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Pharmaceutical Side Effects and Mental Health Paradoxes among Racial-Ethnic Minorities

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Abstract

Sociologists have long struggled to explain the minority mental health paradox: that racial-ethnic minorities often report better mental health than non-Hispanic whites despite social environments that seem less conducive to well-being. Using data from the 2008–2013 Medical Expenditure Panel Survey (MEPS), this study provides a partial explanation for the paradox rooted in a very different disparity. Evidence from MEPS indicates that non-Hispanic whites consume more pharmaceuticals than racial-ethnic minorities for a wide variety of medical conditions. Moreover, non-Hispanic whites consume more pharmaceuticals that although effective in treating their focal indication, include depression or suicide as a side effect. In models that adjust for the use of such medications, the minority advantage in significant distress is reduced, in some instances to statistical nonsignificance. Although a significant black and Hispanic advantage in a continuous measure of distress remains, the magnitude of the difference is reduced considerably. The relationship between the use of medications with suicide as a side effect and significant distress is especially large, exceeding, for instance, the relationship between poverty and significant distress. For some minority groups, the less frequent use of such medications is driven by better health (as in the case of Asians), whereas for others, it reflects a treatment disparity (as in the case of blacks), although the consequences for the mental health paradox are the same. The implications of the results are discussed, especially with respect to the neglect of psychological side effects in the treatment of physical disease as well as the problem of multiple morbidities.

Keywords

comorbidity, disparities, pharmaceuticals, psychological distress, race-ethnicity

The mental health of racial-ethnic minorities sometimes presents a paradox. Despite expectations to the contrary, minorities report mental health that is either somewhat better or no worse than that of the non-Hispanic white population (Williams and Earl 2007). The 12-month prevalence of most psychiatric disorders tends to be lower among minorities (Breslau et al. 2005; Miranda et al. 2008; Vilsaint et al. 2019), including for depression (Hasin et al. 2018). Other patterns are consistent with this observation. Death rates from suicide among non-Hispanic whites are two to three times higher than among blacks, Asians, and Latinos (Miranda et al. 2008). Once adjustments for socioeconomic status are included, African Americans almost always report better mental health on dimensional measures of depression (Barnes and Bates 2017). Hispanics, too, tend to report a lower prevalence of psychiatric disorders (Breslau et al. 2005), and among Asian populations, the prevalence of psychiatric disorders is especially low (Takeuchi et al. 2007). To be sure, relative to whites, African Americans sometimes report worse mental health on cognitive measures of

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well-being, such as life satisfaction (Hughes and Thomas 1998), though they usually have a lower risk of clinically significant distress (Breslau et al. 2005; Riolo et al. 2005).

Differences of this sort confound easy explanations. For one, the apparent racial-ethnic advantage in mental health does not align with racial-ethnic disparities in physical health. On most indicators, African Americans report worse physical health than non-Hispanic whites (Williams 2005). The Hispanic population between the ages of 15 and 34 reports worse health, though overall the health of Hispanics is somewhat better than whites (Williams 2005). Even when racial-ethnic minorities maintain a clear relative advantage in health, as with the Asian population (Acciai, Noah, and Firebaugh 2015), racial-ethnic minorities are often exposed to more stress, especially when considering race-specific stressors (Williams 2018). Black and Hispanic populations, for instance, report higher levels of discrimination along with lower socioeconomic status (Kessler, Mickelson, and Williams 1999; Pérez, Fortuna, and Alegría 2008). Asian populations, too, report high levels of discrimination, and some specific Asian populations (e.g., Hmong and Burmese) also report lower socioeconomic status than whites (Yip, Gee, and Takeuchi 2008).

In light of these facts, sociologists interested in explaining the racial-ethnic minority advantage have focused less on stress exposure and more on protective factors. These efforts, however, have not been entirely successful. Research has uncovered some important protective factors, to be sure, but these factors are not always relevant or suitable to explaining the minority paradox. They either apply only to coping with certain forms of stress, have highly complex and contingent associations with mental health, or are not uniformly more common among racial-ethnic minorities (Ellison, Musick, and Henderson 2008; Jackson, Knight, and Rafferty 2010; Odom, Garrett-Peters, and Vernon-Feagans 2014; Phinney and Haas 2003). As an alternative explanation for the paradox, some scholars have turned to measurement biases in common measures of mental health (Sue et al. 2012). Yet here, too, such explanations are limited, at least for purposes of explaining the paradox. Measurement differences between groups are generally not large or consistent enough to explain the paradox altogether (Alegría and McGuire 2003). Mixed findings of this sort have prompted some to argue that the literature on minority mental health, no matter how well developed, still has more questions than answers (Williams and Earl 2007).

This study seeks to push the literature in a different direction and provides a different answer for the paradox. In particular, this study explores whether the paradox is a function of the side effects of medications frequently prescribed for common physical conditions. This study presents evidence that much of the paradox in mental health can be explained by the more frequent use of medications that have either depression or suicide as a side effect among non-Hispanic whites. In this way, this study argues that the resolution of one paradox might rest on another: The elusive protective factor apparent among racial-ethnic minorities might be, in part, in their relatively low levels of medical intervention.

BACKGROUND

Patterns in racial-ethnic minority mental health are confounding in large part because most sociological theory points to the many ways that minorities are disadvantaged (Williams 2018). For good reason, most explanations for minority mental health begin with socioeconomic status (Williams et al. 1997). At least in the context of physical health, socioeconomic status explains some though not all of the black disadvantage, including with respect to life expectancy (Geruso 2012; Hayward et al. 2000). Socioeconomic status also affects mental health, largely through chronic stress (Thoits 2010), but given the relatively low socioeconomic status of blacks, socioeconomic status only suppresses the black–white difference in depression. For some dimensional measures of depression specifically, African Americans report less depression even before adjustments for socioeconomic status, though such adjustments further inflate the advantage (Barnes and Bates 2017). A similar pattern is found for other groups and is sometimes even more pronounced. The Mexican immigrant disadvantage in socioeconomic status, for instance, is quite large even as the 12-month prevalence of most psychiatric disorders is very low (Vega et al. 1998; Vilsaint et al. 2019).

As research on minority mental health has evolved, it has increasingly focused on uncovering other sources of stress. This focus has done much to forefront previously neglected race-based stressors (Williams 2018). Perhaps the most prominent of such stressors is discrimination, which is both common among racial-ethnic minorities and consequential for mental health. Blacks, Hispanics, and Asians all report more discrimination than the white population, including major lifetime experiences

with unfair treatment, such as losing a job, as well as everyday experiences of discrimination and disrespect, such as inferior service (American Psychological Association 2016). Discrimination has, in turn, been related to subsequent psychological distress and an increased risk of major psychiatric disorders (Brown et al. 2000; Lewis, Cogburn, and Williams 2015; Williams 2018).

