Articles

Socioeconomic Status and Psychiatric Disorders: The Causation-Selection Issue

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Are inverse relations between psychiatric disorders and socioeconomic status due more to social causation (adversity and stress) or social selection (downward mobility of genetically predisposed)? This classical epidemiological issue is tested by focusing on ethnic status in relation to socioeconomic status. Ethnic status cannot be an effect of disorder because it is present at birth whereas socioeconomic status depends on educational and occupational attainment. A birth cohort sample of 4914 young, Israel-born adults of European and North African background was selected from the country's population register, screened, and diagnosed by psychiatrists. Results indicate that social selection may be more important for schizophrenia and that social causation may be more important for depression in women and for antisocial personality and substance use disorders in men.

IN 1855, EDWARD JARVIS, A MASSACHUSETTS EPIDEMIOLOGIST, reported the results of his classic study of the prevalence of psychiatric disorders (*1*). His most striking finding was that "the pauper class furnishes, in ratio of its numbers, sixty-four times as many cases of insanity as the independent class" (*1*, p. 52). Despite changes in concepts and methods used to define cases and measure socioeconomic status (SES), such relations have proved remarkably consistent, occurring for overall rates of functional disorders (*2–4*), and for important subtypes of schizophrenia (*2, 3, 5*), depression (at least in women) (*4, 6,* 7), and disorders involving antisocial behaviors and substance use (at least in men) (*6*).

Two very different types of explanation have been proposed. One, social causation, attributes the relation to adversity and stress associated with low social statuses (*8–11*); the other, social selection, posits that genetically predisposed persons drift down to or fail to rise out of such positions (*1*, pp. 52–56; *12*, pp. xxi–xxv; *13*, p. 31). Credibility for the social selection explanation has been provided indirectly by twin and adoption studies, which show that genetic factors contribute to disorders that are inversely related to SES (*14*). These investigations also show that genetic factors alone cannot account for the disorders; environmental factors must also play a part (*15–18*). Twin and adoption studies are not very informative about the environmental part, however; in particular, they tell us nothing about the role of SES-related environmental conditions (*13, 15–17*). Nor has the most convincing research on environmental factors—that is, investigations showing that many forms of psychopathology develop in previously normal persons exposed to prolonged military combat and other extreme situations (*2*, pp. 110–125; *18*)—clarified the role of SES.

It is likely that both social causation and social selection processes operate to create the observed SES-psychopathology associations. The goal of specifying the relative importance of these processes, however, has proved elusive. Retrospective case control studies of social mobility have been inconclusive (*2*, pp. 41–48; *19–22*). Prospective mobility and family history studies over several generations might clarify matters, but they are not practical (*2, 23*). We describe a strategy that calls for more immediately available information (*24*) and the results of its first systematic application.

Theoretical Formulation

Social causation and social selection theories both predict an inverse relation between SES and various types of psychopathology. Our problem, therefore, has been to identify circumstances in which the two theories make different predictions. This occurs when we consider ethnic status in relation to SES.

Jarvis reported not only that "paupers" had disproportionately high rates of "insanity," but also that disproportionately high numbers of the poor and insane were new Irish immigrants to Massachusetts (*1*). The Irish in Jarvis' 1850's Massachusetts are one example of an ethnic group facing harsh discrimination in the early stages of assimilation into the structures of a relatively open-class, urban society. Twentieth century examples are blacks and Hispanics in New York, Indians and Pakistanis in London, and North African Jews in Israel.

Like SES, ethnic status influences chances of obtaining jobs, goods, and services in a society. Unlike SES, which depends on educational and occupational attainments, however, an individual's ethnicity is determined at birth by immutable characteristics of skin color, national background, and religious origin. It is not affected by psychopathology or personal predispositions to psychopathology and can, therefore, be used to differentiate the two theories.

Social causation prediction. According to a powerful body of sociological and psychological theory, barriers against achieving highly valued goals (goods, services, honor) produce stresses and strains toward deviant behavior (*25*). Because ethnic prejudice and discrimination restrict mobility opportunities, dysjunctions between means and goals are likely to be experienced more frequently by members of disadvantaged than advantaged ethnic groups at every level of SES. This theory predicts, therefore, that the incremental adversity experienced by disadvantaged ethnic groups leads to

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incrementally higher rates of psychopathology at every SES level (Fig. 1).

