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Specifying race-ethnic differences in risk for psychiatric disorder in a USA national sample

JOSHUA BRESLAU[1]*, SERGIO AGUILAR-GAXIOLA[2], KENNETH S. KENDLER[3], MAXWELL SU[1], DAVID WILLIAMS[4] AND RONALD C. KESSLER[1]

[1] *Department of Health Care Policy, Harvard Medical School, Boston, MA*; [2] *University of California, Davis School of Medicine, Sacramento, CA*; [3] *Departments of Psychiatry and Human Genetics, Medical College of Virginia/Virginia Commonwealth University, Richmond, VA*; [4] *Department of Sociology and Institute for Social Research, University of Michigan, Ann Arbor, MI, USA*

ABSTRACT

Background. Epidemiological studies have found lower than expected prevalence of psychiatric disorders among disadvantaged race-ethnic minority groups in the USA. Recent research shows that this is due entirely to reduced lifetime risk of disorders, as opposed to persistence. Specification of race-ethnic differences with respect to clinical and social characteristics can help identify the protective factors that lead to lower lifetime risk among disadvantaged minority groups.

Method. Data on 5424 Hispanics, non-Hispanic Blacks, and non-Hispanic Whites came from the National Comorbidity Survey Replication, a nationally representative survey conducted with the World Mental Health version of the Composite International Diagnostic Interview. Race-ethnic differences in risk of disorders were compared across specific diagnoses, ages of onset, cohorts and levels of education.

Results. Both minority groups had lower risk for common internalizing disorders: depression, generalized anxiety disorder, and social phobia. In addition, Hispanics had lower risk for dysthymia, oppositional-defiant disorder and attention deficit hyperactivity disorder; non-Hispanic Blacks had lower risk for panic disorder, substance use disorders and early-onset impulse control disorders. Lower risk among Hispanics, relative to non-Hispanic Whites, was found only among the younger cohort (age $\leqslant$43 years). Lower risk among minorities was more pronounced at lower levels of education.

Conclusion. The pattern of race-ethnic differences in risk for psychiatric disorders suggests the presence of protective factors that originate in childhood and have generalized effects on internalizing disorders. For Hispanics, but not for non-Hispanic Blacks, the influence of these protective factors has emerged only recently.

INTRODUCTION

Social adversity is commonly associated with increased risk for psychiatric disorders (Dohrenwend, 2000). However, community studies in the USA have not found elevated point prevalence of psychiatric disorders among disadvantaged racial and ethnic minority groups (Somervell *et al.* 1989; Kessler *et al.* 1994), despite higher levels of social adversity experienced by these groups (Clark *et al.* 1999; Williams, 1999; Turner & Lloyd, 2004). In the National Comorbidity Survey (NCS), the first nationally representative survey of psychiatric disorders conducted in the USA, we showed that the lower than expected point prevalence of DSM-III mental disorders among racial-ethnic minorities was attributable exclusively to differences in lifetime risk of these disorders

* 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)

as opposed to course of illness. Indeed, the course of illness tended to be more persistent among minorities than non-Hispanic Whites in that study (Breslau *et al.* 2005). Thus, understanding reasons for the lower risk of ever developing a mental disorder appears to be the key to understanding the lower than expected prevalence of mental disorders among disadvantaged racial and ethnic minority groups in the USA.

In this report we take the next step towards identifying causes of race-ethnic differences in lifetime risk of psychiatric disorders by specifying these differences in greater detail with respect to clinical and sociodemographic characteristics. Using data from a recent national survey, the National Comorbidity Survey Replication (NCS-R), we investigate variation in race-ethnic differences across (1) individual DSM-IV disorders, (2) age of onset of disorder, (3) birth cohorts, and (4) educational attainment. Results of these comparisons will help guide the investigation of potential protective factors that result in lower lifetime risk among race-ethnic minorities.

Our previous report considered race-ethnic differences in three broad classes of disorder: mood, anxiety and substance use disorders. However, it is important to know whether the differences we observed are consistent across disorders within these classes, implicating protective factors with generalized effects, or whether they are attributable to differences in a small number of disorders, implicating factors with disorder-specific effects (Aneshensel *et al.* 1991). In this analysis we test for variation in race-ethnic differences across individual disorders within each class of disorders. In addition, we examine an additional class of disorders, impulse control disorders, that was not included in the earlier survey.

