



The Role of Clinical Risk Factors in Understanding Self-rated Health

NOREEN GOLDMAN, DSc, DANA A. GLEI, PhD, AND MING-CHENG CHANG, PhD

PURPOSE: This study examines the importance of clinical risk factors for predicting self-ratings of general health status, with and without controls for a broad range of self-reported indicators of physical and psychological well-being.

METHODS: Ordered probit models, estimated on 928 respondents aged 54 years and older who participated in an ongoing national survey in Taiwan, are used to examine the correlates of self-rated health. The model is estimated in two stages, first testing the association between clinical risk factors and self-rated health and second, examining whether those clinical measures remain significant after controlling for a large number of other factors hypothesized to affect self-ratings.

RESULTS: Most of the clinical variables are significantly associated with self-rated health, even in the presence of control variables. The largest effects pertain to the BMI, ratio of total to HDL cholesterol (among men) and presence of the $\epsilon 4$ allele of the APOE gene (among women).

CONCLUSIONS: The results suggest a variety of pathways linking clinical measures to the self-ratings. The findings also suggest that the clinical measures are less powerful predictors than self-reports about diverse aspects of well-being.

Ann Epidemiol 2004;14:49–57. © 2003 Elsevier Inc. All rights reserved.

KEY WORDS: Self Assessment, Health Status, Biological Markers, Clinical Markers, Risk Factors, Taiwan.

INTRODUCTION

An extensively used measure of self-rated health is based on a simple question that asks respondents to rate their current overall health, typically on a five-point ordinal scale ranging from “excellent” to “poor.” Regardless of variations in phrasing, responses to this question have consistently been powerful predictors of survival (1–3), functional decline (4–9), future morbidity (10–12), and subsequent health service utilization (13, 14). Most studies find that this measure remains an independent predictor of subsequent health outcomes, even after adjustment for other measures of self-reported and physician-observed health status, behavioral and psychosocial risk factors, and environmental factors (2, 3). Thus, our understanding of the factors that influence self-assessed health status remains incomplete.

Both qualitative and quantitative studies provide evidence that these self-ratings involve complex judgments across multiple domains of health (15, 16). For example, not only do self-assessments take into account physical health

conditions, they also incorporate psychological well-being, social functioning, and positive affect (15, 17–19). In addition, they are associated with health behaviors, health services utilization, medication use, cognitive capacity, social networks, demographic factors, and socioeconomic status (10, 11, 15, 17–26).

Inadequate controls for physical health may partly explain the predictive strength of self-rated health. Idler and Angel argue that researchers’ inability to “unambiguously control for actual physical health status” is the primary methodological problem that limits their capacity to assess the medical importance of self-rated health (27). This inability to control for physical health may arise because of incomplete information about symptoms and conditions—especially those at a preclinical, undiagnosed stage—as well as reliance on self-reported conditions of illness or associated risk factors. Self-reports are frequently plagued by response errors resulting from poor recall, inadequate understanding of the condition, or a reluctance to confide in the interviewer. Validation studies, which use physicians’ assessments, clinical markers, and administrative records to identify underreporting of illnesses and conditions in interview surveys (28, 29), underscore the limited utility of self-reports of physical health as controls in statistical models.

This study uses a recent survey in Taiwan to investigate the role of clinical risk factors in predicting self-rated health and the extent to which those clinical measures remain important in the presence of controls for a broad range of self-reported indicators of health and well-being. Because most prior studies find that self-rated health status is a better predictor of subsequent mortality among men than among

From the Office of Population Research, Princeton University, Princeton, NJ (N.G.); Center for Population and Health, Georgetown University, Washington, DC (D.A.G.); and Center for Population and Health Survey Research, Bureau of Health Promotion, Department of Health, Taiwan, China (M.-C.C.).

Address correspondence and reprint requests to: Noreen Goldman, D.Sc., Office of Population Research, Princeton University, 243 Wallace Hall, Princeton, New Jersey, 08544-2091. Tel.: (609) 258-5724; Fax: (609) 258-1039. E-mail: ngoldman@princeton.edu

Received July 29, 2002; accepted March 26, 2003.

women (2, 3)—suggesting that men may evaluate their health differently than women—we also focus on sex differences in the predictors of self-rated health. The clinical data are derived from a health examination by trained professionals and laboratory analysis of blood and urine specimens. Although a few prior studies have employed so-called objective measures of health based on medical examination (30, 31) or biomarkers (18, 30), none has simultaneously controlled for the wide range of factors associated with self-rated health.

METHODS

Data

The data were originally collected as part of the Survey of Health and Living Status of the Near Elderly and Elderly in Taiwan. This longitudinal survey began in 1989 with a national sample (including the institutionalized population) of 4049 persons aged 60 years and older, and was extended in 1996 to include 2462 near elderly persons aged 50 to 66 years in 1996 (32). Both groups of respondents were re-interviewed in 1999.