Social selection prediction. Social selection theory assumes that the rate of psychopathology in a given SES stratum is a function of intergenerational and intragenerational sorting and sifting processes whereby the healthy and able tend to rise to or maintain high status and the unhealthy and disabled tend to drift down from high SES or fail to rise out of low SES (*12*). These selection processes are likely to affect advantaged and disadvantaged ethnic groups differently. Due to prejudice and discrimination, healthier members of disadvantaged ethnic groups would be less likely to rise out of lower SES positions than similarly healthy members of advantaged ethnic groups, thus diluting rates of disorder among lower SES members of disadvantaged ethnic groups. Moreover, only the healthiest and most able members of disadvantaged ethnic groups would be likely to rise against great obstacles to high SES. With fewer obstacles to block them, the tendency of healthier members of advantaged ethnic groups to rise would leave a relatively undiluted "residue" (*26*, p. 269) of disabled among their lower SES members. Additionally, the more advantaged the ethnic group, the more its members would be able to support unhealthy individuals at higher SES levels. The resulting prediction of higher rates in advantaged ethnic groups, SES constant, is depicted in Fig. 1.

To make the predictions shown in Fig. 1, we focus on ethnic discrimination and low educational level or occupational status, all of which operate to limit opportunity and create stress. There may be other environmental factors, such as exposure to toxic substances, for which lower SES persons but not persons of disadvantaged ethnic status (SES constant) are at greater risk. Such factors would have to be considered separately if they were shown to be related to psychopathology.

Moreover, Fig. 1 implies that there are no interactions between SES and ethnic status, but such interactions may occur. For example, affirmative action programs in United States universities may alter differential pressures faced by blacks and other minorities. This oversimplification does not undermine the test unless the trace lines cross in the center.

Figure 1 also does not distinguish between males and females. However, some disorders, such as depression, show much higher rates in females; others, such as antisocial personality and substance abuse, show much higher rates in males (*27, 28*). These disorders may be inversely related to SES only in the gender in which the disorder is more frequent; if so, the issue may be germane only or mainly within that gender.

Research Setting

To test the predictions in Fig. 1, the research setting needed two things that rarely co-occur. First, it had to be an open-class, urban society with ethnic assimilation under way. Second, it had to have a population register that records and dates births, deaths, and migration into and out of the country. Sampling from such a register makes it possible to identify persons who have died, migrated, or been institutionalized, so that something can be learned about their psychiatric status as a check on bias in rate estimations. Israel, probably uniquely, meets both requirements and was chosen for these reasons. In addition, Israel contains the valuable resource of a psychiatric register to which all hospitalizations are reported by law.

In the years since Israel became a state in 1948, immigration more than trebled its Jewish population to about 3 million by late 1982, the time we started the study. For the most part, the migrants were seeking a haven from oppression; self-selection related to psychiatric

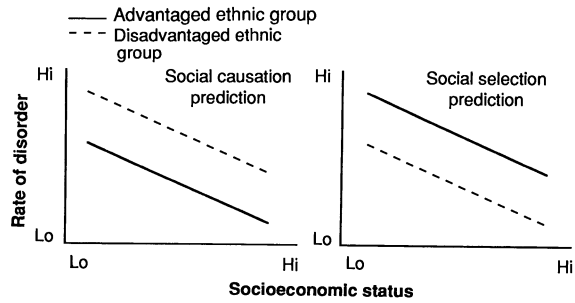

Fig. 1. Summary of social causation versus social selection predictions for any type of psychiatric disorder that is inversely related to socioeconomic status in the general population.

condition, therefore, is likely to have been minimal. "Ashkenazim," Jews of European background, made up the majority of earlier immigrants and emerged as relatively advantaged; "Oriental" Jews from North African and Middle Eastern countries, who dominated the next waves of migration, emerged as relatively disadvantaged (*29*). It has been argued that ". . . over and above the influence of socio-economic background, Oriental youth are exposed to an atmosphere of prejudice and to concrete, if unintended, discrimination" (*30*, p. 578). This describes precisely the conditions we require.

