Published in final edited form as:
Psychol Med. 2005 March ; 35(3): 317–327.

Lifetime risk and persistence of psychiatric disorders across ethnic groups in the United States

JOSHUA BRESLAU[1,*], **KENNETH S. KENDLER**[2], **MAXWELL SU**[1], **SERGIO GAXIOLA-AGUILAR**[3], and **RONALD C. KESSLER**[1]

[1] Department of Health Care Policy, Harvard Medical School, Boston, MA, USA

[2] Departments of Psychiatry and Human Genetics, Medical College of Virginia/Virginia Commonwealth University, VA, USA

[3] Department of Psychology, California State University, Fresno, CA, USA

Abstract

Background—Recent research in the United States has demonstrated striking health disparities across ethnic groups. Despite a longstanding interest in ethnic disadvantage in psychiatric epidemiology, patterns of psychiatric morbidity across ethnic groups have never been examined in a nationally representative sample.

Method—Ethnic differences in psychiatric morbidity are analyzed using data from the National Comorbidity Survey (NCS). The three largest ethnic groups in the United States – Hispanics, Non-Hispanic Blacks and Non-Hispanic Whites – were compared with respect to lifetime risk and persistence of three categories of psychiatric disorder: mood disorder, anxiety disorder, and substance use disorder.

Results—Where differences across ethnic groups were found in lifetime risk, socially disadvantaged groups had lower risk. Relative to Non-Hispanic Whites, Hispanics had lower lifetime risk of substance use disorder and Non-Hispanic Blacks had lower lifetime risk of mood, anxiety and substance use disorders. Where differences were found in persistence of disorders, disadvantaged groups had higher risk. Hispanics with mood disorders were more likely to be persistently ill as were Non-Hispanic Blacks with respect to both mood disorders and anxiety disorders. Closer examination found these differences to be generally consistent across population subgroups.

Conclusions—Members of disadvantaged ethnic groups in the United States do not have an increased risk for psychiatric disorders. Members of these groups, however, do tend to have more persistent disorders. Future research should focus on explanations for these findings, including the possibility that these comparisons are biased, and on potential means of reducing the disparity in persistence of disorders across ethnic groups.

INTRODUCTION

Efforts to address race-ethnic disparities in health have now become central to national health-care policy in the United States (US Department of Health and Human Services, 2000; Smedley *et al*. 2003). Epidemiological research has advanced this agenda by identifying the specific

*Address for correspondence: Dr Joshua Breslau, Department of Health Care Policy, Harvard Medical School, 180 Longwood Avenue, Boston MA 02115, USA. (Email: breslau@hcp.med.harvard.edu).

DECLARATION OF INTEREST
None.

disorders, aspects of illness course, and subgroups of the population [e.g. as defined by gender, birth cohort, or socio-economic status (SES)] where race-ethnic differences are greatest (Sacco *et al*. 1998; Ng-Mak *et al*. 1999; Wong *et al*. 2002). These analyses have helped identify potential causal pathways that might help explain race-ethnic disparities in health and have focussed health policy on the areas where disparities are greatest (LaViest, 1996; Williams, 1997).

Race-ethnic disparities in mental illness have not yet been examined in as much detail as differences in physical illness. This is surprising in light of the long history of interest among mental health researchers in social inequalities associated with race-ethnic status (US Department of Health and Human Services, 2001). Race-ethnic differences in mental hospital admissions were the subject of considerable research before World War II (Malzberg & Lee, 1956; Parker & Kleiner, 1966). This work showed that upwardly mobile Blacks had especially high rates of mental hospitalization, a finding that was interpreted as due to the stresses of 'goal striving'. Numerous community epidemiological surveys carried out beginning shortly after World War II studied race-ethnic differences in non-specific psychological distress and expanded the investigation from only Blacks to consider Hispanics as well. The majority of these studies found significantly higher levels of distress among Hispanics or Non-Hispanic Blacks than Non-Hispanic Whites (Vega & Rumbaut, 1991). However, controlling for indicators of SES, mostly income and education, these differences were greatly reduced or eliminated. This finding led some researchers to argue that race-ethnic differences in distress are due to the disadvantaged SES of minorities (Warheit *et al*. 1975). Other researchers continued to feel that there was more to race-ethnic mental health disparities than simply low SES, however, arguing that discrimination creates additional stresses among minorities that are independent of SES (Jackson *et al*. 1996). This issue has also arisen in recent research in the UK on ethnic differences in psychiatric morbidity (Bhugra & Bahl, 1999; Nazroo, 2001). While one study found that an apparent higher prevalence of psychiatric disorders was completely explained by social class differences (Jenkins *et al*. 1997), another study found experiences of racism to be related to depression even when social class variables were controlled (Karlsen & Nazroo, 2002).

Barbara and Bruce Dohrenwend used the idea that minority discrimination might cause mental illness to develop an innovative epidemiological research design in the 1970s to distinguish the effects of SES on mental illness, which they referred to as 'social causation', from the effects of mental illness on SES, which they referred to as 'social selection'. The reasoning was that low SES minorities should have a lesser concentration than low SES Whites of people with pre-existing mental disorders who 'drifted' into the low SES segment of society, the reason being that many very competent minorities are forced to remain in low SES positions because of discrimination. This being the case, we would expect to find that low SES is associated with a higher prevalence of mental illness among minorities than non-minorities if the effects of discrimination on mental illness are greater than the effects of social selection, while we would expect the opposite if the effects of social selection were greater than the effects of discrimination. In a series of community epidemiological surveys that examined this issue first in comparisons between Blacks and Whites in the United States (Dohrenwend, 1966) and subsequently in comparisons between Europeans and North Africans in Israel (Dohrenwend *et al*. 1992), the Dohrenwends and their colleagues found that low SES is more strongly related to anxiety-mood disorders among minorities than non-minorities (consistent with the argument that the effects of discrimination are greater than the effects of social selection), while low SES is less strongly related to psychosis among minorities than non-minorities (consistent with the argument that the effects of social selection are stronger than the effects of discrimination).