Specifying race-ethnic differences in early *versus* late age of onset of disorder can help identify the developmental periods during which protective factors exert their influence. If, for instance, reduced risk of psychiatric disorder begins with early-onset disorders, we should consider protective factors present in childhood environments rather than adult social experiences. It has been suggested that stressors associated with minority race-ethnicity vary across the lifespan; minority children may be sheltered from the negative impact that discrimination in labor markets and other areas of adult life has on mental health in adults (Gore & Aseltine, 2003). This hypothesis predicts that the lower risk among minorities will be stronger for early-onset than for late-onset disorders.

Specification of race-ethnic differences with respect to birth cohorts can help identify causal factors by locating the differences historically. This is of interest because the social conditions affecting minorities in the USA have changed in recent decades in ways that influence the experience of social adversity, such as income (Levy, 1998) and educational attainment (Kao & Thompson, 2003). However, studies using distress scales have found that race-ethnic differences have not varied from the 1950s through the 1990s, despite these changes (Thomas & Hughes, 1986). We do not know the extent to which the same is true for lifetime risk of psychiatric disorders. Continuity in race-ethnic differences across birth cohorts would point to factors that are transmitted across generations despite historical changes in social conditions.

The association of disadvantaged minority status with lower lifetime risk of disorders suggests that the relationship between socioeconomic status (SES) and risk of onset may vary across race-ethnic groups (Williams *et al.* 1992; Williams, 1997). Two theories of such variation have been suggested. First, the 'double jeopardy' theory suggests that morbidity will be particularly elevated among low SES members of disadvantaged minority groups. This theory was supported by studies using distress scales that found higher levels of distress among non-Hispanic Blacks relative to non-Hispanic Whites only among those with low SES (Kessler & Neighbors, 1986; McLeod & Owens, 2004). Second, the 'declining returns' theory predicts the opposite pattern, with higher morbidity among minority groups at higher levels of SES. This theory, suggested by studies which found that minorities have lower economic returns to investment in educational credentials (Chiswick, 1988), has been supported in research on indicators of general physical health (Farmer & Ferraro, 2005). With respect to mental health the 'declining returns' pattern is also consistent with the suggestion that social stressors are most severe for middle class minorities who have the highest expectations but also face the

most severe competition in labor markets (Parker & Kleiner, 1966; Neckerman *et al.* 1999; Cole & Omari, 2003; Jackson & Stewart, 2003).

METHOD

Sample

As detailed elsewhere (Kessler *et al.* 2004*b*), the NCS-R is a nationally representative survey of English-speaking household residents aged 18 and older in the coterminous USA. Face-to-face interviews were carried out by professional interviewers from the Institute for Social Research at the University of Michigan between February 2001 and April 2003. The response rate was 70·9%. The survey was administered in two parts. Part I included a core diagnostic assessment of all respondents ($n=9282$) that took an average of about one hour to administer. Part II included questions about risk factors, consequences, other correlates and additional disorders. In an effort to reduce respondent burden and control study costs, Part II was administered only to 5692 of the 9282 Part I respondents, including all Part I respondents with a lifetime disorder plus a probability subsample of other respondents ($n=5692$). This paper examines Part II respondents who were Hispanic, non-Hispanic Black or non-Hispanic White ($n=5424$). Interviewers explained the study and obtained verbal informed consent prior to beginning each interview. Recruitment and consent were approved by the Human Subjects Committees of Harvard Medical School and the University of Michigan.

The data were weighted to adjust for differential probabilities of selection, differential non-response, and residual differences between the sample and tract-level 2000 Census population on sociodemographic variables. An additional Part II weight adjusted for over-sampling of Part I cases. Weighting is described in more detail elsewhere (Kessler *et al.* 2004*b*).

Measures

Diagnostic assessment

NCS-R diagnoses are based on the World Mental Health Survey Initiative Version of the World Health Organization Composite International Diagnostic Interview (WMH-CIDI) (Kessler & Ustun, 2004) a fully structured lay-administered diagnostic interview that generates both ICD-10 (WHO, 1991) and DSM-IV (APA, 1994) diagnoses. Four classes of disorder, consisting of a total of 18 DSM-IV disorders, are examined here: anxiety disorders [panic disorder, agoraphobia without panic disorder, specific phobia, social phobia, generalized anxiety disorder (GAD), post-traumatic stress disorder (PTSD), obsessive-compulsive disorder (OCD)]; mood disorders (major depressive disorder, dysthymia, bipolar I and II disorders); a series of four disorders that share a common feature of difficulties with impulse-control [intermittent explosive disorder, oppositional-defiant disorder (ODD), conduct disorder, attention-deficit/hyperactivity disorder (ADHD)]; and two substance use disorders (alcohol abuse or dependence, drug abuse or dependence). As described elsewhere (Kessler *et al.* 2004*a*), blind clinical re-interviews with the Structured Clinical Interview for DSM-IV (SCID) (First *et al.* 2002) found generally good concordance with WMH-CIDI diagnoses for anxiety, mood, and substance use disorders. Impulse-control diagnoses have not been validated.