In view of its importance to our test, we have conducted our own investigation of ethnic prejudice and discrimination by Jews toward other Jews in Israel. This research indicates that attitudinal prejudice is directed by Europeans at North Africans of all SES levels (*31*). Moreover, persons of North African background are at a disadvantage at every step of the status attainment process: for example, at equivalent levels of father's occupation and education, North Africans achieve less education and lower levels of occupation than Europeans (*32*).

About 177,000 Jews of European background and of North African background were born in Israel between 1949 and 1958. By focusing on this cohort, we avoid the problem of confounding the stress of migration with the stress of assimilation that would occur if we had included foreign-born Israelis. Moreover, persons in this 24 to 33 age range (in 1982) had gone through much of the risk period for developing first episodes of the disorders with which we are concerned (*33*). As young adults, most had embarked on their own occupational careers.

However, it was necessary to investigate the possible influence of two unusual factors that could have differentially affected rates of psychopathology in the two ethnic groups. One was experience with the Holocaust by a substantial proportion of the parents of respondents of European background (*34*); the other was exposure to combat by a substantial portion of males, especially those of European background. It does not appear that either factor can account for our results.

Epidemiology

We determined that a sample of about 5000 persons should provide sufficient statistical power for our tests. The people were selected by full probability sampling procedures. To unconfound SES and ethnic status in the analyses, it was necessary to oversample higher SES members of the disadvantaged ethnic group and lower SES members of the advantaged ethnic group. This required demographic prescreening of a much larger sample of 19,000 persons from the Israel Population Register, which was accomplished with a 98% completion rate, prior to drawing the approxi-

mately 5000 member subsample on which the study focused.

We used a two-phase procedure to identify and diagnose people with disorders (*35*). The first phase consisted of a brief structured screening interview with 4914 persons to distinguish possible positives from probable negatives. The completion rate was 94.5%. The second phase consisted of intensive clinical follow-up with all those who were scored positive during screening and a subsample of 18% of the negatives, a total of 2741 of the 4914 study respondents. The completion rate was 90.7% for this phase. Informed consent was obtained from all participants after the nature and possible consequences of the study were explained.

First-phase screening. Once respondents were contacted and found demographically eligible for the study, they were interviewed by social workers, psychologists, and nurses in their homes, hospitals, prisons, or wherever they were located. They were asked anamnestic questions (for example, history of mental hospitalizations or criminal convictions) and questions about symptoms in seven screening scales from the Psychiatric Epidemiology Research Interview (PERI) (*36*). The screening scales had been tested in pilot research in Israel for reliability and criterion validity and calibrated against established psychiatric disorders in the contrasting ethnic groups (*37*).

Several tests were conducted to detect subcultural differences in reporting symptoms that might bias results (*2, 38*). First, we found no indication of important subcultural differences in the calibration study (*37*). Moreover, the scale reliabilities in the present study range from 0.91 to 0.68 overall and 0.50 or better in almost all the subgroups defined by ethnic background, gender, and educational level. We also employed alternately keyed items from the Crowne-Marlowe scale (*39*) to check on acquiescence and possible social desirability response styles, and we tested whether interviewers' ethnic background could have altered our results. None of these factors could account for symptom scale results.

More than 50% of the sample screened positive in the first phase. "Positive" was defined as one or more of the following: a score above empirically derived cut points of a discriminant function that had distinguished known individuals with disorders from controls on the symptom scales in the calibration study (*37*); a positive response on one or more anamnestic items; and inability to complete the interview.

Second-phase clinical interview. The diagnostic interview is an expanded lifetime version of the Schedule for Affective Disorders and Schizophrenia (SADS) (*40*) that we call SADS-I (for Israel). It is designed to make diagnoses according to Research Diagnostic Criteria (RDC) (*41*), a forerunner of DSM-III (*42*). The psychiatrists were intensively trained by Levav, himself trained at the New York State Psychiatric Institute where SADS was developed. A preliminary reliability check involved independent reassessments of 64 taped interviews; the kappa (0.68) between the interviewer's primary diagnosis and that of the reviewer was adequate (*43*) but could be improved. All of the tapes were, therefore, reviewed for completeness by Levav or a clinical psychologist whom he trained. About one-fifth of all respondents receiving the diagnostic interview were recontacted for additional information.