From a purely statistical perspective, these findings show that statistical interactions exist between race-ethnicity and SES in predicting mental illness; i.e. that the joint effect of being

a minority and having low SES is greater than the sum of its parts. This, in turn, means that earlier epidemiological studies, which tried to explain the associations between minority status and psychological distress by controlling statistically for the confounding effects of SES, were flawed because these control analyses were based on the incorrect assumption that the effects of SES are the same for minorities and non-minorities. This error was subsequently documented definitively by Kessler & Neighbors (1986) in a secondary analysis of eight separate community epidemiological surveys that examined the joint effects of minority status and low SES in predicting non-specific psychological distress (Kessler & Neighbors, 1986). A statistically significant interaction was consistently documented in these surveys, with the association between minority status and elevated psychological distress consistently more powerful in the low-SES than the higher-SES segments of the samples. In fact, the association between minority status and elevated distress disappeared entirely in the higher-SES segments of some samples.

Beginning in the early 1980s, psychiatric epidemiology moved away from studies of nonspecific psychological distress and began to focus on surveys that administered fully structured research diagnostic interviews that generated diagnoses based on the Diagnostic and Statistical Manual of Mental Disorders (DSM). The first large-scale study of this type was the Epidemiological Catchment Area (ECA) study, conducted in five metropolitan areas in the United States in the early 1980s (Robins & Regier, 1991). The National Comorbidity Survey (NCS), the first nationally representative survey to use a diagnostic interview, was conducted in the early 1990s (Kessler *et al.* 1994). Data from these studies make it possible to extend the analysis of race-ethnic differences in psychiatric morbidity in two important ways. First, while the earlier studies examined nonspecific distress, diagnostic interviews assess a range of distinct clinical syndromes ranging from depression and anxiety disorders to psychoactive substance use disorders. This allows us to focus race-ethnic comparisons on problems that constitute clinical disorders and to examine the pattern of differences across types of disorder (Kessler, 2002).

Second, while the distress studies could only measure the current prevalence of distress, diagnostic studies provide information about the lifetime occurrence of disorders, allowing analyses that distinguish the risk of becoming ill, the lifetime prevalence, from the risk of having a persistent course (Kessler, 2002). This is an important distinction because race-ethnic differences in lifetime prevalence may involve different causal pathways and call for different solutions than differences in persistence. differences in lifetime prevalence implicate causal factors that occur prior to onset and can only be addressed through primary prevention (Neighbors, 1990). differences in persistence, on the other hand, can potentially be addressed through improvement in the quality and accessibility of mental health treatment. Recent research has shown that race-ethnic minorities are less likely to receive quality mental-health care (Young *et al*. 2001; Schneider *et al*. 2002) and that treatment programs targeted at these groups can improve treatment outcomes in depression (Miranda *et al*. 2003b).

The importance of distinguishing lifetime prevalence from persistence is also indicated by crude estimates (i.e. unadjusted for sociodemographic covariates) of ethnic differences in lifetime *versus* current prevalence reported by the ECA and the NCS. Both studies suggest that race-ethnic differences in lifetime prevalence and persistence are different, with lifetime prevalence generally being lower among minorities than Non-Hispanic Whites, and 12-month prevalence among lifetime cases generally being equal across groups or higher among minorities than Non-Hispanic Whites, when significant differences are found (Somervell *et al.* 1989; Kessler *et al.* 1994).

However, neither the ECA nor NCS analyses made race-ethnic differences a focus of attention. As a result, without more in-depth analysis, we cannot gauge the extent to which differences

suggested by these crude comparisons accurately reflect race-ethnic patterns in psychiatric morbidity. With respect to lifetime prevalence, apparent differences may widen with statistical control for SES. With respect to persistence, we do not know whether any differences will remain with a more stringent definition of persistent disorders when controlling for sociodemographic factors. Moreover, as previous research on non-specific distress and general health has demonstrated, we cannot assume that race-ethnic differences are consistent across all sectors of the population. Differences in the effect of race-ethnic status on mental health across the sexes, SES, stage of life, and birth cohorts may provide important clues to the causes of these disorders.

The goals of this paper are: first, to disaggregate the associations between race-ethnicity and the 12-month prevalence of broadly defined categories of DSM-III-R disorders in the NCS into two separate components due to race-ethnic differences in (1) lifetime risk and (2) conditional 12-month prevalence among lifetime cases controlling for age of onset and time since onset; and, second, to evaluate the extent to which these disaggregated associations vary by SES, using education as an indicator, age, and sex.

METHOD

Sample

As described in detail elsewhere (Kessler *et al.* 1994), the NCS is based on a stratified multi-stage area probability sample of persons aged 15–54 years representative of the non-institutionalized civilian population of the 48 contiguous states. The response rate was 82·4% (*n*=8098). A subsample of 5877 respondents, including all respondents screened positive for a psychiatric disorder or under age 25 years and a random sample of other respondents, completed an extended version of the interview schedule. Of these, 5657 respondents were Hispanic (9%), Non-Hispanic Black (12%) or Non-Hispanic White (78%) (detailed sociodemographic data available on request).