Onset

The timing of onset of disorders was determined retrospectively using reports of age of onset. These reports were obtained in the WMH-CIDI using a question series designed to avoid the implausible response patterns obtained using the standard CIDI age-of-onset question (Simon & VonKorff, 1995). The sequence began with a question designed to emphasize the importance of accurate response: 'Can you remember your *exact age* the *very first time* you (HAD THE SYNDROME)?' Respondents who answered 'no' were probed for a bound of uncertainty by moving up the age range incrementally (e.g. 'Was it before you first started school?', 'Was it before you became a teenager?', etc.). Age of onset was set at the upper end of the bound (e.g. age 12 for respondents who reported that onset was before they became teenagers). Experimental research shows this question sequence yields responses with a much more plausible age-of-onset distribution than the standard CIDI age-of-onset question (Knauper *et al.* 1999).

Race-ethnicity

Race-ethnicity was determined using the two-question format recommended by the U.S. Census Bureau on the basis of the Race and Ethnic Targeted Test (USA Census Bureau, 1997). Respondents were first asked if they were 'of Hispanic or Latino descent'. All respondents were then asked 'Which of the following best describes your race: American Indian, Alaska Native, Asian, Black or African-American, Native Hawaiian, Pacific Islander, or White?' All respondents who indicated Hispanic or Latino descent are considered Hispanic. Non-Hispanics were then classified according to the race category they selected. Non-Hispanics who were neither Black nor White were excluded from this analysis.

Age of onset and cohort variables

Age of onset was dichotomized into early and late time periods using the median of the distribution of age of onset for each class of disorders. The cut-points were: 10 years of age for anxiety disorders, 22 years of age for mood disorders, 10 years of age for impulse control disorders and 18 years of age for substance use disorders. The sample was divided into younger (age $\leqslant 43$) and older (age > 43) cohorts based on the median age of the sample.

Parental and respondent education

Data on parental and respondent education were used as indicators of SES in childhood and adulthood respectively. The educational attainment of the respondent's parent (i.e. chief breadwinner in childhood) was classified into four categories (0–11, 12, 13–15, 16+). Respondent education was defined as a time-varying covariate assuming an orderly progression through years of education. Person-years were classified as (1) student prior to graduation from high school, (2) student after graduation from high school, (3) non-student with less than 12 years of education, (4) non-student with 12 years of education, (5) non-student with 13–15 years of education, (6) non-student with 16 or more years of education.

Statistical analysis

Lifetime prevalence was estimated as the proportion of respondents who ever had a given disorder up to their age at the time of the interview. Since race-ethnic groups differ in their age distributions, comparisons of lifetime prevalence are not equivalent to comparisons of lifetime risk. Lifetime risk was examined using discrete-time survival analysis with person-year the unit of analysis and statistical controls for age and sex (Efron, 1988). Variation in race-ethnic differences across disorders, cohorts, ages of onset, and education were evaluated by including interactions between these variables and race-ethnicity.

Standard errors of prevalence estimates and survival coefficients were estimated using the Taylor series linearization method (Wolter, 1985) implemented in the SUDAAN software system (Research Triangle Institute, 2002). Multivariate significance tests were made with Wald χ^2 tests using Taylor series design-based coefficient variance–covariance matrices. All significance tests were evaluated at the 0·05 level with two-sided tests.

RESULTS

Sample characteristics

Reflecting the USA population, both Hispanics and non-Hispanic Blacks were younger (Meyer, 2001) and had lower levels of education (Bauman & Graf, 2003) and lower levels of parental education (Kao & Thompson, 2003) than non-Hispanic Whites (Table 1). Also consistent with USA census data, the ratio of men to women is highest among Hispanics and lowest among non-Hispanic Blacks (Spraggins, 2003).