Although this research is revealing, it only deepens the paradox of minority mental health. In effect, it shows that once stress is measured more comprehensively, racial-ethnic minorities report even more stress than the white population. In this light, some scholars have shifted their focus from documenting risks to uncovering protective factors among minorities. A variety of factors have been put forward. Much of this work has focused on social support and religious involvement, and both are demonstrably important for coping with stress (Brody et al. 2006; Ellison et al. 2008). In a similar vein, though positing something more novel, Keyes (2009) pointed to the possibility that blacks are better able to create positive meaning from negative events. A somewhat smaller literature has taken a different tact and attempted to identify risk factors that are more common among whites. For instance, some studies point to inflexible coping styles among non-Hispanic whites, including a belief in meritocracy, which can lead to more self-blame in situations of low socioeconomic status (Kwate and Meyer 2010). Other research has explored the ways in which whiteness can sometimes diminish health (Malat, Mayorga-Gallo, and Williams 2018). Yet even as research identifies these racial-ethnic differences, it generally does not address the mental health paradox directly. Moreover, research rarely finds coping resources that are uniformly more common among racial-ethnic minorities or significant risk factors that are plainly more frequent among whites. Although racial-ethnic minorities experience more discrimination, for instance, they are not necessarily better able to cope with that discrimination than non-Hispanic whites (Phinney and Haas 2003). Even among populations that expect discrimination, discrimination remains detrimental to their mental health (Kessler et al. 1999). Neither family relationships nor relationships of choice can explain the paradox, though both are important for well-being (Mouzon 2013, 2014). Although blacks report considerably more religious involvement, religion, too, fails to explain the paradox (Mouzon 2017). In this light, recent research has framed its findings in a different and perhaps narrower fashion, arguing only for a set of offsetting mechanisms, as when studies find evidence for higher self-esteem among blacks relative to whites but greater exposure to trauma (Louie and Wheaton 2019).

Explaining the minority mental health paradox requires something more: It requires thinking about risk factors that both are more common in the most advantaged population and have less contingent relationships with mental health. Very little research has focused on precisely this set of empirical issues, though a different and emerging literature could provide clues (Qato, Ozenberger, and Olfson 2018).

Treatment Disparities in Health Care

A very well-developed literature has focused on disparities in the receipt and quality of health care (Institute of Medicine 2003). African Americans tend to receive less medical care than whites, and when they do receive care, it tends to be of lower quality. Some of this difference is driven by access to care, including the availability of health insurance (Sohn 2017). Yet even when adjusting for health insurance, African Americans still report lower quality care. Consistent with this, some evidence links treatment disparities to physicians' implicit preference for white patients over patients of color (Sabin et al. 2009; van Ryn et al. 2011). The same pattern is found in prescription medications, the most common form of medical intervention. African Americans receive fewer prescription medications, and even among insured patients with the same condition, black and Hispanic patients use significantly fewer medications that white patients (Briesacher, Limcangco, and Gaskin 2003). The interpretation of this difference is not always consistent. In some instances, the difference reflects overtreatment on the part of non-Hispanic white patients, as in the case of the overuse of antibiotics (Goyal et al. 2017). Much of the evidence, though, points to significant undertreatment among minorities, including with respect to the use of new essential drugs (Wang et al. 2006) and the use of analgesics in the treatment of pain (Meghani, Byun, and Gallagher 2012). Pharmaceutical innovations, like medical innovations more generally, are usually given to the most advantaged patients first, producing disparities even as the medical armamentarium improves.

Treatment disparities of this sort have a number of consequences, including higher mortality among African American patients who are not adequately treated (Morris et al. 2010). Yet some treatment disparities might have the paradoxical effect of reducing the burden of depression among minority populations, who either take fewer medications

because they are prescribed fewer medications at a given level of illness (i.e., blacks and Hispanics) or take fewer medications because they have better health (i.e., Asians). Prescription drug use is, in general, very common, representing the average American's most routine medical treatment. In 2012, nearly 60% of adults reported the use of at least one prescription medication, and 15% reported using five or more (Kantor et al. 2015). Such medications are often highly effective in treating the condition they were designed to treat or in reducing a known risk factor. More directly relevant to mental health, though, evidence points to a significant increase in the use of medications with depression or suicide as a significant side effect (Qato et al. 2018). Indeed, at present, about 37% of U.S. adults take at least one such medication, and 15% take three or more (Qato et al. 2018). The same data show a significant increase in the risk of major depression associated with using such medications, especially when taking more than one (Qato et al. 2018).

To date, no study has attempted to link such medications to racial-ethnic differences in depression. It remains unclear whether side effects of this sort are relevant to understanding racial-ethnic differences in depression, both because racial-ethnic differences in the use of such medications are not well understood and because few studies have explored the implications of such medications for population mental health. The present study addresses these issues by using the Medical Expenditure Panel Survey (MEPS), a high-quality nationally representative survey useful for assessing medication use.

DATA AND METHODS

Data came from the 2008–2013 MEPS (see Cohen, Cohen, and Banthin 2009), including the file made available at the Integrated Public Use Microsample repository at the University of Minnesota (Blewett et al. 2018). The MEPS is a nationally representative probability survey of the civilian noninstitutionalized U.S. population, drawn from a subsample of households that participated in the prior year's National Health Interview Survey (NHIS). Both MEPS and NHIS include oversamples: Black, Hispanic, and low-income respondents are oversampled to improve the precision of estimates for these subgroups. The overall MEPS response rate (after taking into account the nonresponse rate from the NHIS) ranges from 46% to 71%. All the analyses presented here employed survey weights to account for oversampling and nonresponse.

The data are organized as annual files, though data collection occurred through a series of overlapping household panels. MEPS respondents enter the survey annually as members of a panel. A new panel is selected each year, and data are collected over five rounds in two calendar years. The data are then organized into annual files, each of which will include multiple panels. For each calendar year, data from interview rounds one, two, and three are included for individuals in their first of two years, and data from interview rounds three, four, and five are included for individuals in their second of two years. Collectively, respondents from the two overlapping panels in each year provide nationally representative estimates for that year. More information is available at https://meps.ahrq.gov/survey_comp/hc_data_collection.jsp. We began with 2008 because it was the first year to include comprehensive chronic disease indicators. Listwise deletion was used to eliminate a small number of missing cases, less than 3% for the dependent variable and an additional 1% across the set of remaining covariates.

Nonspecific Psychological Distress

The MEPS survey includes the Kessler K6, a well-validated instrument assessing nonspecific psychological distress (Kessler et al. 2003). The K6 was not fielded in every round: In each calendar year, respondents in the first year of their panel were given the K6 in the second round of interviews, and respondents in the final year of their panel were given the K6 in the fourth round. The K6 asks about the frequency of six symptoms in the past 30 days. Respondents were asked: "how often did you feel so sad that nothing could cheer you up; nervous; restless or fidgety; hopeless; that everything was an effort; and worthless," with response categories ranging from none of the time (0) to all of the time (4). The six items were combined to create a summary measure with scores ranging from 0 to 24. In addition to a continuous score, some models employed a categorical measure of significant distress, based on a threshold score of 13 or higher, as used in previous studies (Reeves et al. 2011).