Assessment

DSM-III-R diagnoses were evaluated using the Composite International Diagnostic Instrument (CIDI), a structured instrument designed for use by trained non-clinician interviewers. The 13 DSM-III-R diagnoses examined in this report were grouped into three broad categories: Mood disorders (major depression, dysthymia, and mania), Anxiety disorders (agoraphobia, generalized anxiety disorder, panic disorder, simple phobia, social phobia, and post-traumatic stress disorder) and Substance Use disorders (alcohol abuse and dependence, drug abuse and dependence). Psychometric studies of the CIDI have found it to have good reliability and validity for diagnosing these disorders (Wittchen, 1994; Kessler *et al.* 1998). All interviews were conducted in English.

To determine race-ethnicity, respondents were first asked if they were Hispanic. All respondents were then asked which race they considered themselves with the options being White, Black, American Indian, Asian, and Other. If they indicated more than one race, respondents were asked which category best describes their racial origin. For the purposes of these analyses, respondents were divided into four mutually exclusive groups, Hispanics, Non-Hispanic Blacks, Non-Hispanic Whites and Others. Because of small numbers and heterogeneity, respondents categorized as Other are not included in the analysis. This approach to survey classification by race/ethnic status was found to be the best method in extensive tests conducted by the United States Census (Census Bureau, 1997; Snipp, 2003).

Age, gender and SES were considered as potential modifiers. Age was analyzed as a categorical variable. Data on educational attainment were used to provide statistical control for SES. To

accommodate the age range of the sample, we used data on respondents' parents' education to control for SES for time periods prior to the attainment of adult SES.

Analysis procedures

All analyses were conducted using the SUDAAN software package (Research Triangle Institute, NC, USA) that corrects standard errors to reflect the complex survey design. Differences in prevalence of disorders across ethnic groups were tested using χ^2 tests. Odds ratios associated with differences in lifetime prevalence across ethnic groups were estimated using discrete-time survival models to allow for statistical control for gender, age, period of the lifespan and SES (Allison, 1982; Willett & Singer, 1993).

Disorders were considered persistent if their first onset occurred at least 2 years prior to the interview and were present in the 12 months preceding the interview. The relationship between race-ethnicity and persistence was examined in logistic regression equations including controls for age of onset and time since onset so that comparisons are between people with equal opportunity to have remission. Controls were also introduced for gender and SES.

In analyses of both lifetime prevalence and persistence, we explored the consistency of odds ratios associated with race-ethnicity across cohort, gender and SES by testing for significant improvements in model fit with the addition of interaction terms. In the survival models for lifetime risk, we were also able to examine variation across periods of the lifespan. To increase the power of our tests of complex interactions we dichotomized the measures of age and period of the lifespan with a cut-point at age 25 years. In survival models these two dichotomies divided person-years into three groups: younger cohort earlier in life (all person-years for respondents who were less than 25 years at the time of interview), older cohort early in life (person-years prior to age 25 years among respondents who were 25 years or older at the time of interview), and older cohort later in life (person-years at or after age 25 years). Education was dichotomized into less than high school *versus* high school graduate.

RESULTS

Prevalence of psychiatric disorders

Table 1 presents comparisons across ethnic groups in 12-month prevalence, lifetime prevalence, and 12-month prevalence among lifetime cases. Few differences exist in 12-month prevalence. Compared with Non-Hispanic Whites, Non-Hispanic Blacks have significantly lower levels of substance use disorder (χ^2=29·35, df=1, p<0·001) and any disorder (χ^2=5·41, df=1, p=0·025).

The pattern, however, is quite different for lifetime prevalence, where Hispanics and Non-Hispanic Blacks have lower prevalence than Non-Hispanic Whites for all four categories; these differences are significant for six out of the eight comparisons. Hispanics have lower lifetime prevalence of substance use disorder (χ^2=6·01, df=1, p=0·018) and any disorder (χ^2=3·23, df=1, p=0·08). Non-Hispanic Blacks have lower lifetime prevalence that Non-Hispanic Whites across all four categories (mood: χ^2=13·32, df=1, p=0·001; anxiety: χ^2=3·13, df=1, p=0·084; substance use: χ^2=51·63, df=1, p<0·001; any: χ^2=40·78, df=1, p<0·001).

For 12-month prevalence among lifetime cases, however, six of the eight comparisons show Hispanics or Non-Hispanic Blacks to have higher 12-month prevalence with five of these comparisons reaching statistical significance. Hispanics have higher prevalence of mood disorder (χ^2=7·14, df=1, p=0·011), anxiety disorder (χ^2=9·04, df=1, p=0·005), and any disorder (χ^2=3·28, df=1, p=0·077). Non-Hispanic Blacks have higher levels of anxiety disorder (χ^2=10·18, df=1, p=0·003) and any disorder (χ^2=12·05, df=1, p=0·001), combined with lower prevalence of substance use disorder (χ^2=3·92, df=1, p=0·054).

Lifetime prevalence

Table 2 presents results from survival models predicting lifetime prevalence of disorders, with controls for age at interview, gender and period of the lifespan (Model 1). With Non-Hispanic Whites as the reference group, Hispanics have significantly lower odds of substance use disorder and Non-Hispanic Blacks have significantly lower odds of mood, substance use, and any disorder. Based on the fact that SES is strongly related to ethnicity, an expanded equation (Model 2) was estimated controlling for SES. Differences between Non-Hispanics Whites and other groups were magnified when SES was controlled. In this model, in addition to the significant differences found in Model 1, Hispanics have lower odds of any disorder and Non-Hispanic Blacks have lower odds of anxiety disorder.