Lifetime prevalence

Compared with non-Hispanic Whites, Hispanics had lower lifetime prevalence for any disorder, two of the four classes of disorders and six of the 16 specific disorders (Table 2). These differences were significant for dysthymia, depression, GAD and social phobia. For non-Hispanic Blacks, lower lifetime prevalence was more pervasive: non-Hispanic Blacks had lower prevalence than non-Hispanic Whites for any disorder, all four classes of disorders and for 13 of the 16 specific disorders. Differences were significant for any disorder, all classes of disorder except impulse control and for depression, GAD, panic disorder, social phobia and alcohol use disorder. There is only one

Table 1. *Sociodemographic characteristics of Hispanics, non-Hispanic Blacks and non-Hispanic Whites in the NCS-R*[a]

	Hispanic		Non-Hispanic Black		Non-Hispanic White		Total	
	%	(S.E.)	%	(S.E.)	%	(S.E.)	%	(S.E.)
Sex								
Female	50·2	(5·0)	57·7	(1·9)*	52·5	(1·1)	52·9	(1·0)
Male	49·8	(5·0)	42·3	(1·9)	47·5	(1·1)	47·1	(1·0)
Age (years)								
18–29	37·4	(2·7)*	29·7	(2·8)*	19·9	(1·3)	23·2	(1·1)
30–44	35·4	(2·9)	30·5	(2·0)	27·3	(1·0)	28·6	(0·8)
45–59	18·0	(2·6)	23·9	(2·1)	28·3	(1·3)	26·6	(1·0)
60+	9·2	(2·3)	15·9	(2·3)	24·5	(1·3)	21·6	(1·0)
Education (years)								
<12	34·8	(3·7)*	22·0	(3·1)*	13·2	(1·0)	16·8	(0·9)
12	35·4	(2·9)	36·6	(4·7)	31·9	(1·5)	32·9	(1·1)
13–15	19·1	(2·4)	27·8	(1·6)	28·7	(1·1)	27·5	(0·8)
16+	10·8	(1·8)	13·6	(1·9)	26·2	(1·4)	22·8	(1·0)
Parental education (years)								
<12	60·1	(3·7)*	40·5	(3·2)*	30·0	(1·4)	34·8	(1·5)
12	18·9	(2·4)	33·2	(2·2)	33·1	(1·0)	31·5	(0·9)
13–15	6·5	(0·7)	15·3	(2·5)	14·4	(0.6)	13·6	(0·5)
16+	14·5	(3·0)	11·0	(1·4)	22·4	(1·6)	20·0	(1·4)
(*n*)	(527)		(717)		(4180)		(5424)	

[a] Part II NCS-R respondents who are either Hispanic, non-Hispanic Black, or non-Hispanic White. All respondents in other race-ethnic groups ($n=268$) are excluded.

* Significant difference from non-Hispanic Whites evaluated with χ^2 test at $p=0{\cdot}05$.

Table 2. *Comparison of lifetime prevalence of psychiatric disorders across race-ethnic groups*

	Hispanic		Non-Hispanic Black		Non-Hispanic White	
	%	(S.E.)	%	(S.E.)	%	(S.E.)
I. Anxiety disorders						
Panic disorder	5·4	(0·9)	3·1*	(0·6)	4·9	(0·2)
Generalized anxiety disorder	4·8*	(0·9)	5·1*	(0·7)	8·6	(0·5)
Agoraphobia without panic	2·7	(0·6)	2·3	(0·5)	2·4	(0·2)
Social phobia	8·8*	(1·3)	10·8	(1·2)	12·6	(0·5)
Specific phobia	13·1	(1·5)	11·7	(1·2)	12·5	(0·5)
Post-traumatic stress disorder	5·9	(1·0)	7·1	(0·9)	6·8	(0·5)
Obsessive-compulsive disorder	1·2	(0·6)	0·5	(0·2)	0·4	(0·1)
Any anxiety	24·9	(2·2)	23·8*	(1·9)	29·4	(1·0)
II. Mood disorders						
Dysthymic disorder	2·2*	(0·5)	3·5	(0·5)	4·3	(0·3)
Major depression	13·5*	(1·5)	10·8*	(1·2)	17·9	(0·7)
Bipolar disorder	4·3	(1·0)	4·9*	(0·6)	3·2	(0·2)
Any	18·3	(1·6)	16·0*	(1·3)	21·9	(0·9)
III. Impulse control disorders						
Intermittent explosive disorder	9·9	(1·4)	7·2	(1·1)	7·3	(0·5)
Attention deficit hyperactivity disorder	4·6	(0·8)	3·4	(0·7)	4·6	(0·4)
Oppositional-defiant disorder	5·4	(0·8)	5·6	(1·0)	5·7	(0·6)
Conduct	6·9	(1·0)	4·9	(1·2)	5·0	(0·4)
Any	17·9	(1·8)	14·5	(1·9)	15·3	(0·8)
IV. Substance disorders						
Alcohol abuse or dependence	15·0	(2·3)	9·5*	(0·9)	13·4	(0·6)
Drug abuse or dependence	9·1	(1·4)	6·3	(1·0)	7·9	(0·4)
Any	16·1	(2·3)	10·8*	(1·0)	14·8	(0·6)
V. Any disorder	43·7	(4·0)	38·5*	(2·8)	47·6	(1·2)

* Significant difference from non-Hispanic Whites evaluated with χ^2 test at $p=0{\cdot}05$.