Medication Use

Among the most valuable features of MEPS is its collection of detailed medication use. MEPS includes the names of all the drugs taken by the respondent. Although MEPS collected information on drugs in every round, we did not include drugs reported in rounds after respondents reported their

mental health to ensure proper time ordering. Drug information was then linked to information on whether the specific drug taken by the respondent involved suicide or depression as a side effect (in separate categories). This linkage was based on procedures described in detail in Qato et al. (2018) and, in particular, on lists provided in an online appendix to that article. In that study, Qato and colleagues (2018) linked information on medications, as reported in the National Health and Nutritional Examination Study (NHANES), to information on adverse effects provided in Micromedex, a proprietary database created by Truven Health Analytics, which, in turn, is based on information provided by the U.S. Food and Drug Administration (Barrons 2004; Cheng et al. 2010). After using the information provided in Qato et al. (2018) we created two variables, the first corresponding to the number of drugs the respondent was taking with depression as a side effect and the second corresponding to the number of drugs the respondent was taking with suicide as a side effect. In each case, we divided the sum into four categories, consistent with the Qato et al. (2018) analysis of the NHANES: no drugs with such a side effect (the reference category), one drug, two drugs, or three or more drugs. In some of the descriptive information presented in our study, we refer to actual counts rather than categories. In addition, in some analyses, we removed antidepressants from the suicide side-effect count (a sensitivity test discussed in more detail shortly). The list of drugs included both prescription and over-the-counter medications, though most were prescription. The Qato et al. (2018) analysis was based on reports of prescription medications in NHANES, though some prescription medications have over-the-counter formulas.

Sociodemographic Variables

Race-ethnicity was divided into four groups: non-Hispanic white, non-Hispanic black, Hispanic, and Asian. Less than 2% of the sample consisted of an "other" race-ethnicity group, and because of their small numbers, they were dropped from the analysis. The models also included controls for the sex of the respondent, marital status (married; widowed, divorced, or separated; never married), age (18–to 39, 40–64, 65–79, and 80 and over), education (less than high school, high school or equivalent, college or higher), insurance status, and poverty (below 100% of the federal poverty line for household income). All the models also controlled for the year of the survey using a series of dummy variables.

Chronic Conditions

Many of the medications with either depression or suicide as a side effect are used in the treatment of chronic conditions (as opposed to prevention). To control for physical health comorbidities, which themselves could play a powerful role in depression, the models included controls for whether the respondent was ever told they had each of 11 conditions: angina pectoris, arthritis, asthma, coronary heart disease, diabetes, high cholesterol, emphysema, a heart attack, heart disease or other heart condition, hypertension, or a stroke. These conditions were entered into the regression model as separate covariates, and in some specifications, interactions among all pair-wise conditions were explored to assess the influence of comorbidity.

RESULTS

The results begin by describing basic differences in the use of medications with depression and suicide as side effects, as presented in Tables 1, 2, and 3. Table 1 presents basic demographic information, Table 2 presents drug use information, and Table 3 presents means for the distress measures arrayed over race-ethnicity and drug use. Table 1 shows, among other things, few significant differences in distress on average. There is no black-white or Hispanic-white paradox, though in basic multivariate models (see Table 3), a significant difference emerges. Table 2 shows large racial-ethnic differences in the use of medications with side effects. About 60% of non-Hispanic whites do not use medications with depression as a side effect, but this percentage is much higher among minorities, especially Asians and Hispanics. About 5% of whites use three or more such medications, relative to slightly over 4% of blacks, 1% of Asians, and 2% of Hispanics. The same pattern is found for drugs involving suicide as a side effect, though the racial-ethnic differences are, if anything, larger. About 72% of whites use no such medications, relative to 85% of blacks, 91% of Asians, and 87% of Hispanics. The white population uses three or more such medications at levels that are at least two-to-one relative to racial-ethnic minorities. These differences are not driven by any particular class of medication. For most specific subclasses, the same pattern is evident, with the notable exception of analgesics with depression as a side effect, where blacks are slightly more likely to take such medications than non-Hispanic whites. The single largest contributor to the overall count is antidepressants with suicide as a side effect. These medications are

Table 1. Descriptive Statistics for Socioeconomic and Demographic Variables.

	Race and Ethnicity			
	Non-Hispanic White	Non-Hispanic Black	Non-Hispanic Asian	Hispanic
Significant distress (K6 ≥ 13) (%)	4.95	5.36	3.09***	5.35
K6 score (continuous, minimum = 0, maximum = 24)	3.38	3.29	2.87***	3.34
Female (%)	51.60	54.94***	53.16*	49.30***
Age (%)				
18–44	34.13	43.71***	45.09***	53.68***
45–64	45.13	43.67*	41.90**	37.48***
65–79	15.18	9.82***	10.14***	6.93***
80+	5.56	2.80***	2.87***	1.90***
Marital status (%)				
Married	57.96	34.19***	62.02**	48.82***
Widowed, divorced, separated	20.27	23.44***	11.54***	16.45***
Never married	21.78	42.37***	26.44***	34.73***
Educational attainment (%)				
Less than high school	9.90	18.38***	13.03***	38.30***
High school graduate	29.79	34.02***	16.36***	27.44***
College or above	60.31	47.60***	70.61***	34.26***
Less than 100% of poverty guideline (%)	8.16	20.70***	9.73*	19.38***
Has any type of insurance (%)	89.28	81.50***	85.30***	63.81***
Number of observations	58,508	24,899	9,796	34,055

Note: All estimates are weighted using survey weights. Data from Medical Expenditure Panel Survey 2008–2013.
*$p < .05$, **$p < .01$, ***$p < .001$ (for test of significant differences between non-Hispanic whites and other racial/ethnic groups).

taken by nearly 16% of non-Hispanic whites but fewer than 7% of racial-ethnic minorities.

Table 2 also presents the prevalence of specific chronic conditions, once again arrayed over race-ethnicity. In this case, the racial-ethnic differences are somewhat different. In general, many chronic conditions are more common among non-Hispanic whites than minorities, though there are notable exceptions. Blacks report a considerably higher prevalence of diabetes, hypertension, stroke, and asthma. For many chronic conditions, Asians and Hispanics report a considerably lower prevalence than non-Hispanic whites.

Table 3 presents means for the distress variables arrayed by race-ethnicity and drug use. It also shows two patterns in particular that set the stage for the multivariate models. In general, the use of medications with side effects increases the prevalence of significant distress and the K6 score. Furthermore, racial-ethnic differences in distress are different within the rows denoting the use of no drugs with side effects. Within this group, the prevalence of significant distress is generally higher among blacks than among whites (the difference is statistically significant for three of four comparisons). Similarly, the prevalence of distress is higher among Hispanics (for three of four comparisons).