Persistence of disorders

Analysis of ethnic patterns in persistence of disorders is presented in Table 3. Model 1, which controls for age and gender, shows higher odds of persistence of mood disorder among Hispanics as well as higher odds of persistence of mood, anxiety and any disorder among Non-Hispanic Blacks. There are no changes in the significance of these coefficients when SES is controlled (Model 2).

Variations in ethnic differences across subpopulations

Part (*a*) of Table 4 presents tests of interactions between ethnicity, gender, cohort, period of lifespan and SES in the prediction of lifetime prevalence. Few of the interactions are significant, indicating that ethnic differences are robust with respect to variation across subgroups of the population. This finding was confirmed by tests of all higher order interactions involving these variables (results not shown). The major exception to this overall consistency is that for both Hispanics and Non-Hispanic Blacks, males but not females have lower lifetime prevalence of anxiety disorders. Compared with Non-Hispanic Whites, the odds ratio (95% CI) among males was 0·73 (0·53–0·99) for Hispanics and 0·53 (0·37–0·76) for Non-Hispanic Blacks. Examination of the significant interaction between Hispanic ethnicity, birth cohort and period of the lifespan revealed variation in magnitude but not direction of the association found in the total sample (results not shown).

Results of tests for interactions between ethnicity, gender, birth cohort and SES in the prediction of persistence are reported in part (*b*) of Table 4. None of these interactions are significant, demonstrating consistency of ethnic differences across the population. Because of the reduced sample size for these models, we were not able to test higher order interactions in the analysis of persistence.

DISCUSSION

Disaggregation of the 12-month prevalence of psychiatric disorders into its components, lifetime prevalence and persistence, reveals a pattern that was unanticipated by the earlier generation of studies utilizing distress scales. Compared with Non-Hispanic Whites, Hispanics and Non-Hispanic Blacks tend to have a lower risk of having a psychiatric disorder in their lifetime, but, those who become ill tend to have more persistent disorders. In both cases, these differences are not explained by differences in SES and are generally consistent across subgroups of the population. The finding of greater persistence is the first finding of significant ethnic disparities in mental health reported in a national study of psychiatric disorders.

Hispanics had lifetime prevalence that was intermediate between Non-Hispanic Whites and Non-Hispanic Blacks, with differences from Non-Hispanic Whites reaching statistical significance for substance use disorders, any disorder, and, among males, anxiety disorders when gender, age and SES were controlled. Hispanics with mood disorders, on the other hand,

were nearly twice as likely to be persistently ill as Non-Hispanic Whites. Non-Hispanic Blacks had significantly lower lifetime prevalence for all categories of disorder, when age, gender and SES were controlled, although differences in anxiety disorder were significant only for males. Disorders among Non-Hispanic Blacks were significantly more likely to be persistent for the mood, anxiety and any disorder categories.

This finding adds to evidence that the association between psychiatric morbidity and socio-demographic risk factors is dependent on which aspect of course is examined. Sex differences in depression, for instance, have been consistently found for lifetime prevalence but not for persistence (Kessler, 2000). SES, on the other hand, has been shown to have independent effects on both prevalence and persistence, increasing morbidity in both cases (Lorant *et al*. 2003). With respect to race-ethnic differences, an earlier analysis of data on use of illicit drugs in the NCS found that Non-Hispanic Blacks were less likely to ever use illicit drugs than Non-Hispanic Whites, equally likely to become dependent after initiating use, but more likely to remain persistently dependent (Warner *et al*. 1995). If these findings stand up to further scrutiny, they have significant implications both for understanding the causal pathways that link social statuses and mental health outcomes and for public health policy designed to ameliorate health disparities.

Potential biases and limitations

The possibility that bias built into the survey methodology over- or under-estimates levels of disorder among some groups cannot be ruled out. Three particular types of bias may be involved. First, recall bias may play a role if it is different across ethnic groups, with Non-Hispanic Blacks in particular less likely to recall episodes of illness. Differential recall would lead to an underestimation of levels of disorder for particular groups and for the population as a whole. This bias does not appear to play a role here, however. If differential recall did play a role, we would expect ethnic differences to be greatest among the oldest age group during the earliest years of life, i.e. for those disorders that posed the greatest test of the respondent's memory. We did not find a significant interaction between ethnicity, age at interview, and period of the lifespan that would be consistent with this pattern.

Second, differential non-response would bias ethnic comparisons if rates of non-response differed across groups or if levels of psychiatric disorder among non-responders differed across groups. The NCS had a relatively high response rate at 82·4% and utilized a supplemental non-response survey to weight the sample for differences between responders and nonresponders (Little *et al*. 1997). Differences between non-responders from different ethnic groups would have to be quite large to account for these findings. An analysis of predictors of non-response in the NCS failed to find evidence of any such ethnic differences (Kessler *et al*. 1995).

Third, the survey items may differ across groups in their psychometric properties either because of their wording or because of phenomenological differences between groups in the experience of these disorders. Some evidence of differential item functioning across ethnic groups has been found for mental-health rating scales (Iwata *et al*. 2002). Such a bias in the diagnostic instrument could lead to either over- or under-estimation among certain groups. Differences in understanding the language of the survey instrument, however, are not likely to cause bias in the same direction across a wide variety of diagnostic categories. Moreover, differences in the psychometric properties of the survey questions do not necessarily imply measurement error. On the contrary, such differences may also arise from meaningful phenomenological differences between groups (Camilli & Shepard, 1994). Future research should examine whether differences in psychometric properties of diagnostic instruments across ethnic groups might account for these findings.