Table 3. *Race-ethnic differences in lifetime risk of DSM-IV disorders*[a]

	Hispanic		Non-Hispanic Black	
	OR	(95% CI)	OR	(95% CI)
I. Anxiety disorders				
Any anxiety disorder	0·8*	(0·6–0·9)	0·7*	(0·6–0·9)
	$\chi^2_{(6)}=24$ ($p=0·001$)		$\chi^2_{(6)}=19·3$ ($p=0·004$)	
Panic disorder	1	(0·8–1·4)	0·6*	(0·4–0·8)
Generalized anxiety disorder	0·6*	(0·4–0·8)	0·6*	(0·4–0·8)
Agoraphobia without panic	1·1	(0·6–1·9)	0·9	(0·6–1·5)
Social phobia	0·6*	(0·4–0·9)	0·8*	(0·6–1·0)
Specific phobia	1	(0·8–1·4)	0·9	(0·7–1·1)
Post-traumatic stress disorder	0·9	(0·6–1·2)	1	(0·7–1·3)
Obsessive-compulsive disorder	2·7	(0·8–8·9)	1	(0·4–2·5)
II. Mood disorders				
Any mood disorder	0·7*	(0·6–0·9)	0·6*	(0·5–0·8)
	$\chi^2_{(2)}=11·4$ ($p=0·003$)		$\chi^2_{(2)}=23·9$ ($p<0·001$)	
Dysthymic disorder	0·5*	(0·3–0·8)	0·8	(0·6–1·1)
Major depression	0·7*	(0·5–0·9)	0·6*	(0·4–0·7)
Bipolar disorder	1	(0·7–1·6)	1·2	(0·9–1·5)
III. Impulse disorders				
Any impulse disorder	0·8	0·6–1·1)	0·8	(0·6–1·1)
	$\chi^2_{(3)}=9·2$ ($p=0·027$)		$\chi^2_{(3)}=3·0$ ($p=0·397$)	
Intermittent explosive disorder	1	(0·7–1·5)	—	
Conduct disorder	0·9	(0·7–1·3)	—	
Oppositional defiant disorder	0·6*	(0·4–0·9)	—	
Attention-deficit/hyperactivity disorder	0·6*	(0·4–1·0)	—	
IV. Substance disorders				
Any substance disorder	1	(0·7–1·3)	0·7*	(0·6–0·9)
	$\chi^2_{(1)}=0·2$ ($p=0·642$)		$\chi^2_{(1)}=1·5$ ($p=0·217$)	
V. Any disorder	0·8*	(0·6–1·0)	0·7*	(0·6–0·8)

OR, Odds ratio; CI, confidence interval.

[a] OR estimated in discrete time survival models controlling for sex and age. Non-Hispanic Whites are the reference group. χ^2 represent tests for variation in difference (OR) between minority group and non-Hispanic Whites across individual disorders within each class of disorders.

* Significant difference between minority group and non-Hispanic Whites at $p=0·05$ level, two-sided test.

instance in which a minority group had significantly higher lifetime prevalence relative to non-Hispanic Whites: non-Hispanic Blacks had significantly higher lifetime prevalence of bipolar disorder.

Lifetime risk

Comparisons of lifetime risk, estimated in survival models that take into account differences in the age distribution across race-ethnic groups, show an even stronger pattern of lower risk for disorders among minorities than was indicated by comparisons of lifetime prevalence (Table 3). Both Hispanics and non-Hispanic Blacks had significantly lower lifetime risk for mood disorders, anxiety disorders and for any psychiatric disorder, and non-Hispanic Blacks had lower lifetime risk for substance use disorders. Variation in race-ethnic differences across the individual disorders within a class was significant in all but three cases: impulse control disorders among non-Hispanic Blacks, and substance use disorders among both minority groups. Both groups had lower lifetime risk than non-Hispanic Whites for depression, GAD and social phobia. There were also group specific differences: compared to non-Hispanic Whites, Hispanics had lower lifetime risk for dysthymia, oppositional defiant disorder, and ADHD, while non-Hispanic Blacks had lower lifetime risk for substance use disorders and panic disorder.