In 2000, a national sub-sample of respondents was randomly selected for the Social Environment and Biomarkers of Aging Study (SEBAS). Elderly respondents (71 years and older in 2000) and residents of urban areas were oversampled. On a scheduled day several weeks after an initial interview, participants collected a 12-hour overnight urine sample (to provide integrated measures of cortisol, norepinephrine, and epinephrine), fasted overnight, and visited a nearby hospital the following morning. As part of the hospital visit, medically-trained professionals drew a blood sample and took blood pressure and anthropometric measurements, and participants filled out a questionnaire regarding their health history, health-related behaviors, and current medications. Written informed consent was obtained for participation in the interview and physical examination.

Among the 1713 respondents selected, 1497 provided interviews (92% of survivors) and 1023 participated in the physical examination (68% of those interviewed). Compliance with the clinical protocol was extremely high: almost all participants followed the urine protocol and provided a suitable blood sample. Seventeen participants were excluded from the analysis because a proxy completed the initial interview and 78 were excluded due to missing data on the dependent variable or a covariate. Thus, the sample includes 928 participants.

Although respondents over 70 were less likely than younger persons to participate, sex and various measures of socioeconomic status were not significantly related to

participation. Moreover, persons who received the medical exam reported the same general health status, on average, as those who did not. These results suggest that, in the presence of controls for age, estimates derived from clinical information are unlikely to be seriously biased (33).

Measures

The dependent variable comes from the 2000 interview and is based on responses to the following question: “Regarding your current state of health, do you feel it is excellent, good, average, not so good, or poor?” (English translation). This five-point ordinal measure is scored so that five indicates “excellent” health.

All of the clinical measures are derived from the physical examination and biomarker collection in 2000. The specific markers selected for inclusion have well-established associations with chronic illness and are comparable to measures in two recent population-based surveys in the US (34, 35). The body mass index (BMI) and waist-hip ratio provide indices of obesity and adipose tissue deposition, which are associated with type II diabetes, cardiovascular disease, stroke, female carcinomas, and premature death (36). Systolic and diastolic blood pressure are markers of hypertension, a well-known risk factor for heart disease, stroke, and kidney disease: the measurements are derived from the average of two readings from a mercury sphygmomanometer with the respondent in a seated position. Ratio of total to high-density lipoprotein (HDL) cholesterol is included because of its known effect on the development of atherosclerosis, a condition that increases the risk of heart disease and stroke. Glycosylated hemoglobin (HbA_{1c}) serves as an integrated measure of glucose metabolism over the previous 30 to 90 days (37, 38); it is an important indicator of a diabetic condition (39), which is itself a risk factor for a variety of illnesses including heart disease, stroke, and memory deficits.

We also include a measure of hypothalamic-pituitary-adrenal (HPA) axis activity: the ratio of cortisol to dehydroepiandrosterone sulfate (DHEA-S); DHEA and its sulfate form DHEA-S are thought to have anti-glucocorticoid effects, and thus, this ratio is believed to be a preferable measure of hypercortisolemia than cortisol alone (40). Norepinephrine and epinephrine excretion levels provide integrated indices of sympathetic nervous system (SNS) activity. HPA-axis and SNS activity have been linked to risk of diabetes, heart disease, and cognitive impairments (41, 42). Finally, a variable that identifies persons with at least one copy of the $\epsilon 4$ allele of the APOE gene is included because of the association of the allele with ischemic heart disease and Alzheimer’s disease (43).

The other covariates are based on self-reports and include measures of physical health and functioning, health care utilization, health behaviors, psychological well-being, social involvement, and sociodemographic factors. All of these measures are obtained from the 2000 interview, with the exception of pain or discomfort and incontinence, which are based on the 1999 interview.

The number of current illnesses is a count of 12 conditions: high blood pressure, diabetes, heart disease, cancer or malignant tumor, lower respiratory tract disease, arthritis or rheumatism, gastric ulcer or stomach ailment, liver or gall bladder disease, cataracts, kidney disease, gout, and spinal or vertebral spurs. These 12 conditions were chosen because they represent serious chronic illnesses (7 of the 10 leading causes of death in Taiwan), risk factors for serious illness, and conditions that are likely to cause considerable physical discomfort and/or severely limit functional mobility even if they are not ultimately fatal.

The number of mobility limitations is a count, out of nine activities, that the respondent has difficulty performing without aid: standing continuously for 15 minutes and for two hours, squatting, raising both hands over his or her head, grasping or turning objects with his or her fingers, lifting or carrying an object weighing 11 to 12 kg, running a short distance (20–30 m), walking 200 to 300 m, and climbing two or three flights of stairs. The most common mobility limitations involved standing for two hours (49%) and running a short distance (40%), while the least frequent was difficulty raising both hands over the head (4%). The total number of limitations has a skewed distribution: 37% of respondents have no limitations, while 11% have more than five limitations (data not shown).