Most past epidemiological studies report SES findings for "current" prevalence, with "current" ranging from a few months to about a year before the interview. To approximate this, we have emphasized prevalence during the year before the clinical interview. However, prevalence is a function of both incidence and duration; factors contributing to occurrence may be quite different from those contributing to recurrence or duration. Because of this limitation of current prevalence, we have conducted supplementary analyses of lifetime rates despite possible problems of recall (*44*). Following conventions with the lifetime version of SADS, RDC diagnoses based on SADS-I have been made at either the "definite" or "probable" level.

We checked for possible ethnic bias in the diagnoses in four ways. First, we examined whether higher treatment rates in one ethnic group might have made it easier for psychiatrists to arrive at diagnoses in that group. Second, capitalizing on the fact that the first-phase screen was blind to ethnicity, we analyzed the ratios of probable to definite diagnoses made at each level of the screen (symptom scales plus anamnestic items; symptom scales alone; anamnestic items alone) in each ethnic group. Third, we investigated whether screening scale scores are higher for definite diagnoses in one ethnic group. Finally, we considered whether Levav's knowledge of the hypotheses could have influenced ethnic differences by examining the distribution of diagnoses he himself made. In no case did any of these checks reveal ethnic biases in the diagnoses that would have altered the results.

The starting point for our study was provided by SES findings from previous epidemiological research in other settings. Do the SES relationships occur in the Israeli cohort?

Table 1 shows the expected inverse relationships, with SES indicated by educational levels. The results are similar when occupational levels are used instead. As would also be expected on the basis of past research, base rates of major depression are higher in women and base rates of antisocial personality (which is diagnosed only as lifetime) and substance use disorders (including alcoholism) are higher in men. We will return below to the base rates according to ethnic background.

Test of the Issue

Statistical procedures. Because SES and ethnic status are confounded in the population, we conducted our tests in a sample that was specially constructed to overrepresent ethnically disadvantaged persons who are high in SES and ethnically advantaged persons who are low in SES. Although the balance remains far from complete, oversampling of the scarcely populated SES groupings permits statistical control of SES.

To make this control comprehensive, we have included both continuous and categorical measures of each respondent's formal education and occupational status or, for married women, occupation of the respondent's spouse. The two measures of education are, first, level completed in the categories of low (not high school graduate), middle (high school graduate but not college graduate), and high (college graduate or more) and, second, number of years of formal education completed. The categorical occupation indicator consists of employed versus currently unemployed; if employed, the Kraus (*45*) occupational prestige groupings of low (scores of 1 to 30), middle (31 to 66), and high (67 to 100) were used. We also investigated a continuous measure that ranged from 0 (currently unemployed) to 100 (highest occupational prestige possible on the Kraus scale); however, this measure showed no statistically significant (P = 0.05 or better) relationship, independently of the other three indicators of SES, to any diagnostic rate and was, therefore, dropped as redundant. We added dummy variables to represent military personnel (n = 310), full-time students (n = 73), and persons who said they were employed but whose job description was missing (n = 55). Diagnostic rates are regressed on these indicators of SES, ethnic background (birth of father in Europe versus North Africa), date of birth, birth cohort used for sampling purposes (1949–52, 1953–56, 1957–58), and gender.

Because only a minority of first-phase negatives were sampled for inclusion in the second phase (SADS-I), it was necessary in the analyses to weight the diagnostic results to represent the entire

first-phase sample. Diagnostic interviews were weighted by the inverse of the SADS-I sampling rates in each stratum. Regression analyses were done by computer software (*46*) that implements the methods of Fuller (*47*).

To gain confidence in the robustness of our results to model specifications, both logistic regression and weighted least squares (WLS) multiple regression analyses were conducted. Except when the results are affected by the form of the regression model, we report the findings in terms of the logistic models. Separate analyses were done for males and females for disorders that typically have different rates in the two genders. For each set of analyses, we first tested main effects only and then included interactions between ethnic status and SES. When statistically significant interactions ($P = 0.05$ or better) were found, it is these rather than main effects that are interpreted.

Table 2 shows ethnic differences for all four types of disorder, diagnosed at the current definite level, according to one indicator of SES, the categorical measure of educational level. The regression analyses are reported for lifetime as well as current rates at the combined probable and definite levels, as well as definite only, with controls on the multiple indicators of SES described above.

