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The Combined Association of Psychological Distress and Socioeconomic Status With All-Cause Mortality

A National Cohort Study

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Background: Psychological distress and low socioeconomic status (SES) are recognized risk factors for mortality. The aim of this study was to test whether lower SES amplifies the effect of psychological distress on all-cause mortality.

Methods: We selected 66 518 participants from the Health Survey for England who were 35 years or older, free of cancer and cardiovascular disease at baseline, and living in private households in England from 1994 to 2004. Selection used stratified random sampling, and participants were linked prospectively to mortality records from the Office of National Statistics (mean follow-up, 8.2 years). Psychological distress was measured using the 12-item General Health Questionnaire, and SES was indexed by occupational class.

Results: The crude incidence rate of death was 14.49 (95% CI, 14.17-14.81) per 1000 person-years. After adjustment for age and sex, psychological distress and low SES category were associated with increased mortality rates. In a stratified analysis, the association of psychological distress with mortality differed with SES (likelihood ratio test-adjusted $P < .001$), with the strongest associations being observed in the lowest SES categories.

Conclusions: The detrimental effect of psychological distress on mortality is amplified by low SES category. People in higher SES categories have lower mortality rates even when they report high levels of psychological distress.

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PSYCHOLOGICAL DISTRESS IS becoming recognized increasingly as a risk factor for mortality and a trigger for cardiovascular disease (CVD) events.¹⁻³ Socioeconomic status (SES) is also a recognized determinant of health status: in developed countries, lower SES levels signal worse health. Even in the most affluent countries, people in lower SES levels have considerably shorter life expectancies and more disease than people in higher SES levels,⁴⁻⁶ and low SES levels are associated with a high risk for CVD and death in developed countries, such as England.⁷

People in higher SES categories may have greater economic, social, and psychological resources and better coping strategies for dealing with adversity.⁸ These assets may be acquired through learning or better access to resources. Consequently, when both risk factors are present (high levels of psychological distress and low SES levels), we can argue that the resulting effect on mortality is not the mere sum of the two (additive effect) but that some extra risk may appear (multiplicative effect). We therefore hypoth-

esized that SES can operate as an amplifier of psychological distress and that the effect of psychological distress on mortality would be greater in groups with lower compared with higher SES levels. As a consequence, vulnerable populations of adults may be more susceptible to the detrimental effects of psychological distress and may have unmet health care needs.

See Invited Commentary at end of article

Identifying people who are more vulnerable to the health consequences of psychological distress may have clinical and public health implications. For example, questionnaires such as the 12-item General Health Questionnaire (GHQ-12) could be of value in systematic screening by family physicians with the aim of improving the recognition rate of common mental disorders and thereby reducing the risk for CVD and other fatal conditions. We sought to analyze the association of psychological distress and low SES levels on the incidence of all-cause mortality, with an emphasis on the interaction between both risk factors.

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STUDY DESIGN AND VARIABLE SELECTION

The analysis was based on the Health Survey for England (HSE), a nationally representative, general population-based study that recruits individuals living in private households in England using stratified random sampling. The HSE consists of a series of annual surveys beginning in 1991 and is designed to provide regular information on various aspects of the nation's health. The HSE has a set of core annual measurements, including general health, SES, height, weight, blood pressure, health behaviors (eg, smoking, alcohol consumption, and physical activity), and blood and saliva factors. Psychosocial factors, such as psychological distress and social relationships, are also assessed through household visits, during which information is collected using the Computer-Assisted Personal Interviewing method. Trained interviewers collect information about physician-diagnosed CVD and diabetes mellitus and measure height and weight. In a separate household visit, trained nurses collect blood samples and measure resting blood pressure using a digital monitor (HEM-907; Omron Healthcare Inc).⁹ Diabetes mellitus was defined as presenting with a self-reported clinician's diagnosis. Hypertension was defined as presenting with a clinical blood pressure reading using the conventional criteria ($>140/90$ mm Hg), a self-reported clinician's diagnosis, or a prescription of antihypertensives. Smoking and physical activity (defined as the number of sessions of moderate or vigorous physical activity per week excluding domestic activity) were self-reported. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared.

