained using these instruments and procedures. Internal consistency coefficients (Cronbach’s $\alpha$) are satisfactory, ranging from .42 to .70 for the personality disorder symptom scales (Bernstein et al., 1993). Every personality disorder predicted five or more types of social impairment, school or work problems, and psychopathology (Bernstein et al., 1993).

Parental psychopathology was assessed using four instruments: (a) Current parental emotional problems were assessed in 1983 and 1985–1986 using the Hopkins Symptom Checklist (SCL-90; Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974) Anxiety, Depression, and Interpersonal Difficulties subscales; (b) parental substance abuse was assessed in 1983 and 1985–1986 in the maternal interview; (c) parental problems involving police officers were assessed in 1975, 1983, and 1985–1986 in the maternal interview; and (d) parental history of psychiatric disorders was assessed in 1983 and 1985–1986 with items assessing whether the parents had ever been treated for a mental disorder. Parental psychopathology was considered present if either significant emotional problems, substance abuse, or problems involving police officers were present in either parent in 1975, 1983 or 1985–1986; or if, in 1985–1986, either parent had ever been treated for a mental disorder.

Parental SES was assessed in 1975, 1983, and 1985–1986 across three separate domains: (a) maternal and paternal years of education, (b) maternal and paternal occupational status, and (c) family income (Hollingshead & Redlich, 1958). Continuous and dichotomous versions of these variables were used to investigate whether the effects of SES characterized the entire range of SES or were associated only with the presence or absence of low SES. For parental income, the mean percentage of 1975, 1983, and 1985–1986 U.S. poverty levels (USPL) was computed in 1975, 1983, and 1985–1986. Then, each family was assigned to one of the following categories: (a) $< 100\%$ USPL or (b) $> 100\%$ USPL. In addition to the mean number of years of parental education, we investigated differences between offspring of parents with and without a high school education. The highest occupational status attained by either parent in 1975, 1983, and 1985–1986 was identified on a 7-point scale and assigned to one of two categories: (a) $\geq$ skilled worker or (b) $<$ skilled worker (Hollingshead & Redlich, 1958). If any data were missing, the computational procedures were conducted using available data. For example, if data regarding the father’s income were not available, the mother’s mean income was used. Offspring SES was assessed in 1991–1993 with interview questions regarding failure to complete high school and whether offspring had completed 1 or more years of education after high school. Offspring verbal IQ was assessed in 1983 and 1985–1986 using a picture-vocabulary test (Ammons & Ammons, 1962). IQ scores were averaged across the two assessments.

Results

Tests of the Social Causation Hypothesis

Statistical procedures. Analyses of contingency tables and logistic regression analyses were conducted to investigate the social causation hypothesis that youths with low-SES parents would have a higher prevalence of psychiatric disorders than youths with higher SES parents. Logistic regression analyses were conducted to determine whether parental income, education, and occupational status remained significantly associated with the prevalence of youth psychiatric disorders after controlling for single-parent status, parental psychopathology, and offspring age, gender, and IQ. In these analyses, offspring psychiatric disorders were considered to be present if they were diagnosed in either 1983 or 1985–1986.

Anxiety and depressive disorders. Fifty-three youths were diagnosed with anxiety disorders, and 53 were diagnosed with depressive disorders. Logistic regression analyses indicated that low parental education (continuous variable) and low parental occupational status (dichotomous variable), but not low parental income, were associated with increased risk for offspring anxiety disorders after single-parent status, parental psychopathology, and offspring age, gender, and IQ were controlled statistically (see Table 1). Similarly, low parental education (dichotomous variable) and low parental occupational status (continuous variable), but not parental income, were significantly associated with increased risk for depressive disorders among the offspring after the covariates were accounted for. To interpret the differences between the findings obtained with the continuous and dichotomous versions of the SES variables, it should be noted that a significant association with a continuous SES variable indicates that decreases along the entire range in an SES variable are associated with increases in risk for psychiatric disorder. In contrast, a significant association with a dichotomous SES variable indicates that offspring of parents with very low SES are at greater risk for psychiatric disorder than offspring of parents in higher SES groups. Because low parental education and occupational status predicted offspring anxiety and depressive disorders, the present findings are consistent with the social causation hypothesis with regard to anxiety and depressive disorders.