Limitations in the psychiatric nosology itself represent a separate potential challenge to this analysis (Kleinman, 1996; Leff, 1999). This possibility has been raised in particular with respect to Hispanics among whom the experience of psychiatric distress may not be captured in the official diagnostic criteria (Guarnaccia & Good, 1990). There is some evidence, for instance, that Puerto Ricans with significant levels of distress identified as *attaques de nervios* do not meet criteria for a DSM disorder (Lewis-Fernandez *et al*. 2002). If this is the case, then even perfectly unbiased estimates of DSM disorders in the community will underestimate clinically significant psychiatric distress in particular cultural groups. If such differences do play a role, however, we would expect some evidence of hidden psychopathology among Hispanic and Non-Hispanic Black respondents without disorders. It is important to note in this regard, however, that Non-Hispanic Blacks have been found to have more positive attitudes towards mental health treatment (Diala *et al*. 2001) and to be less likely to seek help for a mental disorder from a member of the clergy (Wang *et al*. 2003).

We also note the possibility of residual confounding of our race/ethnic comparisons by differences in SES that are not captured by our measures (Kaufman *et al*. 1997). We would expect, however, that better control for SES would increase the apparent differences between disadvantaged groups and Whites with respect to lifetime risk.

Interpretations and implications

There are two alternative models for interpreting race-ethnic differences in lifetime prevalence and persistence that point towards different causal pathways and different public health strategies. In one model, each of these parameters is seen as an outcome of a distinct process. Following this model, our data suggest that factors occurring before the onset of disorders reduce rather than increase the risk of incident psychiatric disorders among disadvantaged race-ethnic minorities. Factors that are concurrent with or subsequent to the onset of disorders, however, increase the risk of persistence among members of these groups who become ill. In both cases, these factors act equally across levels of SES, across birth cohorts, and, for lifetime prevalence, across periods of the lifespan with variation only by gender in the lifetime prevalence of anxiety disorders.

Based on this model, access to quality mental health treatment is a possible cause of and remedy for disparities in persistence. Although the differences we found were not changed when we controlled for receipt of any mental health treatment (results available on request), we know that the quality of mental health treatment is highly variable (Wang *et al.* 2000, 2003) and that Non-Hispanic Blacks are less likely to receive quality care following psychiatric hospital admissions (Schneider *et al*. 2002) or the most effective antidepressant medications (Skaer *et al*. 2000). A study of Medicaid-covered youth showed that Hispanics were less likely than Non-Hispanic Whites to receive treatment for depression after being diagnosed with the disorder (Richardson *et al*. 2003). There is some evidence from randomized trials of active recruitment of minority patients to treatment for depression that improvements in access and quality of care can improve clinical outcomes in these groups (Miranda *et al*. 2003*a*, b).

More generally, lack of quality care may be one of many factors, such as access to economic resources and disadvantages in the labor market, that make members of disadvantaged groups more vulnerable to adverse consequences of psychiatric disorders and thereby less likely to recover once they become ill. This more general point is partially supported by research on depressive episodes indicating that recovery can be impeded by events that exacerbate stressful life conditions or promoted by events that reverse or resolve those conditions (Kessler, 1997). Future research should examine whether race/ethnic differences in persistence of psychiatric disorders can be explained by risk of or vulnerability to life events that occur after the onset of those disorders.

We cannot rule out, however, a second model that views these two parameters as connected through a single mechanism that raises the threshold for onset of disorders among ethnic minorities but produces more persistent disorders when that threshold is crossed. According to this model, the relevant causal exposures occur prior to onset of the disorders, and cannot be reduced through interventions after the onset of disorder. Primary prevention in this model is of particular importance to disadvantaged groups because they are more likely to have persistent disorders once they become ill (Neighbors, 1990).

CONCLUSION

Hispanics, Blacks and Whites in the United States differ in many factors that may affect mental health status, such as experiences of discrimination, wealth, acculturation, religious affiliation, social mobility, and employment status (Schaefer, 2001; Nazroo, 2003). This descriptive analysis of nationally representative data on psychiatric disorders indicates that these various factors combine to have opposite impacts on lifetime prevalence and persistence of psychiatric disorders. Data soon to be available from the replication and follow-up of the NCS will allow us to update and extend these analyses with larger sample sizes, more detailed assessment of severity and course of disorders, and longitudinal data. The pattern of ethnic differences described here provides an empirical basis for future investigations that should focus both on methodological issues concerning the measurement of psychiatric morbidity across race/ethnic groups and on explanations for the patterns of morbidity that have been observed.

ACKNOWLEDGMENTS

Dr Breslau and Dr Su received support for this work from the National Institutes of Mental Health Grant 5 KO1 MH66057-02.

REFERENCES

Allison, PD. Discrete-time methods for the analysis of event histories. In: Leinhardt, S., editor. Sociological Methodology. Jossey-Bass; San Francisco: 1982. p. 61-98.

Bhugra, D.; Bahl, V., editors. Ethnicity: An Agenda for Mental Health. Royal College of Psychiatrists; London: 1999.

Camilli, G.; Shepard, LA. Methods for Identifying Biased Test Items. Sage Publications; Thousand Oaks: 1994.