We pooled HSE years 1994 through 2004 and used all participants 35 years or older to constitute a baseline sample for a cohort study. Consenting study members were linked to National Health Service mortality data, which record and certify all deaths in the United Kingdom. Information about their status was obtained to February 28, 2008 (censoring date). Besides our main outcome variable of all-cause mortality, we included secondary outcomes, such as mortality due to stroke and coronary heart disease (CHD). Classification of the underlying cause of death was based on information collected from the death certificate together with any additional information provided subsequently by the certifying physician (eg, secondary death cause). The diagnosis for the primary cause of death was recorded using the *International Classification of Diseases, Ninth and Tenth Revisions (ICD-9 and ICD-10, respectively)*. Codes for CVD were 390 to 459 for ICD-9 and I01 to I99 for ICD-10, which were further categorized into CHD (410-414 [ICD-9] and I20-I25 [ICD-10]) and cerebrovascular disease (430-438 [ICD-9] and I60-I69 [ICD-10]). Patients with a history of stroke (including transitory ischemic attack), CHD (including angina), and any other CVD or cancer at baseline were excluded from the analysis based on the ad hoc findings at individual nurse visits. The variables of hypertension and physical activity were planned to be recorded only in the years 1994, 1997 (physical activity only), 1998, 1999, 2003, and 2004. We have therefore excluded those variables from the main multivariate analyses.

We used the profession of the individual as an indicator of SES. The Registrar General's social classification of occupations attempts to classify groups on the basis of employment using characteristics such as career prospects, autonomy, mode of payment, and period of notice.¹⁰ The HSE uses a 6-category system in which informants are classified as managerial and professional, intermediate, small employers and self-employed workers, lower supervisory and technical, semiroutine, and routine occupations. For some analyses, we further collapsed the 6 cat-

egories into 3 (professional or managerial positions, skilled manual or nonmanual workers, and semiroutine or unskilled workers). The classification is based on data from the head of the household. If this person was unemployed at the time of the survey, the classification was based on their most recent employment.¹¹

We used the GHQ-12 to measure psychological distress.¹² The GHQ-12 is generally considered to be a unidimensional scale,¹³ and it consists of 12 items relating to anxiety, depression, social dysfunction, and loss of confidence. Interpretation of the answers is based on a 4-point response scale scored using a bimodal method (for symptom present, 0 indicates not at all and same as usual; 1, more than usual and much more than usual). The questionnaire therefore gives a score for psychological distress from 0 to 12. At the analysis stage, the variable GHQ-12 can be used as ordered categorical (0, low distress; 1-3, medium distress; and ≥ 4 , high distress) or as binary (0-3, low distress and ≥ 4 , high distress) data.¹²

Study participants gave full informed consent. Ethical approval was obtained from the London Research Ethics Committee.

DATA ANALYSIS

Data were cleaned before the analysis. Inconsistent, duplicate, outlier, and missing values and digit preferences were checked. The normality of each continuous variable was checked. We calculated the proportion of participants who died within the follow-up period and the crude incidence rate for the cohort. The main exposure variables were SES (occupational class) and psychological distress (GHQ-12).

Data were analyzed using Cox proportional hazards regression with follow-up time (in months) as the time scale. We constructed a multiple Cox proportional hazards regression model for the association of SES (ordered categorical with 3 categories), GHQ-12 (ordered categorical with 3 categories), age (per 1-year linear increase), sex (binary), current smoking (binary), BMI (categorical: <18.5 , 18.5-29.9 [reference], and ≥ 30.0), diabetes mellitus (binary), and an interaction variable calculated as the multiplication of SES and GHQ-12 (3×3), with the outcome using the forward stepwise approach. In this approach, the variables were sequentially added to an "empty" (intercept-only) model, one at a time, giving priority to those variables that had shown the strongest evidence of association at the univariate stage (smallest *P* value). At each round, the importance of the added variable was assessed according to changes in the rate ratios, Wald tests, and likelihood ratio tests (LRTs of all) *P* value changes (cutoff, .05) in all variables in the model. If a variable lost significance, we removed it from the model. After fitting the final model, we checked the proportional hazards assumption and the departure from linearity.