Variations across age of onset

Race-ethnic differences did not vary significantly between early and late onset disorders ($\chi^2_1=0·1$–$2·4$, $p=0·800$–$0·121$), with one exception. Non-Hispanic Blacks had significantly lower risk for early-onset (prior to age 10) impulse control disorders, but not for late-onset

Table 4. *Variations in race-ethnic differences in lifetime risk of DSM-IV disorders by cohort*[a]

	Younger cohort[b]				Older cohort			
	Hispanic		Non-Hispanic Black		Hispanic		Non-Hispanic Black	
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Anxiety disorder	0·6	(0·5–0·9)	—		1·0	(0·7–1·6)	—	
Mood disorder	0·7	(0·6–0·9)	—		1·1	(0·7–1·6)	—	
Impulse control disorder	0·7	(0·5–1·0)	—		1·6	(0·9–3·0)	—	
Substance use disorder	0·8	(0·6–1·2)	0·5	(0·4–0·7)	1·5	(1·1–2·0)	1·1	(0·8–1·6)

OR, Odds ratio; CI, confidence interval.

[a] Strata specific odds ratios presented for those models where variation in the OR across cohorts was statistically significant. See text for significance tests. Estimates derived from discrete time survival models with controls for age and sex.

[b] Younger cohort includes those who were 43 years of age or younger at the time of interview. Older cohort includes all others.

impulse control disorders [$\chi^2_1 = 4{\cdot}5$, $p = 0{\cdot}034$: OR (early-onset) 0·7, 95% CI 0·5–1·0; OR (late onset) = 1·0, 95% CI 0·7–1·5].

Variations across cohorts

Analysis of cohort variation in race-ethnic differences revealed that the lower lifetime risk among Hispanics is attributable entirely to the younger cohort, i.e. those aged 43 or younger (Table 4). Significant variation was found for each of the four classes of disorder ($\chi^2_1 =$ 4·3–7·2, $p = 0{\cdot}039$–$0{\cdot}007$). Specifically, in the older cohort, there were no significant differences in risk for anxiety, mood and impulse control disorders between Hispanics and non-Hispanic Whites, but in the younger cohort Hispanics had significantly lower risk than non-Hispanic Whites for each of these classes of disorder. Results for substance use disorders showed a trend in the same direction: in the older cohort Hispanics had significantly *higher* lifetime risk, whereas in the younger cohort there was no significant difference between Hispanics and non-Hispanic Whites.

In contrast with variation across cohorts for Hispanics, for non-Hispanic Blacks lower risk for disorders was constant across cohorts for anxiety, mood, and impulse control disorders ($\chi^2_1 = 0{\cdot}3$–$1{\cdot}8$, $p = 0{\cdot}598$–$0{\cdot}179$). An exception to this pattern was risk for substance use disorders, for which lower lifetime risk was limited to the younger cohort ($\chi^2_1 = 10{\cdot}9$, $p = 0{\cdot}001$).

Because race-ethnic differences might vary simultaneously by cohort and age of onset, we tested this possibility using three-way interactions between race-ethnicity, cohort and age of onset but found no significant interaction in any model ($\chi^2_1 \leqslant 0{\cdot}01$–$1{\cdot}0$, $p = 0{\cdot}982$–$0{\cdot}313$).

Variations across parental and respondent education

Lower lifetime risk among both minority groups was most pronounced at lower levels of education (Table 5). For Hispanics, there was significant variation in the odds ratio for three of the four classes of disorder: parental education for substance use disorders ($\chi^2_3 = 0{\cdot}6$, $p = 0{\cdot}022$) and impulse control disorders ($\chi^2_3 = 9{\cdot}3$, $p =$ $0{\cdot}026$) and respondent's education for mood disorders ($\chi^2_5 = 18{\cdot}6$, $p = 0{\cdot}002$). In each instance, lower lifetime risk in Hispanics relative to non-Hispanic Whites was restricted to lower education levels. For non-Hispanic Blacks there was significant variation across respondent education for impulse control disorders ($\chi^2_5 = 21{\cdot}3$, $p = 0{\cdot}001$) with the same trend as observed in Hispanics. No other interactions were detected for parental education ($\chi^2_3 = 1{\cdot}7$–$6{\cdot}3$, $p = 0{\cdot}645$–$0{\cdot}098$) or respondent education ($\chi^2_5 = 2{\cdot}0$–$10{\cdot}4$, $p = 0{\cdot}849$–$0{\cdot}064$).