Census Bureau. Results of the 1996 Race and Ethnic Targeted Test. US Bureau of the Census; Washington DC: 1997.

Diala C, Muntaner C, Walrath C, Nickerson K, LaViest TA, Leaf PJ. Racial/ethnic differences in attitudes toward seeking professional mental health services. American Journal of Public Health 2001;91:805–807. [PubMed: 11344893]

Dohrenwend BP. Social status and psychological disorder: an issue of substance and an issue of method. American Sociological Review 1966;31:14–34. [PubMed: 5903385]

Dohrenwend BP, Levav I, Shrout P, Schwartz S, Naveh G, Link B, Skodol A, Stueve A. Socioeconomic status and psychiatric disorders: the causation-selection issue. Science 1992;255:946–952. [PubMed: 1546291]

Guarnaccia PJ, Good BJ. A critical review of epidemiological studies of Puerto Rican mental health. American Journal of Psychiatry 1990;147:1449–1456. [PubMed: 2221155]

Iwata N, Turner RJ, Lloyd DA. Race/ethnicity and depressive symptoms in community-dwelling young adults: a differential item functioning analysis. Psychiatry Research 2002;110:281–289. [PubMed: 12127478]

Jackson JS, Brown TN, Williams DR, Torres M, Sellers SL, Brown K. Racism and the physical and mental health status of African Americans: a thirteen year national panel study. Ethnicity and Disease 1996;6:132–147. [PubMed: 8882842]

Jenkins R, Lewis G, Bebbington P, Brugha T, Farrell M, Gill B, Meltzer H. The National Psychiatric Morbidity surveys of Great initial – Britain findings from the household survey. Psychological Medicine 1997;27:775–789. [PubMed: 9234456]

Karlsen S, Nazroo JY. Relation between racial discrimination, social class, and health among ethnic minority groups. American Journal of Public Health 2002;92:624–631. [PubMed: 11919063]

Kaufman JS, Cooper RS, McGee0 DL. Socioeconomic status and health in blacks and whites: the problem of residual confounding and the resiliency of race. Epidemiology 1997;8:621–628. [PubMed: 9345660]

Kessler, RC. Annual Review of Psychology. Annual Reviews; Palo Alto, CA: 1997. The effects of stressful life events on depression; p. 191-214.

Kessler RC. Psychiatric epidemiology: selected recent advances and future directions. Bulletin of the World Health Organization 2000;78:464–474. [PubMed: 10885165]

Kessler RC. The categorical versus dimensional assessment controversy in the sociology of mental illness. Journal of Health and Social Behavior 2002;43:171–188. [PubMed: 12096698]

Kessler RC, Little RJA, Groves RM. Advances in strategies for minimizing and adjusting for survey nonresponse. Epidemiologic Reviews 1995;17:192–204. [PubMed: 8521937]

Kessler RC, McGonagle KA, Zhao S, Nelson CB, Hughes M, Eshleman S, Wittchen H-U, Kendler KS. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. Archives of General Psychiatry 1994;51:8–19. [PubMed: 8279933]

Kessler RC, Neighbors HW. A new perspective on the relationships among race, social class and psychological distress. Journal of Health and Social Behavior 1986;27:107–115. [PubMed: 3734380]

Kessler RC, Wittchen H-U, Abelson JM, McGonagle KA, Schwarz N, Kendler KS, Knauper B, Zhao S. Methodological studies of the Composite International Diagnostic Interview (CIDI) in the United States. International Journal of Methods in Psychiatric Research 1998;7:33–55.

Kleinman, A. How is culture important for DSM-IV?. In: Mezzich, J.; Kleinman, A.; Fabrega, H.; Parron, D., editors. Culture and Psychiatric Diagnosis: A DSM-IV Perspective. American Psychiatric Press; Washington, DC: 1996. p. 15-25.

LaViest TA. Why we should continue to study race … but do a better job: an essay on race, racism and health. Ethnicity and Disease 1996;6:21–29. [PubMed: 8882833]

Leff, J. Epidemiological factors in research with ethnic minorities. In: Bhugra, D.; Bahl, V., editors. Ethnicity: An Agenda for Mental Health. Royal College of Psychiatrists; London: 1999. p. 40-47.

Lewis-Fernandez R, Guarnaccia PJ, Martinez IE, Salman E, Schmidt A, Liebowitz M. Comparative phenomenology of ataques de nervios, panic attacks, and panic disorder. Culture, Medicine and Psychiatry 2002;26:199–223.

Little RJA, Lewitsky S, Heeringa S, Lepkowski J, Kessler RC. Assessment of weighting methodology for the National Comorbidity Survey. American Journal of Epidemiology 1997;146:439–449. [PubMed: 9290504]

Lorant V, Deliege D, Eaton WW, Robert A, Philippot P, Ansseau M. Socioeconomic inequalities in depression: a meta-analysis. American Journal of Epidemiology 2003;157:98–112. [PubMed: 12522017]

Malzberg, B.; Lee, ES. Migration and Mental Disease: A Study of First Admissions to Hospitals for Mental Disease, New York, 1939–1941. Social Science Research Council; New York: 1956.