Disruptive and personality disorders. In 1983 or 1985–1986, 83 youths were diagnosed with disruptive disorders and 193 were diagnosed with personality disorders. Low parental education, low parental income, and low parental occupational status
### Table 1

**Association Between Parental Socioeconomic Status and Psychiatric Disorders Among Offspring During Childhood and Adolescence**

<table>
<thead>
<tr>
<th>Offspring psychiatric disorder</th>
<th>% prevalence in offspring of college graduates (n = 108)</th>
<th>% prevalence in offspring of parents without H.S. diploma (n = 230)</th>
<th>Years of parental education</th>
<th>No high school diploma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety disorders</td>
<td>1</td>
<td>9</td>
<td>1.16, 1.03-1.30</td>
<td>1.48, 0.83-2.62, 1.39, 0.75-2.58</td>
</tr>
<tr>
<td>Depressive disorders</td>
<td>5</td>
<td>12</td>
<td>1.17, 1.05-1.31</td>
<td>2.25, 1.32-3.81, 1.77, 1.01-3.17</td>
</tr>
<tr>
<td>Disruptive disorders</td>
<td>9</td>
<td>22</td>
<td>1.22, 1.11-1.33</td>
<td>3.26, 2.11-5.04, 2.34, 1.45-3.82</td>
</tr>
<tr>
<td>Substance use disorders</td>
<td>5</td>
<td>9</td>
<td>1.04, 0.92-1.17</td>
<td>1.75, 0.97-3.16, 1.38, 0.67-2.63</td>
</tr>
<tr>
<td>Personality disorders</td>
<td>21</td>
<td>46</td>
<td>1.21, 1.13-1.30</td>
<td>3.02, 3.16-4.21, 2.33, 1.62-3.35</td>
</tr>
</tbody>
</table>

**Low parental income**

<table>
<thead>
<tr>
<th>Offspring psychiatric disorder</th>
<th>% prevalence in families with incomes ≥4 times the poverty level (n = 130)</th>
<th>% prevalence in families with incomes below the poverty level (n = 78)</th>
<th>Income as percentage of poverty level</th>
<th>Income as below vs. above poverty</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety disorders</td>
<td>5</td>
<td>10</td>
<td>1.13, 0.92-1.40, 1.11, 0.88-1.38</td>
<td>1.68, 0.76-3.71, 1.74, 0.70-4.31</td>
</tr>
<tr>
<td>Depressive disorders</td>
<td>5</td>
<td>14</td>
<td>1.23, 1.00-1.51, 1.14, 0.92-1.41</td>
<td>2.23, 1.11-4.50, 1.88, 0.83-4.24</td>
</tr>
<tr>
<td>Disruptive disorders</td>
<td>6</td>
<td>28</td>
<td>1.61, 1.32-1.96, 1.34, 1.09-1.65</td>
<td>3.45, 1.99-5.96, 1.99, 1.05-3.79</td>
</tr>
<tr>
<td>Substance use disorders</td>
<td>2</td>
<td>6</td>
<td>1.15, 0.92-1.43, 1.25, 0.96-1.63</td>
<td>1.08, 0.42-2.82, 1.53, 0.48-4.85</td>
</tr>
<tr>
<td>Personality disorders</td>
<td>14</td>
<td>53</td>
<td>1.44, 1.26-1.64, 1.25, 1.05-1.43</td>
<td>3.18, 1.97-5.14, 1.97, 1.13-3.41</td>
</tr>
</tbody>
</table>

**Low parental occupational status**

<table>
<thead>
<tr>
<th>Offspring psychiatric disorder</th>
<th>% prevalence in offspring of parents with high occupational status (n = 346)</th>
<th>% prevalence in offspring of parents with low occupational status (n = 92)</th>
<th>Scaled parental occupational status</th>
<th>Unskilled vs. skilled occupation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety disorders</td>
<td>5</td>
<td>13</td>
<td>1.27, 1.05-1.53, 1.24, 0.99-1.54</td>
<td>2.41, 1.22-4.78, 2.39, 1.14-5.02</td>
</tr>
<tr>
<td>Depressive disorders</td>
<td>4</td>
<td>13</td>
<td>1.34, 1.12-1.61, 1.27, 1.03-1.56</td>
<td>2.03, 1.03-3.98, 1.88, 0.91-3.81</td>
</tr>
<tr>
<td>Disruptive disorders</td>
<td>8</td>
<td>26</td>
<td>1.35, 1.16-1.57, 1.23, 1.04-1.45</td>
<td>3.08, 1.82-5.21, 2.46, 1.36-4.46</td>
</tr>
<tr>
<td>Substance use disorders</td>
<td>2</td>
<td>6</td>
<td>1.06, 0.87-1.29, 1.05, 0.83-1.34</td>
<td>1.09, 0.45-2.65, 1.34, 0.51-4.30</td>
</tr>
<tr>
<td>Personality disorders</td>
<td>21</td>
<td>52</td>
<td>1.36, 1.22-1.51, 1.22, 1.08-1.37</td>
<td>3.20, 2.04-5.03, 2.43, 1.48-3.98</td>
</tr>
</tbody>
</table>