Schizophrenia. Table 2 suggests that the rates of current, RDC definite schizophrenia are higher among respondents of European background. According to the regression analyses, this ethnic difference is statistically significant after comprehensive controls on SES and the other relevant variables (logistic regression results: $b = 1.34$; SE = 0.55; $z = 2.42$; $P < .05$; adjusted odds ratio 3.82). Additional analyses with current definite plus probable and with lifetime rates of both types give similar, slightly stronger results (logistic $b = 1.46$; SE = 0.52; $z = 2.80$; $P < .01$; adjusted odds ratio 4.30 for lifetime probable and definite combined).

Tests for interactions of ethnicity and the various indicators of SES were not statistically significant. However, the regression analyses show that there is an unexpected gender difference, with rates tending to be higher for males ($P < .05$ for current definite). This may be due to the youthfulness of the sample and gender differences in age of onset; women tend to develop schizophrenia an average of 4 to 6 years later than men (*48, 49*).

Levav *et al.* (*53*) also found higher rates of schizophrenia among Europeans in a study of first hospitalizations in Jerusalem. Because the large majority of people with schizophrenia in our sample were treated in psychiatric hospitals for their first episode, treatment rates are probably not as biased by treatment selection factors as in the United States (*6*). Taken together with Levav *et al.*'s earlier findings, therefore, our results provide strong support for the social selection prediction.

It is possible that the causal importance of environmental factors has been underestimated by our exclusive focus on the social environment. If compelling demonstrations of physical environmental risks [for example, occupationally related noxious conditions (*22*) or viruses (*50*)] emerge for schizophrenia, these would have to be controlled in the analysis. Meanwhile, the plausibility of our results is increased by evidence from two additional sources: First, findings from twin, adoption, and family studies indicating that genetic factors play an important role in schizophrenia (*15, 17*); second, results from case control studies showing that adversity from stressful life events is less important in schizophrenia than in depression (*51, 52*). The relevance of this comparison will become evident as we turn to major depression.

Major depression. By contrast with schizophrenia, the results for major depression are more consistent with the social causation prediction. For all four types of rate, North African women are higher ($P < 0.01$), with the ethnic contrast strongest for diagnoses at the current, definite level (logistic $b = 1.17$; SE = 30; $z = 3.85$; $P < 0.001$; adjusted odds ratio 3.22). The main effects of ethnic status are not statistically significant ($P = 0.05$ or better) for males, and there are no significant ethnicity by SES interactions for males or females.

Depression is not inversely related to educational level among North Africans, especially women (Table 2). In fact, when current probable and definite diagnoses are combined, we find an inverse relation for males but a direct relation between depression and educational level for North African females. The reasons may be related to marital status and patterns of intermarriage. Only 49% of highly educated North African women have ever been married, and 70% of these to Europeans. This is in contrast to North African women of lower educational levels, the large majority of whom (81%) are married and, as a rule, to North Africans. Marginal status

Table 1. Base rates by demographic characteristics of educational level, gender, and ethnic background. "Current" (1 year before interview) and lifetime prevalence rates per hundred for disorders diagnosed at RDC definite (D) and at RDC definite plus probable (D&P) levels of confidence are weighted to reflect the population from which the sample was drawn. Sample $n = 4914$ for first-phase screening interview; sample $n = 2741$ for second-phase diagnostic interview.

Demographic characteristics	Schizophrenia		Major depression		Antisocial personality (lifetime only)*		Substance use disorders	
	D	D&P	D	D&P	D	D&P	D	D&P
			Current					
Not high school graduate	1.36	1.54	4.43	7.01			4.01	5.60
High school graduate	0.52	0.52	4.24	5.63			0.35	0.96
College graduate	0.34	0.34	2.58	3.47			0.19	0.19
Female	0.35	0.42	4.90	6.39			0.33	0.56
Male	1.02	1.04	3.10	4.78			2.07	3.30
North African	0.57	0.60	6.58	9.12			2.85	4.31
European	0.75	0.80	2.65	3.80			0.36	0.72
			Lifetime					
Not high school graduate	1.36	1.54	17.52	27.50	2.40	5.54	6.50	9.95
High school graduate	0.52	0.52	14.76	21.84	0.14	0.79	1.55	4.13
College graduate	0.84	0.84	11.91	15.69	0.00	0.00	0.40	1.60
Female	0.53	0.60	20.02	29.08	0.17	0.47	1.00	2.38
Male	1.02	1.04	9.92	15.28	1.13	3.09	4.04	7.73
North African	0.57	0.60	19.80	29.12	1.75	4.05	4.99	8.71
European	0.89	0.94	12.39	18.48	0.09	0.63	1.27	3.21

*Antisocial personality is by definition the same for lifetime as current prevalence.

in the assimilation process may involve an increment of stress for highly educated women of North African background.