An index of depression is based on 10 items from the Center for Epidemiologic Studies Depression (CES-D) scale. Previous studies have demonstrated that a shortened form of the CES-D (10, 8, or 5 items) yields similar internal consistency, factor structure, and accuracy in detecting depressive symptoms as the full 20-item CES-D (44–46). This short-form of the CES-D is coded according to standard practice yielding an index ranging from 0 to 30, with higher scores indicating more symptoms (47).

An index of stress is based on the respondent's report of whether each of seven situations "make you feel stressed or troubled": the respondent's own health, financial situation, and relations with family members; and his or her family's or children's health, financial situation, job, and marital situation. Each item is coded on a three-point scale (0 = none, 1 = some, 2 = a lot of stress), and the final score is the average for respondents with at least four valid responses.

Social involvement is measured as a count of eight activities in which the respondent reports current membership or participation: neighborhood association, religious association, professional or civic group, social service group, political association, village or lineage association, elderly club,

and elderly education. Socioeconomic status (SES) is measured by how respondents rank themselves relative to other people in Taiwan, as depicted by 10 rungs on a ladder (10 indicates those who are the best off) (48, 49). A previous study (26) as well as our own preliminary analyses indicate that this subjective evaluation is a better predictor of self-assessed health status than standard measures of SES such as education and income.

Analytical Approach

Given the ordinal nature of the outcome variable, we estimate an ordered-probit model (50). The model is estimated in two stages, to address the following questions, respectively: 1) Do clinical risk factors predict subjective self-rated health? and 2) If so, do these variables retain their importance after adjustment for self-reported measures?

Descriptive statistics for all variables are shown in Table 1 and variables included in each of the models are presented in Table 2. Model 1 includes the clinical measures, together with controls for age (linear and quadratic terms) and sex. (Despite the oversampling of urban residents, we do not control for residence because this variable revealed almost no association with self-rated health.) All variables are continuous with the exception of the dummy variable denoting the presence of the $\epsilon 4$ allele. In preliminary models, we tested linear and quadratic terms for each of the clinical variables (except APOE) as well as sex interactions. This decision was motivated by the scientific literature indicating that: 1) both low and high values are associated with poorer health outcomes than moderate values; and 2) the effects of clinical variables differ by sex. We retained only those quadratic terms and sex interactions that were marginally significant ($p < .10$) in at least one of the two models.

Model 2 incorporates the remaining explanatory variables. Initially, we explored additional variables hypothesized to affect self-rated health: education, income, marital status, social ties with family and non-relatives, cognitive function, and locus of control. None of these variables remained significant ($p < 0.05$) in the presence of the other covariates shown in Table 1 and thus, were excluded from the final models. Again, we retained only sex interactions that were marginally significant ($p < .10$).

To demonstrate the magnitude of a given effect, predicted proportions in each category of self-rated health are shown in Table 3 for clinical variables that are significant ($p < .05$) in model 2 and for self-reported measures of physical health. For a given variable, these proportions are calculated using the coefficients from model 2, selecting the 10th, 50th, and 90th percentile values, respectively, of the given variable, and retaining other explanatory variables at observed values.

TABLE 1. Descriptive statistics for all measures

Variable	Mean (standard deviation) or % in category ^c
Self-reported health status	
Poor (%)	3.2
Not so good (%)	23.3
Average (%)	48.3
Good (%)	12.5
Excellent (%)	12.7
Clinical risk factors	
Body mass index (BMI): weight in kg / (height in m) ²	24.52 (3.57)
Waist-hip ratio	0.88 (0.07)
Systolic blood pressure (mmHg)	137.52 (20.53)
Diastolic blood pressure (mmHg)	82.69 (11.27)
Ratio of total cholesterol to HDL cholesterol	4.40 (1.49)
Total glycosylated hemoglobin (HbA _{1c})	5.76 (1.39)
Ratio of urinary cortisol (ug/g creatinine) to DHEA-S ^a (ug/dL)	1.03 (4.09)
Urinary norepinephrine (ug/g creatinine)	21.74 (9.58)
Urinary epinephrine (ug/g creatinine)	2.53 (2.45)
Carries at least one copy of ε4 allele of APOE genotype (%)	15.4
Demographic characteristics	
Age	66.23 (7.96)
Female (%)	42.6
Self-reported measures of physical health	
Number of current illnesses/conditions (0–12)	1.25 (1.25)
Number of mobility limitations (0–9)	1.84 (2.25)
Fall or injury in the past year that caused problems walking or bathing (%)	7.8
Number of long-term medications (0–4)	0.72 (0.86)
Usual level of pain or discomfort ^b	
None (%)	59.8
A little (%)	29.3
Medium level (%)	8.0
More serious, but bearable (%)	2.2
Very serious/unbearable (%)	0.8
Trouble with incontinence ^b (%)	5.0
Number of hospital days in past year	1.55 (5.74)
Psychological well-being	
CES-Depression scale (0–30)	5.36 (5.25)
Stress index (0–2)	0.28 (0.38)
Health-related behaviors	
Eats at least three vegetables and two fruits daily (%)	53.3
Frequency of exercise	
None (%)	38.2
Less than once a week (%)	3.0
Once or twice a week (%)	7.1
Three to five times a week (%)	13.2
Six or more times a week (%)	38.4
Smoked daily in past six months (%)	22.5
Social and socioeconomic variables	
Number of social activities R participates in (0–8)	0.74 (1.09)
Subjective socioeconomic status ladder (1–10, where 10 = Best off)	3.85 (1.86)
Number of cases	928