**Note.** H.S. = high school; OR = unadjusted (zero-order) odds ratio; CI = confidence interval; AOR = adjusted odds ratio, controlling for the age and sex of the child, the child's IQ, single-parent status, and parental psychopathology. Odds ratios are defined as the odds that a case has that condition divided by the odds that a case does not have that condition. They are considered statistically significant if the number 1.0 falls outside the 95% confidence limits. Odds ratios for continuous variables are odds associated with a one-unit change in the independent variable.

* Mean years of formal education for both parents, assessed in 1975, 1983, and 1985–1986; reverse-scored, so that college graduation was coded as zero. 
* Mean parental education was assigned to one of two categories: <high school education or ≥high school education. 
* Youths in families that were below the poverty level were in the lowest risk group; those whose parents were above poverty level were in the highest risk group. 
* Mean % of U.S. poverty level in 1975, 1983, and 1985–1986 was assigned to one of the following categories: ≤100% of poverty level or >100% of poverty level.  
* The highest occupational status attained by one or both parents, assessed in 1975, 1983, and 1985–1986 with a 7-point scale and reverse-scored so that the highest occupational status was coded as zero. 
* Highest occupational status attained by either parent, assigned to one of two categories: ≥skilled worker or <skilled worker.

Predicted risk for both offspring disruptive and personality disorders, controlling for single-parent status, parental psychopathology, and offspring age, gender, and IQ (see Table 1). Supplemental analyses indicated that parental education, income, and occupational status all predicted offspring Clusters A, B, and C personality disorders. Therefore, the present findings are consistent with the social causation hypothesis with regard to the disruptive and personality disorders.
Values represent the percentage of offspring with the psychiatric disorder or low educational attainment.

Note.

Association Between Study Covariates and Prevalence of Psychiatric Disorders and Low Educational Attainment

"Psychiatric disorder present in either 1983 or 1985-1986."

Among Offspring (in Percentages)

Failure to continue education beyond high school

\[ p < .05. \quad ** p < .01. \quad *** p < .001. \quad **** p < .0001. \]

Psychiatric disorders. Forty-three adolescents were diagnosed with substance use disorders in 1983 or 1985–1986. Of these, 41 were diagnosed with alcohol abuse, 19 with marijuana abuse, and 8 with both alcohol and marijuana abuse. None of the three measures of parental SES was significantly associated with offspring substance abuse disorders, either before or after the covariates were controlled (see Table 1). Supplemental analyses indicated that parental SES did not predict either alcohol abuse or marijuana abuse. Therefore, the present findings are not consistent with the social causation hypothesis that risk for substance use disorders is highest among adolescents from low-SES families.

Associations of covariates with offspring psychiatric disorders. Low offspring IQ predicted offspring personality disorders (adjusted odds ratio [AOR] = 1.03; 95% confidence interval [95% CI] = 1.02–1.04), but it did not predict offspring anxiety, depressive, disruptive, or substance use disorders after single-parent status, parental psychopathology, parental SES, and offspring age, gender, and comorbid psychiatric disorders were controlled statistically. As Table 2 indicates, the prevalences of anxiety, depressive, disruptive, and personality disorders were elevated among offspring of parents with psychopathology. However, after the other covariates were controlled statistically, parental psychopathology continued to predict offspring anxiety (AOR = 2.44; 95% CI = 1.29–4.60) and disruptive disorders (AOR = 1.75; 95% CI = 1.04–2.96) but no longer predicted offspring depressive or personality disorders. All of the tests of the social causation hypothesis were repeated with a version of the parental psychopathology variable that did not include parental problems with the police as a component. The results of these analyses did not differ from those reported above and in Tables 1 and 2. As Table 2 indicates, the prevalences of some psychiatric disorders were associated with offspring gender and presence of a single parent.