The regression models presented in Table 4 explore the consequences of such medications for the minority advantage in mental health. Pairs of models are presented for each model specification, the first using linear regression for the K6 summary score and the second using logit regression for the categorical coding of the K6. The use of control variables is critical both for understanding the magnitude of the paradox and for contextualizing the influence of pharmaceutical use. The baseline model controls for socioeconomic status (as well as other variables) and shows a minority advantage, both with respect to a dimensional measure of distress and a categorical one. The magnitude of the difference is large, with minorities showing at least a 20% reduction in the odds of reporting significant distress. The second pair of models inserts controls

Table 2. Descriptive Statistics for Medications with Depression or Suicide as a Potential Side Effect.

	Race and Ethnicity			
	Non-Hispanic White	Non-Hispanic Black	Non-Hispanic Asian	Hispanic
Taking any drugs with depression side effects (%)				
None	60.14	66.85***	79.44***	75.52***
1 drug	25.20	20.80***	15.43***	16.77***
2 drugs	9.79	8.10***	3.84***	5.48***
3+ drugs	4.88	4.25**	1.29***	2.23***
Taking any drugs with depression side effects by subclass (%)				
Analgesics	14.62	15.52*	5.82***	11.05***
Antihypertensives	11.53	9.79***	6.45***	5.46***
Corticosteroids	5.50	3.63***	2.03***	2.48***
Gastrointestinal agents	9.20	6.98***	4.13***	5.56***
Hormones/hormone modifiers	9.05	4.89***	4.47***	4.09***
Respiratory agents	.54	.65	.24***	.48
Others	5.18	4.63*	1.94***	2.65***
Taking drugs with suicide side effects (%)				
None	72.45	84.98***	91.05***	86.82***
1 drug	16.38	9.57***	6.42***	8.49***
2 drugs	6.51	3.36***	1.50***	2.65***
3+ drugs	4.66	2.09***	1.02***	2.04***
Taking any drugs with suicide side effects by subclass (%)				
Analgesics	2.67	2.51	.72***	1.83***
Anticonvulsants	7.96	5.00***	2.61***	3.86***
Anxiolytics, hypnotics, sedatives	7.28	2.90***	1.87***	3.27***
Antidepressants	15.85	6.17***	3.34***	5.98***
Gastrointestinal agents	.38	.43	.22*	.31
Hormones/hormone modifiers	1.04	.47***	.55***	.38***
Respiratory agents	1.74	1.18***	.92***	.88***
Others	5.65	3.61***	2.19***	3.21***
Chronic conditions (%)				
Angina pectoris	3.04	2.14***	1.43***	1.58***
Arthritis/gout/lupus/fibromyalgia	28.94	24.27***	12.04***	13.88***
Asthma	9.66	11.07***	5.42***	6.76***
Coronary heart disease	6.52	4.35***	2.84***	3.62***
High cholesterol	33.90	26.25***	27.35***	23.80***
Diabetes	8.53	12.13***	8.88	9.32*
Emphysema	2.82	1.30***	.49***	.55***
Heart attack	4.30	3.14***	1.56***	2.01***
Heart disease (exclude coronary heart disease, angina, heart attack)	13.06	8.62***	4.27***	4.53***
Hypertension	34.02	40.67***	24.17***	23.59***
Stroke	3.85	4.53**	1.57***	1.95***
Number of observations	58,508	24,899	9,796	34,055

Note: All estimates are weighted using survey weights. Data from Medical Expenditure Panel Survey 2008–2013.
*$p < .05$, **$p < .01$, ***$p < .001$ (for test of significant differences between non-Hispanic whites and other racial-ethnic groups).

Table 3. Descriptive Statistics for Nonspecific Psychological Distress by Race-Ethnicity and the Use of Medications with Depression and Suicide Side Effects.

	Race and Ethnicity			
	Non-Hispanic White	Non-Hispanic Black	Non-Hispanic Asian	Hispanic
Significant distress (K6 ≥ 13), % (*SE*)				
No drugs with depression side effects	3.26 (.13)	3.96 (.20)**	2.29 (.23)**	3.80 (.17)**
1 drug with depression side effects	5.46 (.23)	6.05 (.33)	4.49 (.66)	7.77 (.59)***
2 drugs with depression side effects	9.33 (.45)	10.03 (.73)	10.06 (1.86)	12.17 (1.19)*
3 drugs with depression side effects	14.35 (.78)	15.16 (1.30)	15.00 (3.75)	22.93 (2.60)***
No drugs with suicide side effects	2.46 (.11)	3.55 (.17)***	2.20 (.22)	3.21 (.15)***
1 drug with suicide side effects	6.52 (.27)	10.21 (.78)***	9.05 (1.34)*	12.39 (.90)***
2 drugs with suicide side effects	12.39 (.69)	19.45 (1.62)***	14.79 (3.75)	25.41 (1.80)***
3 drugs with suicide side effects	27.67 (1.16)	34.04 (2.27)**	28.15 (6.27)	41.10 (2.60)***
K6 score, mean (*SE*)				
No drugs with depression side effects	2.86 (.03)	2.81 (.05)	2.62 (.07)**	2.87 (.05)
1 drug with depression side effects	3.62 (.05)	3.73 (.09)	3.40 (.13)	4.27 (.10)***
2 drugs with depression side effects	4.62 (.09)	4.57 (.14)	5.02 (.37)	5.36 (.21)**
3 drugs with depression side effects	5.99 (.14)	6.30 (.22)	5.68 (.65)	7.16 (.39)**
No drugs with suicide side effects	2.63 (.03)	2.79 (.04)**	2.61 (.06)	2.80 (.05)**
1 drug with suicide side effects	4.16 (.06)	4.89 (.13)***	4.74 (.28)*	5.52 (.16)***
2 drugs with suicide side effects	5.90 (.11)	7.12 (.29)***	6.37 (.56)	8.14 (.30)***
3 drugs with suicide side effects	8.74 (.17)	9.97 (.32)**	9.06 (1.00)	10.91 (.39)***
Number of observations	58,508	24,899	9,796	34,055

Note: All statistics are weighted using survey weights. Data from Medical Expenditure Panel Survey 2008–2013.
*$p < .05$, **$p < .01$, ***$p < .001$ denote whether the estimates are significantly different between non-Hispanic whites and other racial-ethnic groups.

for each of the chronic health conditions. As expected, this adjustment alters the magnitude of the racial-ethnic differences, though in ways specific to each racial-ethnic group. The black-white difference is suppressed somewhat by physical health, such that the black advantage would be even larger—about 7% larger for the K6 score—were it not for a black disadvantage in physical health. For both Hispanics and Asians, however, the inclusion of controls for chronic conditions explains some of the apparent mental health advantage. The Asian advantage for the K6 is reduced by about 67%, whereas the Hispanic advantage is reduced by 46%. The difference between Hispanics and whites in significant distress, meanwhile, is reduced to statistical nonsignificance.