Finally, we assessed interaction between SES level and the GHQ-12 score using the LRT in 3 separate models: one without any adjustment, one with adjustment for age and sex, and one with further adjustment for smoking, BMI, physical activity, diabetes mellitus, and hypertension. The following analysis strategy was used: we ran a model with SES level, GHQ-12 (binary scores 0-3 vs ≥ 4), and eventually the other covariates; the model was then repeated adding in an interaction variable between the SES level and GHQ-12, and the estimates from this second model were then compared with the estimates from the initial model using the LRT. For this test to be valid, the comparison has to be performed on the same group of individuals (missing values can distort the results), and this assumption was always satisfied.

We performed a sensitivity analysis by restricting the multivariate analyses to those years that included data collection for the variables hypertension and physical activity ($n = 35\,090$)

Table 1. Sample Description and Unadjusted HRs for All-Cause Mortality for 66 518 HSE Participants^a

Factor and Category at the Beginning of Follow-up	Mortality Within the Follow-up Time		HR (95% CI)	P Value
	Yes (n = 7875)	No (n = 58 643)		
Male sex, %	49.7	44.9	1.19 (1.14-1.24)	<.001
Age, mean (SD), y	72.1 (12.1)	52.5 (12.4)	1.11 (1.11-1.11)	<.001
Age group, % ^b				
35-49 y	5.8	46.5	5.88 (5.64-6.12)	<.001
50-64 y	16.9	34.2		
≥65 y	77.3	19.3		
SES category, % ^b				
Professional/managerial positions (high)	24.5	35.1	1.20 (1.18-1.22)	<.001
Skilled manual/nonmanual workers (medium)	45.8	42.9		
Semiroutine/unskilled workers (low)	29.7	22.0		
GHQ-12 score, mean (SD) ^c	1.7 (2.8)	1.4 (2.6)	1.04 (1.03-1.05)	<.001
GHQ-12 score group, %				
0	54.3	62.0	1.30 (1.25-1.36)	<.001
1-3	27.6	24.1		
≥4	18.1	13.9		
Smoking, % ^d				
≥20 cigarettes/d	8.6	8.9	1.54 (1.47-1.61)	<.001
1-19 cigarettes/d	15.0	13.8		
Ex-smoker	44.3	34.6		
Never smoked	32.1	42.6		
BMI, mean (SD)	26.7 (4.9)	27.2 (4.7)	0.99 (0.98-0.99)	<.001
BMI group, %				
<18.5	1.9	0.6	3.56 (2.97-4.27)	<.001
18.5-30.0	56.0	66.4	1 [Reference]	
>30.0	42.1	33.0	1.13 (1.08-1.19)	<.001
Physical activity, mean (SD) ^e	0.6 (1.9)	1.6 (2.8)	0.80 (0.78-0.81)	<.001
Diabetes mellitus, %	6.7	2.6	2.78 (2.54-3.03)	<.001
Hypertension, % ^f	29.4	17.5	1.85 (1.73-1.98)	<.001

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); GHQ-12, 12-item General Health Questionnaire; HR, hazard ratio; HSE, Health Survey for England; SES, socioeconomic status.

^aParticipants were 35 years or older and free of cancer and cardiovascular disease at baseline (1994-2004). Hazard ratios were computed using unadjusted Cox proportional hazards regression.

^bIndicates per 1-category increase.

^cTreated as a binary variable (≥4 vs 0-3).

^dTreated as a binary variable (current smokers vs ex-smokers or those who never smoked).

^eIndicates number of sessions of moderate or vigorous physical activity per week excluding domestic activity (n = 39 610).

^fIncludes 35 090 participants.

and by adding those variables into the models. The entire analysis was repeated separately for men and women, participants aged 35 to 54 years and 55 years or older, and participants visited during 1994 through 1999 and 2000 through 2004.

RESULTS

The initial study sample consisted of 96 605 adults, although 10 065 (10.4%) did not consent to mortality follow-up and were therefore removed from any analysis. Nonconsenting adults were on average older than those consenting (mean ages, 64.3 vs 56.1 years [$P < .001$]). Of the consenting adults, 5864 (6.8%) had a history of stroke or CHD or another prevalent CVD or cancer at baseline and were therefore excluded. Of the resulting 80 676 participants, 15.4% had missing values for psychological distress and 2.6% for SES. Participants with GHQ-12 missing values were slightly older compared with those who completed the GHQ-12 questionnaire (56.4 vs 55.1 years [$P < .001$]), whereas the sex structures of the 2 subgroups were similar (men, 45.4% vs 44.8% [$P = .23$]). The outcomes of 6 participants could not be recorded during

the follow-up, and 27 were excluded from the analysis because they experienced an outcome within 1 month from recruitment. The final analytic sample consisted of 66 518 participants. The measures of hypertension and physical activity had about 40% missing values.