DISCUSSION

Consistent with previous national surveys in the USA, data from the NCS-R indicate that Hispanics and non-Hispanic Blacks have lower lifetime risk of psychiatric disorders than non-Hispanic Whites (Breslau *et al.* 2005). This finding suggests the need to investigate potential protective factors that might explain the advantage that race-ethnic minority groups enjoy despite the social disadvantages they experience. Ethnic identification (Herd & Grube, 1996; Mossakowski, 2003) and religious participation (Wallace & Forman, 1998; Varon & Riley, 1999; Ellison *et al.* 2001; Lee & Newberg,

Table 5. *Variations in race-ethnic differences in lifetime risk across respondent education and parental education*[a]

	OR (95% CI)
I. Mood disorders	
Respondent education[b]	Hispanics *v.* Whites
Student < HS	0·6 (0·5–0·8)
Student > HS	1·2 (0·6–2·4)
Less than HS	0·4 (0·2–0·9)
HS graduate	0·9 (0·6–1·5)
13–15 years	1·5 (0·8–2·9)
16+ years	1·5 (0·8–2·6)
II. Substance use disorders	
Parental education	
< HS	0·8 (0·5–1·2)
HS	1·1 (0·5–2·1)
13–15 years	2·3 (0·9–6·0)
16+ years	1·1 (0·6–2·0)
III. Impulse control disorders	
Parental education	
< HS	0·5 (0·3–0·7)
HS	0·8 (0·4–1·6)
13–15 years	1·2 (0·6–2·4)
16+ years	1·3 (0·8–2·3)
Respondent education	Blacks *v.* Whites
Student < HS	0·7 (0·5–1·0)
Student > HS	2·7 (1·2–6·0)
Less than HS	0·5 (0·2–1·6)
HS graduate	0·8 (0·3–2·3)
13–15 years	2·1 (0·7–6·4)
16+ years	3·6 (0·8–15·3)

OR, Odds ratio; CI, confidence interval; HS, High School.

[a] Strata-specific OR shown where interactions between education and race-ethnicity were significant. See text for significance tests. Estimates derived from discrete time survival models with controls for age and sex.

[b] Respondent education is coded as a time-varying covariate as explained in the text.

2005) have been suggested as protective factors that might explain lower lifetime risk for disorder among race-ethnic minorities. However, cultural and social factors that might be related to risk for psychiatric disorder are complex and have undergone significant changes in recent decades.

As noted in the introduction, our goal in this analysis was to advance the examination of suspected protective factors by investigating variation in race-ethnic differences across specific disorders, age of onset of disorders and subgroups of the population defined by cohort and education. The key findings of this analysis are:

(1) Lower lifetime risk among race-ethnic minority groups relative to non-Hispanic Whites is attributable to a small number of common disorders.
(2) Lower lifetime risk among both Hispanics and non-Hispanic Blacks relative to non-Hispanic Whites begins in childhood (i.e. prior to age 10).
(3) Differences in lifetime risk favoring minorities are more pervasive in the younger cohort (i.e. born *circa* 1958 or more recently).
(4) Variation in race-ethnic differences across levels of education is sporadic and, where it occurs, it shows that lower lifetime risk in members of minority groups is more pronounced at lower levels of education (i.e. parent or respondent had less than high school education).

Lower risk for mood and anxiety disorders among both minority groups was accounted for by five disorders: major depression, dysthymia, GAD, social phobia, and panic disorder. These disorders fall within a set of 'internalizing' disorders, which cluster together in factor analyses, suggesting that they may share etiologic pathways (Krueger, 1999; Kendler *et al.* 2003). The finding that these disorders vary together in lifetime risk across race-ethnic groups suggests the presence of common protective factors across disorders for both minority groups. This possibility would be further supported if future analyses show that patterns of co-morbidity among these disorders are similar across race-ethnic groups. Substance use and impulse control disorders, which comprise the externalizing factor identified in analyses of co-morbidity, do not differ consistently across race-ethnic groups, suggesting that the explanation for the observed differences in these disorders is more complex.

The finding that race-ethnic differences emerge in childhood strongly suggests that the search for causes should focus on factors present in the early life environment. A focus on childhood is a particularly important consideration for studies of religious participation and ethnic identification. These factors are measured in adults, but may also reflect the cultural contexts in which children are socialized, rather than the direct effect of religious participation on adult experience.

