One of the most challenging findings in psychiatric epidemiology is that the highest overall prevalence rates of psychiatric disorders are consistently found in persons of the lowest socioeconomic status (SES) (Dohrenwend & Dohrenwend, 1974; Holzer et al., 1985). Our best evidence is that this relationship holds for a number of important subtypes of psychopathology: schizophrenia, antisocial personality, alcoholism and drug abuse (Dohrenwend & Dohrenwend, 1974), major depression (at least in women) (Dohrenwend, 1990a) and nonspecific distress or «demoralization» (Link & Dohrenwend, 1980a).

These findings have raised and re-raised over the years the classic social causation-social selection issue. The social causation explanation, proposed by environmentally oriented theorists, holds that rates of some types of psychiatric disorder are higher in lower SES groups because of greater environmental adversity (e.g., Faris & Dunham, 1939; Hollingshead & Redlich, 1958; Leighton et al., 1963; Srole et al., 1962). The selection explanation, proposed by genetically oriented theorists, argues that rates of one or another type of disorder are higher in lower SES groups because persons with the disorders or other personal characteristics predisposing to the disorders drift down into or fail to rise out lower SES group (e.g., Jarvis, 1855; Dunham, 1965; Wender et al., 1973). It is likely, even for schizophrenia where the selection evidence is strongest, that both processes are operating (e.g., Dohrenwend & Dohrenwend, 1981; Kohn, 1972; Link et al., 1986). What have been missing are decisive indications of their relative importance. One reason for this unsatisfactory state of affairs is that there are major practical and/or ethical obstacles to direct approaches to the problem such as multigeneration prospective studies or human experiments (Dohrenwend & Dohrenwend, 1969, pages 39-48; Dohrenwend, 1975; Link et al., 1986). Over the years, therefore, my colleagues and I have been working on a less direct, quasi-experimental strategy for resolving the issue.

Where relations between SES and various types of psychopathology are concerned, social causation and social selection hypotheses both make the same prediction about an inverse relationship. Our problem, therefore, has been to find a set of circumstances in which the two contrasting theoretical orientations lead to different predictions. We have argued that the assimilation of ethnic groups into the SES structures of relatively open urban societies provides such an opportunity (Dohrenwend, 1966; Dohrenwend & Dohrenwend, 1969; Dohrenwend, 1975; Dohrenwend & Dohrenwend, 1981). The opportunity arises out of the contrast between ethnic status and SES. Unlike SES which depends on educational and occupational achievement, an individual’s ethnicity cannot even in small part be a function of his or her prior psychopathology or personal predispositions to psychopathology. It depends on immutable characteristics such as skin color, national or regional background, and religious origin that are determined at birth.

Consider first the social causation prediction under circumstances of ethnic assimilation. It is reasonable to assume that greater adversity, stemming from prejudice and discrimination, produce an increment in stress for members of disadvantaged ethnic groups, over and above that stemming from SES. On the basis of this assumption, the social causation theorist would predict higher rates of psychopathology for members of disadvantaged ethnic groups — such as
blacks or Latinos in New York City, Indians or Pakistanis in London, Jews of North African background in Israel, and Italians of southern origin in the northern city of Milan — than for members of advantaged ethnic groups at the same SES levels.

The social-selection theorist, by contrast, would predict just the opposite. Such a theorist would expect the rate of psychopathology in a given SES grouping to be a function of sorting and sifting processes whereby the healthy and able tend to rise to or maintain high status and the unhealthy and disabled to drift down from high status or fail to rise out of low status. Since the adversity from prejudice and discrimination is greater for members of disadvantaged ethnic groups, the social-selection theorist would expect that more of their healthier members would be kept down in lower SES positions. This would dilute the rate of disorder among lower SES members of disadvantaged ethnic groups, with only the very healthiest and most able members rising against great obstacles to higher SES positions. With less pressure to block them, the tendency of healthier members of more advantaged ethnic groups to rise would leave a residue of disabled among lower SES members. Moreover, the more advantaged the ethnic group, the more unhealthy individuals it would support at higher SES levels. The result would be higher rates in members of advantaged ethnic groups than in their SES counterparts in disadvantaged ethnic groups.

These alternative hypotheses suggest that the social causation-social selection issue could turn on what deceptively appears to be a simple question of fact: Are the rates of the various types of psychiatric disorder that are inversely relate to SES higher or lower in members of disadvantaged ethnic groups than in members of advantaged ethnic groups with SES held constant?