The participants were followed up for a mean of 8.2 (SD, 3.4; median, 7.9) years. During this period, 555 (0.8%) died of a stroke, 1007 (1.5%) died of a CHD event, and 7875 (11.8%) died of any cause. The crude incidence rates for stroke, CHD, and all-cause mortality were 1.02 (95% CI, 0.94-1.11), 1.85 (1.74-1.97), and 14.49 (14.17-14.81) per 1000 person-years, respectively. The 3 outcomes have shown very similar patterns in all analyses; hence, we only report the results relative to all-cause mortality.

Table 1 shows the baseline characteristics of the sample. On average, 14.4% of the sample reported psychological distress based on the established cutoff point of a GHQ-12 score of 4 or more. Participants from lower occupational classes were older, were less likely to be male, had higher GHQ-12 scores, and were more likely to be smokers. Psychological distress and low SES level were associated with increased mortality rates, as were diabe-

Table 2. Multivariate Cox Proportional Hazards Regression Model Showing HRs for All-Cause Mortality

Factor at the Beginning of Follow-up	Mutually Adjusted Risk for Death	
	HR (95% CI)	P Value
Male sex	1.47 (1.40-1.54)	<.001
Per 1-y increase in age	1.11 (1.11-1.11)	<.001
Per 1-unit increase in SES from high to low (3 categories) ^a	1.21 (1.16-1.26)	<.001
Per 1-unit increase in GHQ-12 (3 categories) ^a	1.38 (1.26-1.50)	<.001
Interaction, SES × GHQ-12 (3 × 3) ^b	1.06 (1.01-1.10)	.02
Current smoking	1.42 (1.35-1.49)	<.001
BMI		
<18.5 vs 18.5-29.9	2.39 (2.04-2.80)	<.001
≥30.0 vs 18.5-29.9	1.08 (1.02-1.15)	.008
Diabetes mellitus	1.62 (1.48-1.77)	<.001

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); GHQ-12, 12-item General Health Questionnaire; HR, hazard ratio; SES, socioeconomic status.

^aCategories are described in Table 1.

^bDescribed in the "Data Analysis" subsection of the "Methods" section.

tes mellitus, hypertension, and smoking. Physical activity was associated with a lower risk for mortality. Participants with a BMI of less than 18.5 or more than 30.0 had higher mortality rates than did those with a BMI of 18.5 through 30.0 (reference category).

Table 2 shows the results from the multivariate analysis performed using Cox proportional hazards regression. After adjusting for age, sex, smoking, BMI, diabetes mellitus, and SES level, psychological distress was associated with higher mortality rates. Socioeconomic status demonstrated a similar pattern: after adjusting for age, sex, smoking, BMI, diabetes mellitus, and psychological distress, SES level was associated with higher mortality rates. An interaction variable (multiplication between the SES level and psychological distress) that was inserted into the model as an independent linear variable showed an association with higher mortality rates after being adjusted for SES level, psychological distress, and all other covariates (hazard ratio [HR], 1.06 [95% CI, 1.01-1.10; $P = .02$]).

Table 3 presents the results of an analysis focused on the interaction between the SES level and GHQ-12; that is, it shows the crude and adjusted HRs of psychological distress on mortality stratified by SES level. After adjusting for age, sex, smoking, BMI, and diabetes mellitus, we found a significant interaction showing that psychological distress demonstrated stronger associations with mortality in participants with lower SES levels (LRT-adjusted $P = .01$).