Tests of the Social Selection Hypothesis

Statistical procedures. Analyses of contingency tables and logistic regression analyses were conducted to investigate the social selection hypothesis that youths with psychiatric disorders would have lower educational attainment than youths without psychiatric disorders. Logistic regression analyses were conducted to determine whether youth psychiatric disorders, assessed in 1983 and 1985–1986, predicted low educational attainment in 1991–1993 after controlling for single-parent status, parental psychopathology and SES, and offspring age, gender, IQ, and comorbid psychiatric disorders. In these and all of the following analyses reported below, two indexes of educational attainment were used: (a) high school dropout status \((n = 736)\) and (b) failure to continue education beyond high school, assessed to youths who were at least 20 years old in 1991–1993 \((n = 493)\). Analyses were also conducted to investigate (a) whether parental psychopathology predicted youths’ educational attainment, after controlling for parental SES, single-parent status, and youths’ age, gender, and IQ; and (b) whether offspring psychiatric disorders interacted with parental SES, parental psychopathology, or both, to predict low educational attainment.

Anxiety and depressive disorders. As Table 3 indicates, youths who were diagnosed with anxiety or depressive disorders during adolescence were not more likely than adolescents without these disorders to subsequently drop out of high school or to fail to continue their education beyond high school, either before or after the covariates were accounted for. To the contrary, after the covariates were controlled, offspring anxiety and depressive disorders were associated with decreased risk for high school dropout. Therefore, the present findings are not consistent with the social selection hypothesis with regard to the anxiety and depressive disorders. Neither anxiety nor depressive disorders interacted with

<table>
<thead>
<tr>
<th>Variable</th>
<th>Boys</th>
<th>Girls</th>
<th>( \chi^2 )</th>
<th>Both parents present</th>
<th>Single parent present</th>
<th>Offspring IQ ( \geq 90 )</th>
<th>Offspring IQ ( &lt; 90 )</th>
<th>Parental psychopathology Absent</th>
<th>Parental psychopathology Present</th>
<th>( \chi^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety</td>
<td>4.9</td>
<td>8.7</td>
<td>4.60*</td>
<td>7.3</td>
<td>4.9</td>
<td>1.29</td>
<td>6.5</td>
<td>8.4</td>
<td>0.72</td>
<td>4.4</td>
</tr>
<tr>
<td>Depressive</td>
<td>5.7</td>
<td>9.7</td>
<td>4.59*</td>
<td>7.3</td>
<td>8.7</td>
<td>0.37</td>
<td>6.3</td>
<td>12.9</td>
<td>7.71**</td>
<td>5.3</td>
</tr>
<tr>
<td>Disruptive</td>
<td>14.9</td>
<td>8.9</td>
<td>6.73**</td>
<td>8.9</td>
<td>22.3</td>
<td>24.10****</td>
<td>10.1</td>
<td>19.4</td>
<td>10.18**</td>
<td>7.5</td>
</tr>
<tr>
<td>Personality</td>
<td>26.5</td>
<td>30.6</td>
<td>1.61</td>
<td>25.5</td>
<td>38.3</td>
<td>11.11****</td>
<td>23.6</td>
<td>48.7</td>
<td>37.76****</td>
<td>23.8</td>
</tr>
<tr>
<td>Substance use</td>
<td>7.6</td>
<td>4.6</td>
<td>3.08</td>
<td>6.8</td>
<td>3.8</td>
<td>2.21</td>
<td>5.0</td>
<td>10.3</td>
<td>6.07**</td>
<td>5.0</td>
</tr>
<tr>
<td>High school dropout</td>
<td>8.9</td>
<td>9.3</td>
<td>0.04</td>
<td>6.7</td>
<td>17.4</td>
<td>17.74****</td>
<td>6.8</td>
<td>17.6</td>
<td>16.58****</td>
<td>5.1</td>
</tr>
<tr>
<td>Failure to continue</td>
<td>48.4</td>
<td>39.8</td>
<td>5.10*</td>
<td>42.1</td>
<td>51.0</td>
<td>3.76</td>
<td>29.7</td>
<td>62.9</td>
<td>34.87****</td>
<td>38.8</td>
</tr>
<tr>
<td>education beyond high school</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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</tr>
</tbody>
</table>

Note. Values represent the percentage of offspring with the psychiatric disorder or low educational attainment. Degrees of freedom for \( \chi^2 \) = 1.