The next two models introduce controls for the use of pharmaceuticals involving depression and suicide as side-effects. In the first pair of models (Model 3), the count of medicines involving suicide includes antidepressants, but in the second pair (Model 4), the count excludes them. The inclusion of the side-effect variables explains what remains of much of the racial-ethnic advantage. Although the black-white difference in the K6 summary remains significant and shows a black advantage, it is reduced a great deal, by 60%, relative to Model 1. The difference in significant distress, meanwhile, is reduced to statistical nonsignificance. A similar pattern is found for the Asian-white difference, and in fact, the Asian advantage in the K6 turns into a significant Asian disadvantage. Similarly, the Hispanic advantage with respect to significant distress in Model 1 is reversed in Model 4, turning into a significant Hispanic disadvantage. Formal tests of mediation using structural equations and the continued K6 outcome provide further evidence for mediation. In Model 3, the indirect effect of black was –.359 ($p < .001$), and the direct effect was –.214 ($p < .001$). The parallel effects for Asians and Hispanics were, respectively, –.375 ($p < .001$) and .235 ($p < .001$) and –.222 ($p < .001$) and –.031 ($p = .583$). In Model 4, similar patterns of significance among direct and indirect effects were found

Table 4. Linear Regression and Logistic Regression Analysis of K6 Score and Significant Distress (K6 ≥ 13).

	Model 1		Model 2		Model 3		Model 4	
	(Basic Controls)		(Model 1 + Chronic Conditions)		(Model 2 + Drugs with Depression/ Suicide Side Effects)		(Model 2 + Drugs with Depression/Suicide Side Effects, Excluding Antidepressants)	
	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)
	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)
Race and ethnicity (non-Hispanic white is reference)								
Non-Hispanic black	−.534*** (.057)	.779*** (.039)	−.573*** (.057)	.754*** (.040)	−.214*** (.052)	1.019 (.059)	−.362*** (.053)	.900 (.050)
Non-Hispanic Asian	−.424*** (.068)	.654*** (.056)	−.140* (.066)	.822* (.069)	.235*** (.065)	1.158 (.099)	.103 (.065)	1.020 (.087)
Hispanic	−.468*** (.066)	.795*** (.043)	−.254*** (.061)	.940 (.050)	−.031 (.057)	1.139* (.062)	−.122* (.058)	1.051 (.056)
Whether taking drugs with depression side effects (none is reference)								
1 drug					.266*** (.033)	1.166*** (.050)	.348*** (.034)	1.254*** (.053)
2 drugs					.543*** (.061)	1.358*** (.079)	.725*** (.063)	1.535*** (.090)
3+ drugs					.860*** (.103)	1.363*** (.100)	1.156*** (.110)	1.590*** (.123)
Whether taking drugs with suicide side effects (none is reference)								
1 drug					1.381*** (.048)	2.600*** (.119)		
2 drugs					2.840*** (.093)	4.502*** (.280)		
3+ drugs					5.258*** (.141)	10.295*** (.735)		
Whether taking drugs with suicide side effects (exclude antidepressant drugs; none is reference)								
1 drug							1.447*** (.055)	2.386*** (.105)
2 drugs							2.625*** (.114)	3.832*** (.267)
3+ drugs							5.081*** (.186)	7.916*** (.640)
Female (0/1)	.473*** (.032)	1.237*** (.044)	.451*** (.032)	1.200*** (.046)	.160*** (.028)	.974 (.037)	.258*** (.029)	1.051 (.040)
Age								
18–39 (reference)								
40–64	.345*** (.045)	1.582*** (.081)	−.309*** (.043)	.932 (.055)	−.365*** (.039)	.873* (.050)	−.351*** (.040)	.884* (.051)
64–79	−.272*** (.062)	.968 (.075)	−1.825*** (.069)	.346*** (.032)	−1.679*** (.066)	.380*** (.035)	−1.762*** (.068)	.363*** (.034)
80+	.015 (.101)	.974 (.089)	−1.754*** (.102)	.334*** (.032)	−1.435*** (.098)	.423*** (.042)	−1.653*** (.102)	.371*** (.036)
Marital status (married is reference)								
Widowed, divorced, separated	1.035*** (.056)	1.944*** (.099)	.812*** (.055)	1.692*** (.088)	.657*** (.050)	1.552*** (.081)	.725*** (.053)	1.621*** (.085)
Never married	.438*** (.050)	1.371*** (.077)	.499*** (.048)	1.445*** (.077)	.424*** (.044)	1.377*** (.073)	.439*** (.046)	1.393*** (.075)

(continued)

Table 4. (continued)

	Model 1		Model 2		Model 3		Model 4	
	(Basic Controls)		(Model 1 + Chronic Conditions)		(Model 2 + Drugs with Depression/ Suicide Side Effects)		(Model 2 + Drugs with Depression/Suicide Side Effects, Excluding Antidepressants)	
	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)
	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)
Educational attainment (less than high school is reference)								
High school graduate	–.435*** (.064)	.776*** (.036)	–.341*** (.061)	.826*** (.039)	–.359*** (.055)	.802*** (.038)	–.347*** (.056)	.816*** (.039)
College or above	–.943*** (.064)	.475*** (.027)	–.707*** (.060)	.557*** (.031)	–.726*** (.055)	.531*** (.029)	–.709*** (.058)	.546*** (.031)
Under federal poverty guideline	1.600*** (.068)	2.522*** (.107)	1.379*** (.063)	2.188*** (.095)	1.189*** (.058)	1.979*** (.085)	1.228*** (.059)	2.007*** (.088)
Has any insurance	–.005 (.061)	1.022 (.053)	–.191** (.058)	.870* (.047)	–.415*** (.056)	.687*** (.037)	–.370*** (.055)	.728*** (.039)
Chronic conditions[a]	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Number of observations	127,258	127,258	127,258	127,258	127,258	127,258	127,258	127,258

Note: All analyses are weighted using survey weights. Data from Medical Expenditure Panel Survey 2008–2013. OR = odds ratio; coeff. = coefficient.

[a]Chronic conditions include a set of binary variables that indicate whether a person has ever been told by a doctor or health professional that he or she has the following conditions: angina pectoris, arthritis, asthma, coronary heart disease, high cholesterol, diabetes, emphysema, heart attack, heart condition/disease (excluding coronary heart disease, angina, and heart attack), hypertension, and stroke.

*$p < .05$, **$p < .01$, ***$p < .001$.

with the exception of the direct effect of Asian was no longer significant ($b = .103$, $p = .115$) and the direct effect of Hispanic emerged as borderline significant ($b = -.122$, $p = .037$).