Antisocial personality. Table 2 shows that there were no cases of antisocial personality among college graduates. For this reason, college graduates from both ethnic groups were excluded from the regression analyses. The results indicate no statistically significant ethnic differences in rates for females. For males, there are significantly higher rates of RDC definite antisocial personality among North Africans (logistic $b = 1.63$; SE = 0.83; $z = 1.96$; $P < 0.05$; adjusted odds ratio 5.09 for definite; and $b = 1.26$; SE = 0.43; $z = 2.92$; $P < 0.01$; adjusted odds ratio 3.51 for probable and definite combined). There were no statistically significant interactions of SES indicators and ethnic status. As with major depression, these results are consistent with the social causation prediction, but for males rather than females.

Substance use disorders. Substance use disorders have similarities with antisocial personality: They are more frequent among males; there are no statistically significant ethnic differences in their rates among females; and these disorders are all but absent among college graduates. With college graduates of both ethnic groups again omitted, regression analyses show significantly higher rates of substance-use disorders diagnosed at the current, definite level among males of North African background (logistic $b = 1.01$; SE = 0.47; $z = 2.17$; $P < 0.05$; adjusted odds ratio 2.76). There are significant main effects as well for definite lifetime diagnoses (logistic $b = 0.57$; SE = 0.29; $z = 1.96$; $P = 0.05$; adjusted odds ratio 1.77). For neither of these definite level diagnoses is there a significant ethnicity by SES interaction. However, when probable cases are pooled with the definite cases for both current and lifetime diagnoses, there are significant interactions of ethnic status and years of education, a quantitative indicator of SES ($P < 0.01$ for current; $P < 0.05$ for lifetime). These latter results imply, as the descriptive findings in Table 2 also suggest, that the social causation effects of disadvantaged ethnic status are strongest among males of lowest SES.

Since there is considerable comorbidity among alcoholism, other drug use disorders, and antisocial personality, we checked whether persons with only one of these disorders were distributed similarly to those with various combinations of the disorders. The highest rates of each type were consistently found among low SES males of North African background.

Comparisons between logistic and WLS multiple regression analyses. The use of statistical adjustments in nonexperimental research to equate groups is problematic, but necessary in fields such as psychiatric epidemiology. The confidence we have in the adjustments depends in part on the confidence we have in the specification of the statistical model. To determine whether our results are robust with respect to the choice of an analytic model, we used both logistic regression and WLS multiple regression methods. The former attempts to model the logarithm of the odds of disorder and the latter the simple risk of disorder.

The results for schizophrenia and major depression are entirely consistent for the two analyses. For substance use disorders and antisocial personality in men, the results are largely consistent, with the analysis of risk differences (WLS) suggesting that SES by ethnic interaction is more important. For substance use disorders, the logistic analysis revealed that the ethnic difference is strongest among less educated men for two of the four diagnostic outcomes, whereas the multiple regression of risks needed this interaction for all four outcomes (for example, multiple regression interaction results for current definite: $b = 0.012$; SE = 0.004; $z = 2.88$; $P < 0.01$). For antisocial personality, the interactions were not statistically significant in the logistic regression, but were significant in the risk regression (for example, multiple regression for definite: $b = 0.011$; SE = 0.004; $z = 3.02$; $P < 0.01$). Regardless of the form of the analysis chosen, results for schizophrenia were consistent with the social selection prediction, and results for major depression, substance use disorders, and antisocial personality with the social causation prediction, at least in the lowest levels of SES.

Table 2. Diagnostic results of second-phase clinical interviews. "Current" prevalence (1 year before interview) rates per hundred of RDC definite diagnoses by education, gender, and ethnic background.