\* \( p < .05 \). \** \( p < .01 \). \*** \( p < .001 \). \**** \( p < .0001 \).
**Prediction of Low Educational Attainment in Offspring by Psychiatric Disorders in Offspring and by Covariates**

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Failure rate among youths without risk factor</th>
<th>Failure rate among youths with risk factor</th>
<th>OR</th>
<th>95% CI</th>
<th>AOR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychiatric disorders*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>61/883 (9%)</td>
<td>5/53 (9%)</td>
<td>1.06</td>
<td>0.41–2.75</td>
<td>0.21&lt;sup&gt;e&lt;/sup&gt;</td>
<td>0.05–0.97</td>
</tr>
<tr>
<td>Depressive</td>
<td>61/883 (9%)</td>
<td>6/53 (11%)</td>
<td>1.33</td>
<td>0.55–3.25</td>
<td>0.23&lt;sup&gt;e&lt;/sup&gt;</td>
<td>0.06–0.82</td>
</tr>
<tr>
<td>Disruptive</td>
<td>39/653 (6%)</td>
<td>26/83 (31%)</td>
<td>6.77</td>
<td>3.86–11.35</td>
<td>3.83&lt;sup&gt;e&lt;/sup&gt;</td>
<td>1.68–8.74</td>
</tr>
<tr>
<td>Substance</td>
<td>55/693 (8%)</td>
<td>9/43 (21%)</td>
<td>2.93</td>
<td>1.34–6.42</td>
<td>4.53&lt;sup&gt;e&lt;/sup&gt;</td>
<td>1.18–17.35</td>
</tr>
<tr>
<td>Personality</td>
<td>33/543 (6%)</td>
<td>35/193 (18%)</td>
<td>3.73</td>
<td>2.21–6.29</td>
<td>1.32&lt;sup&gt;e&lt;/sup&gt;</td>
<td>0.65–2.66</td>
</tr>
<tr>
<td>Covariates</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low parental education&lt;sup&gt;a&lt;/sup&gt;</td>
<td>21/527 (4%)</td>
<td>48/209 (33%)</td>
<td>7.72</td>
<td>4.45–13.39</td>
<td>1.42&lt;sup&gt;e&lt;/sup&gt;</td>
<td>1.19–1.69</td>
</tr>
<tr>
<td>Low parental income&lt;sup&gt;b&lt;/sup&gt;</td>
<td>39/560 (6%)</td>
<td>25/70 (36%)</td>
<td>8.18</td>
<td>4.59–14.58</td>
<td>2.13&lt;sup&gt;e&lt;/sup&gt;</td>
<td>1.39–3.25</td>
</tr>
<tr>
<td>Low parental occupation&lt;sup&gt;b&lt;/sup&gt;</td>
<td>47/666 (7%)</td>
<td>24/86 (28%)</td>
<td>5.46</td>
<td>3.10–9.59</td>
<td>0.90&lt;sup&gt;e&lt;/sup&gt;</td>
<td>0.74–1.32</td>
</tr>
<tr>
<td>Parental psychopathology&lt;sup&gt;c&lt;/sup&gt;</td>
<td>23/458 (5%)</td>
<td>44/278 (15%)</td>
<td>3.48</td>
<td>2.06–5.86</td>
<td>2.29&lt;sup&gt;e&lt;/sup&gt;</td>
<td>1.25–4.20</td>
</tr>
<tr>
<td>Low offspring IQ&lt;sup&gt;d&lt;/sup&gt;</td>
<td>42/594 (7%)</td>
<td>25/142 (18%)</td>
<td>1.06</td>
<td>1.04–1.07</td>
<td>1.04&lt;sup&gt;mn&lt;/sup&gt;</td>
<td>1.02–1.06</td>
</tr>
</tbody>
</table>

Note. OR = unadjusted odds ratio; CI = confidence interval; AOR = adjusted odds ratio.

* Diagnosed at Time 2, Time 3, or both.

*<sup>a</sup> Assessed at Time 4.

*<sup>b</sup> Controlling for comorbid psychiatric disorders, parental SES, parental psychopathology, and the child's age, sex, and IQ.

*<sup>c</sup> Controlling for single parent, parental income and occupational status, parental psychopathology and the child's age, sex, and IQ.


*<sup>e</sup> Controlling for single parent, parental education and occupational status, parental psychopathology and the child's age, sex, and IQ.

*<sup>f</sup> The highest occupational status attained by one or both parents, assessed in 1975, 1983, and 1985–1986 with a 7-point scale.

*<sup>g</sup> Controlling for single parent, parental income and education, parental psychopathology, and the child's age, sex, and IQ.

*<sup>h</sup> Considered present if either significant emotional problems or sociopathy was present in either parent in 1975, 1983, or 1985–1986; or if either parent had ever been treated for a mental disorder.

*<sup>i</sup> Controlling for single parent, parental socioeconomic status, and the child's age, sex, and IQ.

*<sup>j</sup> Mean IQ score in 1983 and 1985–1986.