Miranda J, Chung JY, Green BL, Krupnick J, Siddique J, Revicki DA, Belin T. Treating depression in predominantly low-income young minority women: a randomized controlled trial. Journal of the American Medical Association 2003a;290:57–65. [PubMed: 12837712]

Miranda J, Duan N, Sherbourne C, Schoenbaum M, Lagomasino I, Jackson-Triche M, Wells KB. Improving care for minorities: can quality improvement interventions improve care and outcomes for depressed minorities? Results of a randomized, controlled trial. Health Services Research 2003b; 38:613–630. [PubMed: 12785564]

Nazroo, JY. Ethnicity, Class and Health. Policy Studies Institute; London: 2001.

Nazroo JY. The structuring of ethnic inequalities in health: economic position, racial discrimination and racism. American Journal of Public Health 2003;93:277–284. [PubMed: 12554585]

Neighbors HW. The prevention of psychopathology in African-Americans: an epidemiologic perspective. Community Mental Health Journal 1990;26:167–179. [PubMed: 2191834]

Ng-Mak DS, Dohrenwend BP, Abraido-Lanza AF, Turner JB. A further analysis of race differences in the National Longitudinal Mortality Study. American Journal of Public Health 1999;89:1748–1751. [PubMed: 10553402]

Parker, S.; Kleiner, RJ. Mental Illness in the Urban Negro Community. The Free Press; New York: 1966.

Richardson L, DiGiuseppe D, Garrison M, Christakis D. Depression in Medicaid-covered youth: differences by race and ethnicity. Archives of Pediatric and Adolescent Medicine 2003;157:984–989.

Robins, LN.; Regier, DA., editors. Psychiatric Disorders in America: The Epidemiologic Catchment Area Study. The Free Press; New York: 1991.

Sacco RL, Boden-Albala B, Gan R, Chen X, Kargman DE, Shea S, Paik MC, Hauser WA. Stroke incidence among white, black, and Hispanic residents of an urban community: the Northern Manhattan Stroke Study. American Journal of Epidemiology 1998;147:259–268. [PubMed: 9482500]

Schaefer, RT. Race and Ethnicity in the United States. Prentice Hall; Upper Saddle River: 2001.

Schneider EC, Zaslavsky AM, Epstein A. Racial disparities in the quality of care for enrollees in medicare managed care. Journal of the American Medical Association 2002;287:1288–1294. [PubMed: 11886320]

Skaer TL, Sclar DA, Robison LM, Galin RS. Trends in the rate of depressive illness and use of antidepressant pharmacotherapy by ethnicity/race: an assessment of office-based visits in the United States, 1992–1997. Clinical Therapeutics 2000;22:1575–1589. [PubMed: 11192148]

Smedley, BD.; Stith, AY.; Nelson, AR., editors. Unequal Treatment: Confronting Racial and Ethnic Disparities in Health-care. National Academy Press; Washington, DC: 2003.

Snipp CM. racial measurement in the American census: past practices and implications for the future. Annual Review of Sociology 2003;29:563–588.

Somervell PD, Leaf PJ, Weissman MM, Blazer DG, Bruce ML. The prevalence of major depression in black and white adults in five United States communities. American Journal of Epidemiology 1989;130:725–735. [PubMed: 2788995]

US Department of Health and Human Services. Healthy People 2010: Understanding and Improving Health. US Government Printing Office; Washington, DC: 2000.

US Department of Health and Human Services. Mental Health: Culture, Race and Ethnicity – A Supplement to Mental Health: A Report of the Surgeon General. US Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Center for Mental Health Services; Rockville: 2001.

Vega WA, Rumbaut RG. Ethnic minorities and mental health. Annual Review of Sociology 1991;17:351–383.

Wang PS, Berglund P, Kessler RC. Recent care of common mental disorders in the United States: prevalence and conformance with evidence-based recommendations. Journal of General Internal Medicine 2000;15:284–292. [PubMed: 10840263]

Wang PS, Berglund PA, Kessler RC. Patterns and correlates of contacting clergy for mental disorders in the United States. Health Services Research 2003;38:647–673. [PubMed: 12785566]

Warheit GJ, Holzer CE 3rd, Arey SA. Race and mental illness: an epidemiologic update. Journal of Health and Social Behavior 1975;16:243–256. [PubMed: 1194653]

Warner LA, Kessler RC, Hughes M, Anthony JC, Nelson CB. Prevalence and correlates of drug use and dependence in the United States. Results from the National Comorbidity Survey. Archives of General Psychiatry 1995;52:219–229. [PubMed: 7872850]

Willett JB, Singer JD. Investigating onset, cessation, relapse, and recovery: why you should, and how you can, use discrete-time survival analysis to examine event occurrence. Journal of Consulting and Clinical Psychology 1993;61:952–965. [PubMed: 8113496]

Williams DR. Race and health: basic questions, emerging directions. Annals of Epidemiology 1997;7:322–333. [PubMed: 9250627]

Wittchen H-U. Reliability and validity studies of the WHO-Composite International Diagnostic Interview (CIDI): a critical review. Journal of Psychiatric Reseach 1994;28:57–84.