These patterns are due in part to the particularly powerful relationship between drugs with suicide as a side effect and the risk of significant distress, especially when using three or more such medications. Taking three or more medications with suicide as a side effect results in an increase in the odds of significant distress by a factor of more than 10. Even one such medication more than doubles the odds of significant distress. Drugs with depression as a side effect are consequential, too, though the relationship is considerably weaker than it is for suicide. To be sure, some of the relationship between drugs with suicide as a side effect and significant distress could be driven by the inclusion of antidepressants in the count. Such drugs involve suicide as a side effect and therefore are relevant to considering nonspecific psychological distress, but such drugs obviously do not involve depression as a side effect. The next pair of models eliminates antidepressants from the count. This reduces the relationship between drugs with suicide as a side effect and the risk of significant distress, though it does not reduce the relationship by much. For instance, in the K6 models, the coefficient for three or more such drugs is reduced by only 3% between Models 3 and 4. Moreover, pharmaceutical side effects continue to explain the racial-ethnic minority advantage with respect to significant distress. The mediating effect for the black-white difference in the K6 score is weaker than for Model 3, though side effects still explain 32% of the difference.

Table 5 employs several sensitivity tests. The primary threat to the models presented in Table 4 is that the relationship between the use of pharmaceuticals with side effects and the K6 reflects unobserved heterogeneity rather than side effects

Table 5. Sensitivity Analysis of K6 Score and Significant Distress (K6 ≥ 13) Regression Models.

	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	All Adults 18+		All Adults 18+		All Adults 18+		All Adults 18+		Young Adults 18–30		Young Adults 18–30	
	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)
	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)
Race and ethnicity (non-Hispanic white is reference)												
Non-Hispanic black	−.364***	.904	−.350***	.903	−.707***	.767*	−.463***	.977	−.500***	.811	−.276**	.979
	(.053)	(.050)	(.053)	(.050)	(.094)	(.083)	(.087)	(.106)	(.104)	(.093)	(.104)	(.115)
Non-Hispanic Asian	.115	1.042	.128	1.033	−.245	.552*	.018	.696	−.395**	.589*	−.165	.699
	(.065)	(.088)	(.065)	(.088)	(.129)	(.130)	(.129)	(.163)	(.131)	(.121)	(.133)	(.149)
Hispanic	−.123*	1.061	−.103	1.061	−.447***	.684***	−.260**	.818	−.514***	.779*	−.315***	.912
	(.058)	(.057)	(.058)	(.057)	(.097)	(.069)	(.097)	(.084)	(.087)	(.077)	(.086)	(.096)
Whether taking drugs with depression side effects (none is reference)												
1 drug	.339***	1.223***	.284***	1.204***			.413***	1.317**			.262**	1.058
	(.034)	(.053)	(.034)	(.052)			(.077)	(.111)			(.089)	(.100)
2 drugs	.729***	1.507***	.656***	1.468***			.665***	1.702***			.433**	1.513*
	(.064)	(.090)	(.065)	(.090)			(.135)	(.198)			(.166)	(.264)
3+ drugs	1.172***	1.585***	1.094***	1.527***			1.089***	1.768**			1.373***	1.948*
	(.110)	(.121)	(.111)	(.120)			(.287)	(.375)			(.387)	(.503)
Whether taking drugs with suicide side effects (exclude antidepressant drugs; none is reference)												
1 drug	1.440***	2.358***	1.406***	2.320***			1.466***	2.624***			1.236***	2.006***
	(.055)	(.104)	(.055)	(.099)			(.123)	(.248)			(.166)	(.269)
2 drugs	2.609***	3.777***	2.584***	3.722***			2.600***	3.949***			2.580***	3.501***
	(.114)	(.265)	(.114)	(.255)			(.247)	(.567)			(.365)	(.813)
3+ drugs	5.071***	7.796***	5.043***	7.684***			5.959***	13.792***			5.367***	8.440***
	(.183)	(.621)	(.185)	(.612)			(.456)	(2.298)			(.624)	(2.158)

(continued)

Table 5. (continued)

	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	All Adults 18+		All Adults 18+		All Adults 18+		All Adults 18+		Young Adults 18–30		Young Adults 18–30	
	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)	K6 Score	(K6 ≥ 13)
	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)	Coeff. (SE)	OR (SE)
Whether taking drugs without depression/suicide side effects (none is reference)												
1 drug			.344***	1.180**							.507***	1.419**
			(.041)	(.067)							(.073)	(.153)
2 drugs			.322***	1.266***							.600***	1.593***
			(.049)	(.086)							(.112)	(.218)
3+ drugs			.330***	1.243**							.690***	1.336*
			(.056)	(.084)							(.147)	(.195)
Chronic conditions[a]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Comorbidity[b]	Yes	Yes	No	No	No	No	No	No	No	No	No	No
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Number of observations	127,258	127,258	127,258	127,258	27,494	27,494	27,494	27,494	30,841	30,841	30,841	30,841

Note: All analyses are weighted using survey weights. All analyses control for gender, age, marital status, education, poverty status, and insurance status as in Table 3. Data from Medical Expenditure Panel Survey 2008–2013. OR = odds ratio; coeff. = coefficient.

[a]Chronic conditions include a set of binary variables that indicate whether a person has ever been told by a doctor or health professional that he or she has the following conditions: angina pectoris, arthritis, asthma, coronary heart disease, high cholesterol, diabetes, emphysema, heart attack, heart condition/disease (excluding coronary heart disease, angina, and heart attack), hypertension, and stroke.

[b]Comorbidity includes all pairwise interactions between chronic conditions.

*$p < .05$, **$p < .01$, ***$p < .001$.

specifically, most likely with respect to poor physical health. The models presented in Table 4 already control for a broad set of chronic conditions, though there are other possibilities that can be addressed empirically. The first model of Table 5 presents a regression model identical to Model 3 of Table 4 but includes all possible two-way interactions between the chronic conditions dummy variables (coefficients not shown). This model, in effect, tests whether patterns in comorbidity are important in a nonadditive fashion. The inclusion of these interactions does little to change the parameters for race-ethnicity or the parameters for pharmaceutical side effects.

There are other ways to explore unobserved heterogeneity with respect to health. It is possible that drugs involving depression or suicide as a side effect have no more of a relationship with the K6 than drugs that do *not* have depression or suicide as a side effect—that the entire relationship between pharmaceutical use and the K6 is driven by underlying treated illness. Other studies have explored this possibility and found that drugs without depression or suicide as a side effect are statistically unrelated to depression (Qato et al. 2018), though it is important to see if the same holds true in the present study, which uses both a different data set and a different outcome. Model 2 suggests other drugs have a significant relationship with the K6, too, though the magnitude of the relationship is much weaker than it is for drugs involving suicide as a side effect. For each pair-wise coefficient comparison (i.e., one medication with suicide as a side effect is equal to one drug with neither depression nor suicide as a side effect), the difference is statistically significant at $p < .001$.