The **Figure** shows age- and sex-adjusted HRs for all-cause mortality (y-axis) as a function of psychological distress (x-axis), separately for each stratum of SES, with the reference category (HR, 1) being participants with low psychological distress (GHQ-12 score, 0) and a high SES level (professional or managerial positions). The gradient of the line reflecting the association between psychological distress and all-cause mortality differs according to SES: it is flatter in participants with a high SES level

Table 3. Crude and Adjusted HRs for the Association Between Psychological Distress and All-Cause Mortality Stratified by SES

SES Level ^a	High vs Low Psychological Distress on All-Cause Mortality, HR (95% CI) ^b		
	Crude	Adjusted for Age and Sex	Multi-Adjusted ^c
High	1.18 (1.07-1.29)	1.26 (1.18-1.34)	1.22 (1.13-1.31)
Medium	1.32 (1.23-1.41)	1.37 (1.27-1.49)	1.33 (1.21-1.45)
Low	1.36 (1.25-1.47)	1.46 (1.33-1.59)	1.36 (1.23-1.51)
Likelihood ratio test—adjusted P value	.004	.001	.01

Abbreviations: HR, hazard ratio; SES, socioeconomic status.

^aHigh SES indicates professional/managerial positions; medium SES, skilled manual/nonmanual workers; and low SES, semiroutine/unskilled workers.

^bLow psychological distress level indicates a 12-item General Health Questionnaire (GHQ-12) score of less than 4 (reference category); high psychological distress level, a GHQ-12 score of 4 or more.

^cAdjusted for age, sex, smoking, body mass index, and diabetes mellitus.

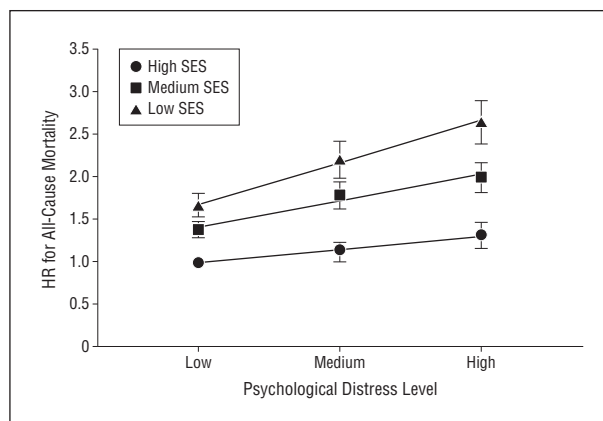


Figure. Age- and sex-adjusted hazard ratios (HRs) for all-cause mortality as a function of psychological distress for each stratum of socioeconomic status (SES). The reference category (HR, 1.00) included participants with low psychological distress (General Health Questionnaire score, 0) and a high SES level (professional or managerial positions) (likelihood ratio test-adjusted $P < .001$). Whiskers represent 95% confidence intervals.

and steeper in participants with a low SES level (LRT-adjusted $P < .001$).

The sensitivity analysis performed by adding the variables hypertension and physical activity into the multivariate models (the number of participants dropped to 35 090) showed a pattern of results that was similar to that found in the main analyses, with no differences in the key interactions. The entire analysis was repeated after stratification by sex, age, and survey year, and in all cases the results were similar to those of the main analysis.

COMMENT

We have shown that the association between psychological distress and all-cause mortality differs according to SES. A low SES level operates as an amplifier of the detrimental effect of psychological distress on mortality.

The differential effect of psychological stress on health outcomes across SES groups has not been directly investigated previously in a large prospective observational study, but limited evidence is available concerning exposure to specific sources of stress. For example, in a study of Japanese workers, job strain was associated with a higher risk for stroke in men from lower occupational classes but not in higher-status white-collar and managerial workers.¹⁴ Similarly, in a register-based cohort study of nearly 3.5 million Swedish men and women, low levels of job control constituted a risk factor for stroke in women working in lower-status manual jobs but not in higher-status non-manual occupations.¹⁵ Given that lower SES groups are more likely to be exposed to greater adversity and stress, several studies have also examined whether occupational stress might explain the social gradient in CVD risk. However, in a cohort of Finnish public sector workers, job demands alone or in combination with job control suppressed rather than explained SES differences in cerebrovascular disease.¹⁶

The explanations of why people from disadvantaged backgrounds are more vulnerable to stress than those from higher SES groups are poorly understood. However, people with higher SES levels might have better coping strategies and larger support networks together with greater biobehavioral and economic resources for dealing with adversity.⁸ In addition, higher SES groups demonstrate more effective recovery in cardiovascular and biological variables after acute stress,^{17,18} which might contribute to less CVD pathology over time.¹⁹

Smoking, BMI, hypertension, diabetes mellitus, and physical inactivity are also known risk factors for CVD and all-cause mortality. We took these factors into account, but we cannot rule out the possibility of residual confounding by the measured or by other unmeasured variables. Nevertheless, these factors may be on the causal pathway between SES level or psychological distress and the outcomes, so adjusting for them could diminish the effect of both main exposure variables and make their interaction less detectable. Under this perspective, the more appropriate analysis would be the age- and sex-adjusted one.