Wong MD, Shapiro MF, Boscardin J, Ettner S. Contribution of major diseases to disparities in mortality. New England Journal of Medicine 2002;347:1585–1592. [PubMed: 12432046]

Young AS, Klap R, Sherbourne CD, Wells KB. The quality of care for depressive and anxiety disorders in the United States. Archives of General Psychiatry 2001;58:55–61. [PubMed: 11146758]

Table 1

Twelve-month prevalence, lifetime prevalence, and 12 months among lifetime prevalence of psychiatric disorders across ethnic groups

	Hispanic		Non-Hispanic Black		Non-Hispanic White		
Disorder	**%**	**(S.E.)**	**%**	**(S.E.)**	**%**	**(S.E.)**	**χ^2 (df=2)**
			12-month prevalence				
Any Mood	13·4	(1·6)	9·3	(1·2)	10·7	(0·6)	*
Any Anxiety	21·4	(1·9)	18·7	(2·3)	18·9	(0·7)	
Any Substance Use	10·7	(1·7)	6·3†††	(0·9)	12·3	(0·4)	***
Any disorder	31·6	(2·4)	26·1††	(2·3)	31·6	(0·8)	*
			Lifetime prevalence				
Any Mood	17·9	(1·8)	13·7†††	(1·6)	19·8	(0·7)	***
Any Anxiety	28·4	(2·4)	24·7†	(2·6)	29·1	(0·9)	
Any Substance Use	22·9††	(2·2)	13·1†††	(1·5)	29·5	(1·0)	***
Any disorder	46·0†	(2·8)	35·1†††	(2·5)	52·0	(1·1)	***
			12-month prevalence among lifetime cases				
Any Mood	29·1††	(2·6)	26·4	(3·0)	20·6	(1·1)	**
Any Anxiety	46·6†††	(3·2)	53·2†††	(4·2)	36·4	(1·1)	***
Any Substance Use	23·3	(3·1)	17·8†	(2·5)	23·6	(0·9)	
Any disorder	68·8†	(3·3)	74·2†††	(3·3)	60·8	(1·1)	***

Asterisks indicate significance of χ^2 test with 2 df for association between race-ethnicity and prevalence across the three groups. Dagger symbols indicate significance of χ^2 test with 1 df for difference in prevalence between Non-Hispanic Whites and each minority group.

*p<0·1

**p<0·05

***p<0·0.

$^{\dagger}p$<0·1

$^{\dagger\dagger}p$<0·05

$^{\dagger\dagger\dagger}p$<0·0.

Table 2

Odds ratios (OR) for first onset of psychiatric disorders comparing African-Americans and Hispanics with Non-Hispanic (NH) Whites

	Mood		Anxiety		Substance Use		Any disorder	
Ethnicity	**OR**	**(95% CI)**	**OR**	**(95% CI)**	**OR**	**(95% CI)**	**OR**	**(95% CI)**
Model 1: Control for gender and age								
NH White	1·00		1·00		1·00		1·00	
NH Black	**0·65**	(0·49–0·85)	0·79	(0·62–1·01)	**0·41**	(0·32–0·53)	**0·59**	(0·49–0·72)
Hispanic	0·91	(0·74–1·13)	1·00	(0·83–1·20)	**0·77**	(0·60–0·98)	0·87	(0·73–1·03)
Model 2: Additional time varying control for education								
NH White	1·00		1·00		1·00		1·00	
NH Black	**0·64**	(0·49–0·85)	**0·75**	(0·58–0·96)	**0·40**	(0·31–0·52)	**0·58**	(0·48–0·70)
Hispanic	0·91	(0·73–1·15)	0·89	(0·72–1·11)	**0·77**	(0·60–0·98)	**0·83**	(0·69–1·00)

Odds ratios estimated in discrete-time survival analyses.

Odds ratios in bold are significantly lower than 1 at p=0·05 level.

Table 3

Persistence of psychiatric disorders across ethnic groups

	Model 1		Model 2	
Disorder	**Non-Hispanic Black**	**Hispanic**	**Non-Hispanic Black**	**Hispanic**
Any Mood	1·87*	2·33*	1·79*	1·90*
Any Anxiety	1·50*	1·33	1·51*	1·14
Any Substance Use	1·48	1·25	1·46	1·07
Any disorder	1·76*	1·23	1·70*	1·03

Numbers represent odds ratios comparing persistence across groups with Non-Hispanic Whites as the reference group. Odds ratios were estimated in logistic regressions of 12-month disorder among respondents with a lifetime disorder.

Model 1 controls for age and sex.

Model 2 adds control for education.

*Significant at p=0·05.

Table 4

Test of interactions between ethnicity and other sociodemographic variables in prediction of onset and persistence

		Mood disorders		Anxiety disorders		Substance Use disorders	
	df	χ^2	*p*	χ^2	*p*	χ^2	*p*
(*a*) Onset							
Interactions							
Hispanic*Cohort-Person Yr	2	5·67	0·06	0·16	0·92	1·71	0·43
Non-Hispanic Black*Cohort-Person Yr	2	3·07	0·22	3·77	0·15	15·73**	<0·001*
Hispanic*Sex	1	0·04	0·84	4·92*	0·03*	0·46	0·50
Non-Hispanic Black*Sex	1	0·36	0·55	5·29*	0·02*	0·04	0·85
Hispanic*Education	1	0·73	0·39	0·00	0·99	0·01	0·94
Non-Hispanic Black*Education	1	0·04	0·84	3·65	0·06	0·16	0·69
(*b*) Persistence							
Interactions							
Hispanic*Cohort	1	0·55	0·46	0·02	0·90	1·16	0·28
Non-Hispanic Black*Cohort	1	0·99	0·32	0·75	0·39	0·26	0·61
Hispanic*Gender	1	2·40	0·12	0·15	0·70	0·01	0·93
Non-Hispanic Black*Gender	1	1·05	0·31	0·05	0·82	0·24	0·62
Hispanic*Education	1	0·18	0·67	0·51	0·48	2·84	0·09
Non-Hispanic Black*Education	1	1·91	0·17	0·03	0·87	0·26	0·61

*Significant at p=0·05.