*<sup>k</sup> Controlling for the child's age and sex, single parent, parental psychopathology, and parental socioeconomic status.

*<sup>l</sup> The youth's achievement, by age 20, of >12 years of education or ≤12 years of education.

### Personality disorders.

Although offspring personality disorders during adolescence predicted whether youths subsequently dropped out of high school and failed to obtain additional education after completing high school, these associations were entirely attributable to offspring IQ, comorbid psychiatric disorders, parental SES, and parental psychopathology (see Table 3). Supplemental analyses conducted with the Cluster A, B, and C personality disorders yielded similar findings. Therefore, the present findings are not consistent with the social selection hypothesis with regard to the personality disorders. Offspring personality disorders did not interact with either parental SES or psychopathology to predict offspring educational attainment.

### Disruptive disorders.

Adolescents with disruptive disorders were more than twice as likely as those without disruptive disorders to drop out of high school, and nearly four times as likely to discontinue their education after completing high school, after single-parent status, parental psychopathology, parental SES, and offspring age, gender, IQ, and comorbid psychiatric disorders were accounted for (see Table 3). Therefore, the present findings are consistent with the social selection hypothesis with regard to the disruptive disorders. Offspring disruptive disorders did not interact with parental SES or psychopathology to predict offspring educational attainment.
Substance use disorders. Adolescents with substance use disorders were more than twice as likely as those without substance use disorders to drop out of high school and more than four times as likely to discontinue their education after completing high school, after single-parent status, parental psychopathology, parental SES, and offspring age, gender, and IQ were accounted for (see Table 3). Supplemental analyses indicated, after the covariates were controlled, that alcohol abuse (AOR = 4.23; 95% CI = 1.05–16.95) and marijuana abuse (AOR = 5.95; 95% CI = 1.35–26.31) independently predicted failure to complete high school. Marijuana abuse also independently predicted failure to continue education beyond high school (AOR = 8.70; 95% CI = 1.75–43.32), but alcohol abuse did not (AOR = 2.03; 95% CI = 0.77–5.33). Considered as a whole, the present findings are consistent with the social selection hypothesis with regard to substance use disorders. Offspring substance use disorders did not interact with either parental SES or parental psychopathology to predict offspring educational attainment.

Associations of covariates with offspring educational attainment. Parental education predicted whether youths dropped out of high school and failed to continue education beyond high school, after parental psychopathology, offspring IQ, and other covariates were accounted for (see Table 3). Parental psychopathology predicted whether youths dropped out of high school and whether they failed to continue education beyond high school (see Table 2). However, after the covariates were controlled, parental psychopathology did not predict failure to continue education beyond high school (see Table 3). All of the tests of the social selection hypothesis were repeated with a version of the parental psychopathology variable that did not include parental problems with the police as a component. The results of these analyses did not differ from those reported above and in Table 3. Low offspring IQ predicted both measures of low educational attainment when no covariates were accounted for (see Table 2). However, when other covariates were controlled statistically, low offspring IQ did not predict whether youths dropped out of high school, although it did independently predict whether youths failed to continue education beyond high school (AOR = 1.04; 95% CI = 1.02–1.06; see Table 3). Exclusion of offspring IQ from the regression equations did not have an impact on the findings reported above regarding offspring educational attainment. As Table 2 indicates, boys were more likely than girls to fail to continue their education beyond high school (48% vs. 40%) but not to drop out of high school. Youths raised by single parents were more likely than those raised by two parents to drop out of high school (17% vs. 7%) but not to fail to continue education beyond high school.

Tests of Mediation Hypotheses

Logistic regression analyses were conducted to test four mediational hypotheses. The first set of analyses were conducted to investigate whether the association between low parental SES and offspring psychiatric disorders was mediated by offspring IQ. Because offspring IQ did not predict offspring anxiety, depressive, disruptive, or substance use disorders, mediational analyses were conducted for only offspring personality disorders. Results indicated that the association between parental SES and offspring personality disorders was not completely mediated by offspring IQ. All three measures of parental SES continued to predict offspring personality disorders after offspring IQ was controlled statistically.

The second set of mediational analyses was conducted to investigate whether the significant associations between low parental SES and offspring anxiety, depressive, disruptive, and personality disorders were mediated by parental psychopathology. Because neither parental education nor parental psychopathology predicted offspring substance use disorders, it was not appropriate to conduct mediational analyses involving offspring substance use disorders. Results indicated that none of the associations between measures of parental SES and offspring anxiety, depressive, disruptive, and personality disorders was completely mediated by parental psychopathology. All of the mediational analyses that were conducted indicated that low parental SES continued to predict increased risk for offspring anxiety, depressive, disruptive, and personality disorders after parental psychopathology was controlled statistically.