A narrower sensitivity test is to estimate the same models among those with only one chronic condition, thereby eliminating respondents with complex comorbidities altogether. Model 3 presents a baseline model, including all the controls presented in Model 1 of Table 4 but limiting the sample to those with a single condition. Model 4 then introduces controls for medications with depression and suicide as side effects. The same pattern is evident for this subsample as it is for the full sample. All the coefficients indicating a racial-ethnic minority advantage are eliminated once controls for medications with depression or suicide as a side effect are introduced.

The final two models provide an even more stringent test. Pharmaceuticals are taken for many reasons, including the treatment of disease but also its prevention and the treatment of risk rather than disease itself. Younger people are, on average, healthier than older people and so consume pharmaceuticals under different conditions. Models 5 and 6 limit the sample to those between the ages of 18 and 30. Although the magnitude of the racial-ethnic difference in distress itself differs in this subsample, the influence of pharmaceuticals with side effects is very similar to that estimated in the full sample, greatly reducing or eliminating the minority advantage.

DISCUSSION

This study used nationally representative data to explore the consequences of pharmaceutical side effects for racial-ethnic differences in distress. The results indicate that medications play a significant role in shaping the apparent mental health advantage among racial-ethnic minorities, though there is some variation between groups and between the continuous and categorical codings of the K6. The black-white difference in significant distress is reduced to statistical nonsignificance in a model controlling for side effects. The black-white difference in the continuous distress score remains significant, though the difference, as estimated with basic controls, is reduced by 32% in a model that excludes antidepressants (Model 4) and by 60% in a model that includes them (Model 3). The advantage in significant distress for Asians is reduced to statistical nonsignificance, and at least in Model 3, the advantage for significant distress turns into a significant disadvantage among Hispanics. In the case of Asians, the advantage for continuous distress is reduced to nonsignificance in Model 4. Although differences between whites and both Hispanics and blacks in continuous distress remain in Model 4, pharmaceutical side effects play an important role in explaining the minority mental health paradox.

These patterns are determined by two things. First, racial-ethnic differences in the use of pharmaceuticals are large. For virtually every class of medication, non-Hispanic whites consume more medications with side effects than do racial-ethnic minorities. The Asian-white difference and Hispanic-white difference is larger than the black-white difference, though all three differences are statistically significant. The fact that medications with side effects cut across disease categories suggests that the role of medications is not limited to domains where treatment disparities are well established or where medications are especially consequential for health, including, for instance, the more aggressive treatment of cardiovascular disease among non-Hispanic

whites (e.g., Davis et al. 2007). For this reason, the role of pharmaceutical side effects in the minority mental health advantage is likely to be preserved even if some especially prominent treatment disparities are eliminated. Second, the pattern is shaped by the large impact of these pharmaceutical on psychological distress, a pattern found in other data sets with different measures of depression and, of course, a pattern anticipated based on the observation of significant side effects in drug trials and postmarketing surveillance (Qato et al. 2018). In our study, drugs with suicide as a side effect are especially consequential. A single such medication more than doubles the odds of significant distress. Relative to other factors implicated in minority mental health, this relationship is large. For instance, it is as large as the relationship between poverty and significant distress and much larger when it involves three or more such medications. Furthermore, the percentage of the population exposed to this risk is not small. About 28% of non-Hispanic whites take at least one such medication, though no more than 15% of racial-ethnic minorities do. To be sure, the reason minority patients consume fewer such pharmaceuticals differs between racial-ethnic groups. Among African Americans, the lower consumption of such pharmaceuticals likely reflects a treatment disparity, whereas among Asians, it likely reflects better health. For this reason, controls for comorbid medical conditions play a different role between these groups, suppressing the difference for the former and reducing it for the latter. Furthermore, the mediating effect of drugs with side effects is smaller for the black-white difference than for other race-ethnic differences. Nonetheless, the fact that the non-Hispanic white population consumes more medications with significant side effects plays an important role in undermining their mental health relative to other racial-ethnic groups.

More research is needed on the social process that produces between-group differences in the use of pharmaceuticals. In this regard, there are at least two distinct topics to address: first, the process that leads to a race-ethnic difference in prescription drug use overall and second, the process that leads to the frequent use of medications with depression or suicide as a side effect. The relatively low levels of pharmaceutical use on the part of African Americans and Hispanics likely reflects a combination of patient and provider influences. On the one hand, racial-ethnic minorities might be less demanding with respect to requesting prescriptions. Some evidence indicates that black and Hispanic patients express greater concern than white patients regarding the quality-of-life effects of pharmaceuticals, including concerns about side effects (Adams et al. 2018; Huang et al. 2009). It is unclear where this concern comes from, though it need not reflect a misapprehension of the risks and benefits of pharmaceuticals. Indeed, recent evidence indicates that non-Hispanic white populations may be, if anything, too credulous of pharmaceuticals, rarely discussing alternative forms of treatment (Adams et al. 2018) or assuming that the use of a pharmaceutical is the privilege and right of a properly treated "pharmaceutical person" (Ballantyne et al. 2018). In addition, evidence indicates that the relationship between physical health and mental health is generally stronger for whites, perhaps increasing the demand for treatment irrespective of the taste for pharmaceuticals (Assari and Lankarani 2016). On the other hand, though, there is evidence that differences in prescription drug use emerge further upstream and involve the behavior of physicians. Physicians may hold beliefs that lead to significant undertreatment among racial-ethnic minorities, including the belief that African Americans tolerate pain better than non-Hispanic whites and therefore require fewer analgesics (Hoffman et al. 2016) or that white patients are more "medically cooperative" and therefore are more likely to follow through with treatment recommendations (Oliver et al. 2014). Understanding the white advantage with respect to pharmaceuticals requires understanding the reasons they are prescribed medications at higher rates as well as the reasons they might persist in taking such medications even when those medications present significant side effects.

It is also important to understand whether and how physicians consider psychological side effects when treating physical disease. One possibility is that physicians largely ignore the risk of depression as a side effect, either because the medications involved have proven value in treating illnesses of putatively greater significance or because physicians infer that a patient's depression is caused by disease. Many drugs with depression or suicide as a side effect are effective in treating the conditions they were designed to treat, to the point of being essential. For instance, some common antihypertensives involve depression as a side effect, though they also reduce blood pressure quickly and reduce the risk of cardiac events (Beers and Passman 1990). When presented with a significant risk factor like hypertension, a physician might ignore potential psychological side effects in favor of effectively treating the risk at hand. At the same

time, physicians might disregard depression in those patients who present with other significant illnesses. Oncologists' sensitivity to depression in their patients, for instance, can be as low as 6% (Newell et al. 1998). There are several explanations for why physicians overlook depression, but one is a straightforward causal inference: that physicians expect depression in cases of severe physical illness and that any depression will resolve if and when the illness that occasioned it is treated. Whatever the explanation, it is almost certainly the case that physicians either disregard the psychological side effects of medications or regard them as less clinically significant.