We found that for Hispanics, differences in lifetime risk relative to non-Hispanic Whites varied significantly across birth cohorts for all classes of disorder, and that the phenomenon

of lower lifetime risk of disorders was specific to the younger cohort. For non-Hispanic Blacks, a similar trend was found for substance use disorders. It is unclear whether the protective factors suggested in the literature, ethnic identification and religious participation, have followed a similar historical trajectory. There is no evidence that relative rates of religious *participation* across groups have changed, but there is evidence that religious *affiliations* within both minority groups have shifted toward conservative protestant groups that tend to promote abstention from alcohol (Hunt, 1999; Sherkat, 2002). The role of religion could be further elucidated if future studies examine whether observed historical changes in race-ethnic differences in risk of disorder are related to contemporaneous changes in specific aspects of religiosity. Future studies should also examine historical changes in patterns of immigration (Portes & Zhou, 2003), educational achievement (Kao & Thompson, 2003) and income (Levy, 1998) across minority groups.

Analysis of variation in race-ethnic differences across levels of SES, using parental and respondent education as indicators, contradict the 'double jeopardy' theory, which would predict that low SES minorities would have the highest levels of risk because of the combined effect of socio-economic and race-ethnic disadvantage. Where variation in race-ethnic differences in lifetime risk was found, the pattern was consistent with predictions of the 'declining returns' theory, which predicts that minority groups enjoy fewer of the health improvements that come with higher SES. This pattern is consistent with research on Hispanics which found that Hispanic enclave communities offer social resources to their members that may have a salutary impact on health (Sanders, 2002; Portes & Zhou, 2003; Eschbach *et al.* 2004).

Limitations

The findings should be interpreted in light of several limitations. First, the assessment of lifetime risk for psychiatric disorders depends on the accuracy of lifetime recall of psychiatric symptoms. As mentioned in the Method section above, the WMH-CIDI included question sequences found in experimental research to improve recall of life events in survey research. There is no evidence that recall of psychiatric symptoms is differential across race-ethnic groups.

Second, differential non-response across race-ethnic groups may have led to underestimation of lifetime disorders among minority groups (USA DHHS, 2001). To minimize the impact of non-response bias in this survey, a non-respondent survey was carried out in which a representative subsample of initial non-respondents was administered a brief telephone survey that collected screening information about anxiety, mood, impulse-control, and substance disorders (Kessler *et al.* 2004*b*). Results were used to weight the main survey data to adjust for any differential under-representation of disorders on the basis of race-ethnicity as well as other sociodemographic factors. In addition, missing data on parental education, a problem that was more common for minorities than for non-Hispanic Whites, were imputed based on information on parental occupation. Results did not change meaningfully when observations with missing values were removed from the analysis, but it remains possible that residual bias remains that might limit the generalizability of the results reported here.

Third, cultural differences may lead members of different race-ethnic groups to respond differently to the same survey questions regarding their psychiatric history despite similar levels of morbidity (Rogler, 1999; Rogler *et al.* 2001). Psychometric studies of differential item functioning examining distress scales have found some differences along these lines across race-ethnic groups. However, these differences generally overestimated levels of distress of minority groups and were attributable to items related to positive mental health (Iwata & Buka, 2002). Positive mental health items are not used to assess disorders in this study. Reporting of psychiatric symptoms might also be influenced by the race-ethnicity of the interviewers. Since the finding of lower lifetime risk among minority groups is consistent across studies using different structured diagnostic instruments, e.g. the Diagnostic Interview Schedule (DIS) (Somervell *et al.* 1989; Weissman *et al.* 1991), the University of Michigan (UM)-CIDI (Kessler *et al.* 1994) and the WMH-CIDI (Kessler *et al.* 2005), methodological studies of differential validity across race-ethnic groups

should focus on methods shared by these instruments.

We have focused on race-ethnic differences in lifetime risk because of the counterintuitive finding of lower lifetime risk among disadvantaged groups. However, this finding of lower lifetime risk does not imply that disadvantaged groups suffer a lower overall burden of psychiatric morbidity. Our previous work suggests that the advantage of reduced lifetime risk is offset by the disadvantage of an increased risk for persistence among members of minority groups who become ill (Breslau *et al*. 2005). Assessment of the aggregate public health implications of race-ethnic differences in psychiatric morbidity, therefore, will require comparisons that consider multiple aspects of the course of disorders, including persistence as well as severity and disability.

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DECLARATION OF INTEREST

None.

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