The third set of analyses was conducted to investigate whether the association between low parental SES and offspring educational attainment was mediated by offspring psychiatric disorders. Because neither offspring anxiety nor depressive disorders predicted offspring educational attainment and because parental SES did not predict offspring substance use disorders, it was not appropriate to conduct mediational analyses involving offspring anxiety, depressive, or substance use disorders. Results indicated that neither offspring disruptive nor personality disorders completely mediated the association between parental SES and offspring educational attainment. Low parental SES predicted both indexes of poor offspring educational attainment after disruptive and personality disorders among the offspring were controlled statistically.

Analyses conducted to test the fourth mediational model indicated that the association between parental psychopathology and offspring educational attainment was not mediated by offspring psychiatric disorders. Because neither offspring anxiety nor depressive disorders predicted offspring educational attainment and because parental psychopathology did not predict offspring substance use disorders, mediational analyses could not be conducted regarding offspring anxiety, depressive, or substance use disorders. Results indicated that neither offspring disruptive disorders nor offspring personality disorders completely mediated the association between parental psychopathology and offspring educational attainment. Parental psychopathology predicted both indexes of poor offspring educational attainment after disruptive and personality disorders were controlled statistically.

Discussion

The present findings indicate that the relative importance of social causation and social selection processes varies across diagnostic categories. These findings are consistent with previous research indicating that social causation and social selection processes both contribute to the association between SES and risk for psychiatric disorders (Dohrenwend et al., 1992; Miech et al., 1999; Pfeffer, 2000). Although the two theories originated as competing explanations for the association between SES and the prevalence of psychiatric disorders, empirical evidence increasingly indicates that the processes described by social causation and social selection theories are not mutually exclusive (Dohrenwend et al., 1992).
Particularly significant in this regard are our findings indicating that offspring disruptive disorders were associated with both low parental SES and low offspring educational attainment. These findings are consistent with previous reports indicating that disruptive disorders predicted poor academic achievement (e.g., Braggio, Pishkin, Gameros, & Brooks, 1993; Miech et al., 1999) and that constitutional and environmental variables both increase risk for onset of disruptive disorders (e.g., Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995; Loeb et al., 1995; Rutter, 1997; Slutske et al., 1997) as well as other psychiatric disorders (e.g., Eaves et al., 1997; Livesley et al., 1993; Robins & Regier, 1991).

Our findings indicate that social causation processes may play a more important role than social selection processes in the association between SES and anxiety, depressive, and personality disorders. Previous research has yielded similar findings regarding anxiety disorders (Miech et al., 1999), depressive disorders (e.g., Cole, Martin, Powers, & Truglio, 1996; Dohrenwend et al., 1992; McGue & Christensen, 1997; Miech et al., 1999), and personality disorders (e.g., Dohrenwend et al., 1992; McGuffin & Thapar, 1992; Nigg & Goldsmith, 1994; see Vernon, Jang, Harris, & McCarthy, 1997). It should be noted that other studies have suggested that social selection processes may be involved in the associations between SES and depressive (Wender et al., 1991) and personality disorders (e.g., Livesley, Jang, Jackson, & Vernon, 1993).

Consistent with social selection theory, offspring substance use disorders predicted low offspring educational attainment and were only marginally associated with low parental SES. It is of interest that previous research has indicated that social causation (Dohrenwend et al., 1992) and social selection processes (e.g., Braggio, Pishkin, Gameros, & Brooks, 1993; Kandel, Davies, Karus, & Yamaguchi, 1986; Merikangas, 1990; Newcomb, 1997) may both account for findings indicating that the prevalence of substance abuse is highest among adults in the general population with low SES (Robins & Regier, 1991). Previous research has also indicated that environmental factors, such as child abuse and neglect (e.g., Fergusson, Horwood, & Lynskey, 1996; Widom, 1989), are associated with increased risk for substance abuse.

Although parental SES did not predict offspring substance use disorders, the association between low parental income, calculated as percentage of poverty level, and offspring substance use disorders approached statistical significance (see Table 1). Furthermore, although the present findings indicating that substance use disorders predicted low educational attainment are consistent with social selection theory, any effect that low SES may have on risk for substance abuse may be mitigated by the cost of alcohol and drugs, which youths with greater financial resources are more able to afford.