In general, the results of this study speak to the problem of multiple morbidity, hinting at some of its complexities for understanding population health. The use of multiple pharmaceuticals has increased over time, in tandem with the increasing intensity of medical intervention (Aronowitz 2012; Swinglehurst and Fudge 2017), a pattern some refer to as "pharmaceuticalization" in reference to the parallel concept of medicalization (Abraham 2010). Polypharmacy itself has been linked to adverse events and nonadherence, and there has been some discussion on how to assist physicians in managing the practice (Grando et al. 2012). Yet little is known about multiple morbidity per se or the ways in which the treatment of one disease has consequences for another, in large part because medical science continues to be organized around discrete disease categories. It is unlikely that medical treatment always involves complementarities between diseases. Some evidence points to a rise, for instance, in emergency room visits for adverse effects of medical treatment (Bernstein et al. 2003). But a lack of complementarities is perhaps especially likely when mental illness has consistently played a secondary role to physical illness. The scope of the side-effects issue is broad, and future research should consider the role of pharmaceutical side effects in other psychiatric disorders, including side effects related to anxiety, confusion, and psychotic syndromes.

As implied in the introduction, the results do not speak directly to any theory in medical sociology for understanding the mental health paradox given how the literature has mostly centered on adumbrating risks among the disadvantaged, though the results are at least thematically consistent with certain facets of the literature. Research on fundamental causes, for instance, has occasionally uncovered countervailing mechanisms, providing some degree of advantage for an otherwise disadvantaged population (Lutfey and Freese 2005). Mechanisms of such sort are not unanticipated when disease is caused by a complex social process involving a mix of intentional and unintentional influences. Subsequent refinements of the fundamental cause theory have explicitly called for a deeper consideration of such countervailing mechanisms and has cast such mechanisms either in terms of well-resourced people pursuing multiple goals, some of which occasionally cut against health, or in terms of imperfect knowledge regarding how to pursue better health (Phelan, Link, and Tehranifar 2010). Yet Phelan and colleagues (2010) cautioned scholars to avoid using the concept of countervailing mechanisms to provide only a post hoc justification for results that are inconsistent with fundamental cause theory. They further suggested that some behaviors can be well accommodated in the theory, as when the pursuit of status undermines health. The results of the current study provide a different way forward. They suggested that countervailing mechanisms can appear even in the context of pursuing better health, in this case, poor mental health emerging in pursuit of optimal physical health. The results also suggest that countervailing mechanisms can be born of something other than imperfect knowledge or asymmetric goals. In the present study, the lack of consideration of side effects likely reflects a structured neglect of certain side effects on the part of both patients and providers, rather than simple ignorance or lack of awareness.

More generally, the results speak to the potential adverse consequences of exceptionally high levels of confidence in medicine. Americans maintain enormous confidence in their personal physicians and in the medical treatment of disease. As medical authority continues to expand over a wider set of conditions and the pharmaceutical armamentarium continues to grow (Clarke et al. 2003), the apparent benefits of pharmaceuticals might loom so large as to overwhelm serious consideration of their numerous side effects. To be sure, the neglect of side effects might be especially prominent with respect to mental health given that patients and physicians might be willing to trade some measure of emotional well-being for an improvement in disease, physical functioning, or longevity. But the tendency to neglect side effects is more general, and other side effects are almost certainly overlooked as well. Adapting theories regarding the minority mental health advantage only requires a more complete accounting of risks and resources, arrayed over a somewhat more complex ledger. It also requires integrating sociology's understanding of the causes of mental health, informed by fundamental cause

theory, with its understanding of how patients interface with medicine.

Limitations

This study has several limitations on both the independent and dependent variable sides. Although the medication data provided in MEPS is among the best among nationally representative surveys for evaluating health care, there are several reasons to suspect the effect of medications with depression and suicide as side effects, as estimated here, is understated. The analyses are premised on drugs that formally list depression or suicide as a side effect, though there are likely other medications that involve such side effects. It is not uncommon, for instance, for new side effects to become apparent only in Phase IV of drug development, during postmarketing surveillance, when drugs are effectively evaluated in a naturalistic setting (Suvarna 2010). At the same time, respondents may neglect to report all the medications they are taking, especially over-the-counter medications taken on an occasional basis. Most medications with significant side effects are prescription medications, though a number of over-the-counter medications also involve depression as a side effect, including proton-pump inhibitors, used to reduce stomach acid production (Laudisio et al. 2018). It is unlikely, however, that a perfectly comprehensive assessment of medication use would substantially change the findings with respect to race-ethnicity and mental health. Racial-ethnic differences in the use over-the-counter medications are similar to those in prescription medications, suggesting a general reluctance to use drugs and not a specific reluctance to use what a doctor prescribes (Fillenbaum et al. 1993).

The dependent variable used in this study is a well-validated measure of psychological distress, and empirically, the models produce the pattern than research has tried to explain. Yet much of the research has focused on major depression, for which the K6 cannot provide a direct analogue. Furthermore, some reviews of the racial-ethnic differences in major depression and distress find that the black-white difference in the K6 is closer to parity than a black advantage (Barnes and Bates 2017). Furthermore, the impact of pharmaceutical side effects among blacks differs between the categorical and dimensional coding of the K6. Other dependent variables are likely to yield similar patterns to those found here, though the magnitude of the minority advantage as well as the mediating effects of pharmaceutical side effects are likely to differ.

Although we performed numerous sensitivity analyses with respect to the relationship between drug use and depression, there remains the possibility of unobserved confounding, perhaps premised on prodromal rather than manifest disease and therefore based on diseases not reported by the respondent. The relationship between physical disease and depression is not limited to the most significant illnesses, the kind that are usually measured in omnibus health surveys. The risk of suicide, for instance, increases among those who have been hospitalized for *any* illness (Qin et al. 2013). At least some of the relationship between the use of pharmaceuticals and depression might be due to unmeasured morbidity, especially among those taking multiple medications. But given the breadth of controls for chronic disease employed in this study as well as specifications that specifically focused on comorbidity, it is unclear what the relevant set of other morbidities might be. Furthermore, the evidence linking pharmaceuticals to depression and suicide is strong. A significant *upward* bias in the coefficients seems unlikely.

CONCLUSION

The racial-ethnic minority advantage in mental health has long puzzled social scientists. Although there is much to be gained from thinking about protective factors among minorities, the present study suggests it is still worth considering risk factors that are more common among non-Hispanic whites. Such risk factors are not easy to identify, given social scientists' interest in uncovering stressors among racial-ethnic minorities. And this interest is theoretically well founded—the idea of fundamental causes suggests multiple overlapping risk factors flowing from one fundamental cause such that any one risk can be replaced by another and still preserve the effects of the cause (Link and Phelan 1995). To explain paradoxes, though, a different perspective is required. Identifying relevant risk factors among non-Hispanic whites might require considering the shadow side of otherwise desirable therapeutic interventions. And explaining disparities in one domain of health might require considering disparities in a seemingly very different one.

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