Body mass index may have a J-shaped association with mortality, with underweight and obese people having higher mortality rates than normal-weight people. Our results are compatible with the existing literature on this topic.²⁰

One limitation of the present study is a lack of follow-up data on psychological distress, so we were unable to account for the effects of changes in distress over time. The GHQ-12 is not designed to assess specific aspects of mental health, such as anxiety and depression. However, measuring symptoms of anxiety, depression, and dysfunction as a unidimensional construct of psychological distress is particularly relevant in community-based samples such as ours because mental health problems in the community are frequently characterized by shifting patterns of symptoms that resist precise clinical classification.²¹ Suls and Bunde²² have argued that different manifestations of psychological distress are not distinctive in their associations with CVD outcomes. Other indicators of SES might have been used, such as educa-

tional level or gross annual income. Occupational class was preferred because it is an indicator of current socioeconomic circumstances, whereas education is typically completed early in life and partly dictates life-course trajectories.²³ As for annual household income, the HSE has a relatively low response rate, like many other population surveys (about 50% of households had no valid data). With these limitations considered, we conclude that the effect of psychological distress on all-cause mortality is more pronounced in people from lower than higher SES groups.

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INVITED COMMENTARY

Differential Mortality for Persons With Psychological Distress and Low Socioeconomic Status

What Does It Mean and What Can Be Done?

We have long known that people who live in poverty have shorter life expectancies than those who are better off.¹ Similarly, psychological distress is a risk factor for early mortality.² Lazarino and colleagues³ provide evidence that the effect of psychological distress on mortality is greater among adults of lower socioeconomic status (SES). The finding is based on 66 518 adults completing the Health Survey for England in 1 of 10 years (1994-2004), with survey data linked to mortality data to 2008 (mean follow-up of 8 years). Their analysis relied on a brief measure of psychological distress (symptoms of anxiety and depression, low confidence, and social dysfunction), an occupation measure (categories from managerial/professional to unskilled), and adjustment for age, sex, body mass index, smoking, and diabetes mellitus and for hypertension and physical activity in sensitivity analyses. They found that occupational status and psychological distress had significant main effects on mortality and an interaction reflecting a stronger effect of distress on mortality among persons of lower social class. The authors featured all-cause mortality but found similar conclusions for mortality due to stroke and coronary heart disease and for men and women, older and younger adults, and early and late survey cohorts.

A higher relative risk for mortality with mental disorders such as depression (relative risk, 1.7), schizophrenia and bipolar disorder (2.6), alcohol abuse (1.8), and substance abuse (2.0)⁴ has been well documented, and the risk is largely owing to medical causes rather than accidents and suicides. A recent nationally representative study in the United States found that socioeco-

nomics factors largely accounted for approximately one-quarter of mortality in persons with mental illnesses, a figure similar to estimates of the role played by SES in the health of the general population.⁵ If this finding is true, the excess mortality associated with lower SES among persons with psychological distress may be accounted for by those who are distressed and poor but do not have mental disorders. On the other hand, depression and anxiety can interfere with adherence to treatments for physical conditions, can affect coping responses in the face of stressors, and have been associated with changes in biological response, such as changes in the inflammatory and cortisol responses. The identification by Lazarino and colleagues of an interactive effect between psychological distress and SES on mortality makes clear the need to understand the mechanisms underlying this compound burden on health and to develop interventions specifically to improve distress and physical health in lower-SES populations.

What are the implications of these findings for lower-SES communities? Culture may be a powerful mediator of health in poor communities. Evidence suggests that immigrant Mexican Americans enjoy better mental and physical health than their US-born Mexican American counterparts.⁶ Similarly, despite higher rates of poverty, ethnic minority populations enjoy lower or similar rates of mental disorders than their wealthier white counterparts.⁶ Efforts to strengthen the social capital of poor communities might well reverse the dual challenges to health of poorer resources and marginalization or demoralization.

What are the clinical implications of this potential interaction, and what might clinicians or health care sys-