**Issues Regarding Interpretation of the Present Findings**

Although low parental education and occupational status predicted offspring anxiety and depressive disorders, low parental income did not do so independently of the effects of covariates. These findings are consistent with prior research indicating that the magnitude of the association between parental SES and offspring psychiatric disorders can vary as a function of the component of parental SES that is under study (Lipman, Offord, & Boyle, 1994) and thus emphasize the importance of assessing multiple indicators of SES when investigating the social causation/social selection issue (Liberatos, Link, & Kelsey, 1988).

Recent epidemiological findings have indicated that anxiety and depressive disorders were associated with both low educational outcomes among young adults (Kessler et al., 1995). However, the Kessler et al. (1995) study was not longitudinal, and it did not control for comorbid psychiatric disorders, offspring IQ, parental psychopathology, or parental SES. Therefore, the present findings and other recent findings (Cole et al., 1996; Miech et al., 1999) suggest that if anxiety and depressive disorders are associated with poor educational attainment, this may be due to the effects of parental psychopathology, parental SES, or other factors.

It should also be noted that supplementary data analyses indicated that the overall pattern of findings reported above was obtained when analyses were restricted to diagnostic criteria that were identified during the offspring interviews and when analyses were restricted to diagnostic criteria that were identified during the maternal interviews. Furthermore, the possibility should be noted that the social causation/social selection issue may not be applicable to anxiety disorders, insofar as previous research has not established in a definitive manner that anxiety disorders are associated with low SES in the general adult population.

**Limitations**

Because this study was not a controlled experiment, its findings do not permit unequivocal inferences about causality. For example, it is possible that unmeasured childhood educational problems may have contributed to onset of psychiatric disorders during adolescence. The association of disruptive and substance use disorders with poor educational attainment may have been due in part to the negative effects of these disorders on adolescents' social relationships. Because the mean age of the sample was only 22.5 years in 1991–1993, the effects of adolescent psychiatric disorders on income and occupational status during adulthood could not be investigated, and educational attainment was the only outcome that could be used to investigate the predictions of social selection theory. In addition, it is acknowledged that unmeasured variables such as the personality trait that is described as "openness to experience" might have influenced the present findings.

Because specific parental psychiatric disorders were not assessed, global parental psychopathology was controlled statistically in the analyses reported above. It is possible that this aggregation masks differences that would emerge if specific parental diagnoses were available. It should also be noted that because the youths' fathers were not interviewed, information regarding paternal psychopathology was obtained from the maternal interviews. In addition, parental IQ was not assessed, and the effects of parental IQ on offspring educational attainment were not investigated. Instead, offspring IQ was used as a proxy in the statistical models. This substitution may result in a degree of overcontrol insofar as offspring IQ is more likely to influence other offspring characteristics than is parental IQ.

Some disorders, such as bipolar disorder and schizophrenia, were not investigated because their prevalences were too low to permit a test of the social causation and social selection theories. Although five major diagnostic categories were included in our analyses, we did not investigate the predictions of social causation...
and social selection theories regarding specific psychiatric disorders, because many of the disorders were low in prevalence and the many statistical tests would lead to capitalization on chance. It will be of interest for future research to investigate social causation and selection processes associated with a restricted range of specific psychiatric diagnoses.

Structural equation modeling, which can be useful in reducing problems of measurement unreliability, was not used to test the mediational hypotheses investigated in the present study because the dependent variables were dichotomous (Fergusson, 1997). Although offspring psychiatric disorders were diagnosed using the best instruments that were available in 1983 and 1985–1986, slightly different results might have been obtained if currently available instruments and diagnostic criteria had been used. Data were not available regarding interrater reliability, and it is possible that the interviewers’ ratings may have been affected by family SES. However, previous research has indicated that fully structured interviews administered by extensively trained and supervised interviewers tend to demonstrate acceptable levels of interrater reliability (e.g., Shaffer, Schwab-Stone, & Fisher, 1993).

Despite the limitations and concerns enumerated above, this study provides unique findings regarding the predictions of social causation and social selection theories pertaining to the association between SES and psychiatric disorders in the general population. The study design has numerous methodological strengths, including prospective longitudinal investigation of a large representative community sample over a 17-year period; assessment of offspring psychiatric disorders on the basis of interviews of the offspring and their mothers; and most important, implementation of statistical controls for the effects of parental psychopathology and offspring IQ on offspring risk for psychiatric disorders and poor educational attainment. These findings thus contribute to our understanding of the roles that social causation and social selection processes play during childhood and adolescence, accounting for the association that has been identified by researchers between socioeconomic status and the prevalence of psychiatric disorders among adolescents and adults in the community.

References