Education	European (n = 1197)		North African (n = 1544)	
	Male (602)	Female (595)	Male (852)	Female (692)
Schizophrenia				
Not high school graduate	4.18	0.84	1.91	0.00
High school graduate	0.29	0.39	0.17	0.00
College graduate	0.00	0.39	0.00	0.00
Major depression				
Not high school graduate	3.01	4.81	3.47	6.60
High school graduate	2.06	2.93	4.61	12.11
College graduate	0.66	1.68	2.94	10.70
Antisocial personality (lifetime)				
Not high school graduate	0.73	0.82	6.97	0.73
High school graduate	0.15	0.00	0.79	0.30
College graduate	0.00	0.00	0.00	0.00
Substance use disorders				
Not high school graduate	2.28	0.84	8.29	0.72
High school graduate	0.44	0.13	0.75	0.28
College graduate	0.49	0.00	0.00	0.00

Comparisons of diagnostic and symptom scale results. The seven psychometric symptom measures used in the first-phase screening consisted of: demoralization, enervation, and suicidal ideation and behavior, each of which contains symptoms more characteristic of depression than of the other disorders; false beliefs and perceptions, and schizoid tendencies, both of which are more likely to be related to psychoses; antisocial history; and drinking problems. Tests of ethnic differences on these scales were conducted that parallel those done with the diagnostic data, with the same comprehensive controls on SES. Because the scales are quantitative, multiple regression was the method of analysis.

By and large, the scale results are consistent with the diagnostic findings. North African women have significantly higher scores on the demoralization and enervation scales than European women ($P < 0.001$), with no significant ethnicity by SES interactions. Among males, there are statistically significant ethnicity by years of education interactions, indicating that scores on the scales of antisocial history ($P < 0.001$) and drinking problems ($P < 0.01$) are higher among North African than European males, with the greatest differences occurring in persons of low SES.

The most striking exception occurs on the scale of false beliefs and perceptions. To be consistent with the diagnostic findings, Europeans should score higher on this scale. However, analyses reveal a significant interaction of ethnicity and years of education ($P < 0.05$), as North Africans with fewest years of education tend to score highest. It is possible that there are ethnic biases in responses to this scale (*37*, p. 318). However, because this particular bias in a first-phase screening scale would lead, if anything, to overestimation of schizophrenia in respondents of North African background, eliminating the bias would not alter our finding of higher rates of schizophrenia in Europeans.

Diagnostic base rates according to ethnic status. The comparisons of population base rates for Europeans and North Africans in Table 1 provide useful additional information. Note first that there is no statistically significant ethnic difference in the base rates of schizo-

phrenia (for example, $z = 0.28$; $P > 0.50$ for current definite). This indicates that the significantly higher European rates with SES controlled are not due to a stronger genetic predisposition among Jews of European background in general. Rather, it appears that differential pressure associated with ethnic prejudice and discrimination affects mainly how persons who have developed or are predisposed to schizophrenia are sorted and sifted into different SES levels within each ethnic group, as the social selection hypothesis predicts.

As Table 1 also suggests and statistical tests support, the current and lifetime base rates of major depression, substance use disorder, and antisocial personality are significantly ($P < 0.001$) higher among persons of North African background. This is what would be expected if social causation processes were stronger than social selection processes for these disorders, with stress from both ethnic prejudice and low SES contributing to their occurrence.

The population and psychiatric registers provide means of evaluating possible biases in rates due to selective mortality (an estimated 2.4% of the cohort sample), migration (an estimated 6.4%), or refusal to participate by nonmigrants (5.5%). It has also been possible to obtain data on causes of death for a subsample of the dead. These checks indicate that the ethnic differences in rates of schizophrenia, antisocial personality, and substance use disorders are, if anything, underestimated from the findings on respondents living in Israel. For example, in the sample of eligible respondents who refused to be interviewed, persons of European background had higher rates of hospital diagnoses of schizophrenia (2.9% versus 1.1%). Moreover, deaths of males of European background were much more likely to have occurred in military combat; by contrast, deaths of males of North African background were far more likely to be associated with drugs, alcohols, or non-war-related violence.

Conclusions

There has long been controversy about social causation and social selection explanations of inverse relations between SES and psychiatric disorders. While proponents of both theories have presented evidence and arguments to support their positions, no one has demonstrated previously that one position is more compelling. The present study offers a resolution, with findings strongly suggesting that the hypothesized processes differ in relative importance by diagnostic type.