Although we have presented preliminary data extracted from our own and others' previous research on contrasting ethnic groups (Dohrenwend & Dohrenwend, 1969; Dohrenwend, 1975; Dohrenwend & Dorenwend 1981), the results have tended to be scarce, inconsistent, and inconclusive. There are a number of reasons for this.

First, the number of appropriate SES controlled comparisons of advantaged and disadvantaged ethnic groups are scarce because SES and ethnic status are confounded; disadvantaged ethnic groups are composed mainly of lower SES individuals and advantaged ethnic groups mainly of middle and upper SES persons. Epidemiological studies have tended to rely on sampling procedures that represent ethnic status and SES in their confounded state, impairing our ability to compare advantaged and disadvantaged ethnic group members who are at the same SES levels.

The second major problem has been the difficulty in securing unbiased estimates of rates of the types of disorders that are inversely related to SES. Hospital admissions and other treatment statistics are inadequate because only minorities of those with most psychiatric disorders have ever come into treatment with members of the mental health professions and, more important for our purposes, treated and untreated cases are often distributed differently in the population (e.g., Link & Dohrenwend, 1980b). We must be able, therefore, to count untreated as well as treated cases of even quite rare disorders such as schizophrenia in very large samples to obtain representative rates. Moreover, there are other problems in securing unbiased estimates. Persons who develop schizophrenia, antisocial personality, substance use disorders (including alcoholism), and some neurotic types of disorder as well, tend to die young (e.g., Martin et al., 1985; Kendler, 1986), be hospitalized, imprisoned, and/or move residence frequently. Such persons are likely to be under-represented in the cross-sectional samples of household members who are selected for study in most epidemiological research on psychiatric disorders in the general population.

These problems exist against a background of controversy about how to conceptualize and measure psychiatric disorders for purposes of epidemiological research (Dohrenwend, 1990b). Prior to 1980, there was little consensus in psychiatry about the criteria for diagnosing particular types of psychiatric disorders; even now, with the widespread adoption of the Diagnostic and Statistical Manual of the American Psychiatric association (DSM-III) (APA, 1980) for research and treatment, consensus is far from complete even in the United States (e.g., Mirowsky & Ross, 1989; Klerman, 1989). Analysis of these problems coupled with new advances in psychiatric screening scales and research diagnostic examinations have led us to advocate a two-phase procedure involving carefully calibrated screening scales at the first phase and follow up clinical examinations by psychiatrists at the second (Dohrenwend & Dohrenwend, 1982; Dohrenwend, 1990b). This approach brings two different methods, each with different strengths and weaknesses, to bear on the problem of case identification and classification.

My colleagues and I recently completed and reported an investigation that was designed to deal better...
then we had been able to do before with these methodological problems (Dohrenwend et al., 1992). It constitutes the first full-scale test of our strategy to resolve the social causation-social selection issue. Let me summarize briefly what we did and what we found. The reader who would like more information can find it in the publication cited above (Dohrenwend et al., 1992).

Our first step was to choose Israel as the research setting. There were two main factors leading to this decision. One was that Israel, like many other modern urban societies, contains advantaged and disadvantaged ethnic groups in an assimilation situation; the other, which makes Israel almost unique among such multiethnic settings, is that the state also maintains a population register in which births, deaths and migration into and out of the country are recorded. When sampling from such a register, it is 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.

We used this population register to select a full probability sample of close to 5,000 young, Israel-born adults from two different ethnic groups: one, an advantaged ethnic group composed of Jews of European background; the other, a disadvantaged ethnic group composed of Jews of North African background. The sample was stratified to balance, insofar as possible, SES in the two ethnic groups. In face-to-face interviews, sample members were then screened with psychometric symptom scales (Shrout et al. 1986) and diagnosed by psychiatrists according to Research Diagnostic Criteria (RDC) (Spitzer et al., 1978), a forerunner of DSM-III.

The results of our analyses strongly suggest that the hypothesized processes differ in relative importance by diagnostic type. While there was a social selection outcome for schizophrenia, there were strong social causation outcomes for major depression in women and for antisocial personality and substance use disorders, including alcoholism, in men. It would be valuable to replicate the study with different sets of advantaged and disadvantaged ethnic groups in different assimilation settings. This would make it possible to rule out idiosyncratic cultural-historical and/or genetic factors that could affect the results in a particular setting. Meanwhile, the present study goes a long way toward resolving the issue. Moreover, the results raise new questions, especially about the role of gender, and set the stage for intensive investigation of the specific stress and selection processes involved in relations among ethnic status, SES, and different types of psychiatric disorders.

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