We have found that rates of schizophrenia are higher for respondents of European background with SES controlled, as would be expected if sorting and sifting processes function to hold down more healthy persons of disadvantaged North African background while leaving behind a residue of severely ill persons of advantaged European background. This outcome implies that social selection processes are more important than social causation processes in the relation between SES and schizophrenia.

By contrast, holding SES constant, we found that rates of major depression in women and antisocial personality and substance use disorders in men are higher in Israelis of North African background, as would be expected if an increment in adversity attaching to disadvantaged ethnic status produces an increment in psychopathology. These results suggest that social causation processes are stronger than social selection processes in inverse relations between SES and these disorders.

These outcomes are more complicated than the diagram of the social causation and social selection predictions shown in Fig. 1. Most important, the ethnic difference in depression may be limited to females. This not wholly unexpected gender difference in depression, considered along with the opposite gender differences in antisocial personality and substance use disorders, is consistent with the possibility that there are gender-specific modes of reacting to adversity that affect the types of disorder that develop (*27*).

In addition to different sets of advantaged and disadvantaged ethnic groups, it may also be possible to extend the tests to incompletely understood physical diseases that are inversely related to SES (*54*). Meanwhile, our study raises new questions, especially about the role of gender, and sets the stage for intensive investigation of the specific stress and selection processes involved in relations among ethnic background, SES, and different types of psychiatric disorders.

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Ozone Depletion: Ultraviolet Radiation and Phytoplankton Biology in Antarctic Waters

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The springtime stratospheric ozone (O_3) layer over the Antarctic is thinning by as much as 50 percent, resulting in increased midultraviolet (UVB) radiation reaching the surface of the Southern Ocean. There is concern that phytoplankton communities confined to near-surface waters of the marginal ice zone will be harmed by increased UVB irradiance penetrating the ocean surface, thereby altering the dynamics of Antarctic marine ecosystems. Results from a 6-week cruise (Icecolors) in the marginal ice zone of the Bellingshausen Sea in austral spring of 1990 indicated that as the O_3 layer thinned: (i) sea surface– and depth-dependent ratios of UVB irradiance (280 to 320 nanometers) to total irradiance (280 to 700 nanometers) increased and (ii) UVB inhibition of photosynthesis increased. These and other Icecolors findings suggest that O_3-dependent shifts of in-water spectral irradiances alter the balance of spectrally dependent phytoplankton processes, including photoinhibition, photoreactivation, photoprotection, and photosynthesis. A minimum 6 to 12 percent reduction in primary production associated with O_3 depletion was estimated for the duration of the cruise.

HUMAN ACTIVITIES, INCLUDING THE PRODUCTION OF chlorofluorocarbons, have reduced the concentration of stratospheric O_3 (*1–3*). The most dramatic loss of O_3 has been observed over Antarctica during the austral spring, where a 50% reduction in O_3—the widely discussed ozone hole—has been documented (*4–7*). Further reduction in average global stratospheric O_3 at all latitudes is predicted over the next century despite international efforts to address the problem (*8*). Reduced stratospheric O_3 results in increased flux of biologically damaging midultraviolet radiation (UVB, 280 to 320 nm) (*9–12*) to the surface of the earth and to ecologically significant depths in the ocean (*13–17*). Consequently, it has been hypothesized (*16, 18–24*) that marine life in the upper portion of the photic zone may be adversely affected by O_3-dependent increases in UVB radiation. Experimental evidence shows that exposure to UVB radiation decreases algal productivity (*19, 25–28*) and causes damage to various forms of aquatic larvae and other organisms (*29–37*). Furthermore, evidence suggests that environmentally relevant levels of UVB radiation now incident at the surface of the ocean should negatively impact various natural populations of marine organisms (*22, 33*). In spite of the mounting evidence, the extrapolation of these findings to allow prediction of impacts on the dynamics and vitality of natural, mixed communities of marine organisms is uncertain (*10, 11, 38–42*), and estimated impacts to the Antarctic marine ecosystem have ranged from insignificant to catastrophic (*43–45*).

Phytoplankton blooms in the Antarctic marginal ice zone (MIZ) are believed to contribute significantly to the overall productivity of the Southern Ocean throughout the austral spring and summer

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