^aDehydroepiandrosterone sulfate.

^bMeasured in the 1999 survey.

^cThese values have been weighted by age group and urban/rural residence to reflect the sampling design.

RESULTS

The estimates from model 1 reveal that BMI, waist-hip ratio, ratio of total to HDL cholesterol, glycosylated hemoglobin, ratio of cortisol to DHEA-S, epinephrine, and the

ε4 allele are significantly associated with self-rated health (Table 2). All except epinephrine and BMI have negative associations. BMI is the only clinical variable that reveals a non-linear association with self-rated health; consistent with the J- or U-shaped association between BMI and mortality

TABLE 2. Ordered probit models of self-reported health status

Variable	Model 1 ^a	Model 2 ^a
Clinical risk factors		
BMI	0.356***	0.295**
BMI ²	−0.007***	−0.005**
Waist-hip ratio	−2.187**	−1.284 [†]
Systolic blood pressure	0.004	0.002
Diastolic blood pressure	−0.001	0.002
Ratio of total to HDL cholesterol	−0.070*	−0.118**
Female × Ratio of total to HDL cholesterol	0.076	0.127*
Glycosylated hemoglobin	−0.059*	0.008
Ratio of cortisol to DHEA-S	−0.168***	−0.089*
Female × Ratio of cortisol to DHEA-S	0.151***	0.087*
Norepinephrine	0.000	0.002
Epinephrine	0.035*	0.032*
APOE ε4 allele	0.015	−0.069 ^b
Female × APOE ε4 allele	−0.411*	−0.327 ^b
Demographic characteristics		
Age	−0.245***	−0.185*
Age ²	0.002**	0.001**
Female	−0.730**	−0.663*
Self-reported measures of physical health		
Number of current illnesses/conditions	−	−0.140***
Number of mobility limitations	−	−0.109***
Fall or injury in the past year that caused problems walking/bathing	−	−0.332*
Number of long-term medications	−	−0.200***
Usual level of pain or discomfort	−	−0.173**
Trouble with incontinence	−	−0.404*
Number of hospital days in past year	−	−0.015*
Psychological well-being		
CES-Depression scale	−	−0.030***
Stress index	−	−0.377**
Health-related behaviors		
Eats at least three vegetables and two fruits daily	−	0.256**
Frequency of exercise	−	0.069**
Smoked daily in past six months	−	0.327**
Social and socioeconomic variables		
Number of social activities R participates in	−	−0.044
Female × Number of social activities R participates in	−	0.153*
Subjective socioeconomic status ladder	−	0.052*
Number of cases	928	928
Log L	−1183.27	−1003.17
Pseudo R ²	0.04	0.19

[†]p < 0.10, *p < 0.05, **p < 0.01, ***p < 0.001;

^aThe models are based on unweighted data. In model 1, the cutpoints (C1, C2, C3, and C4) are: −8.28, −6.98, −5.62, and −5.10, respectively. In model 2, the cutpoints are −6.25, −4.44, −2.74, and −2.13. These cutpoints assume that the observed response Y_i results from grouping an underlying continuous variable Z_i , where $Z_i = \alpha_i\beta + \epsilon_i$. The observed Y_i takes the value 1 (poor) if $Z_i < C_1$, the value 2 (not so good) if $C_1 < Z_i < C_2$, and so on, taking the value 5 (excellent) if $Z_i > C_4$.

^bAlthough APOE ε4 and the interaction term with sex are not individually significant in model 2, they are jointly significant (p < 0.05 based on likelihood ratio test). Also, if the interaction is excluded, the main effect becomes significant (p < 0.05).

(51, 52), the poorest self-ratings occur at both extremes. Most of these coefficients remain significant after controlling for self-reported indicators (model 2), although the effects are typically smaller. Three clinical variables—diastolic and systolic blood pressure and norepinephrine—are not significant in either model. Glycosylated hemoglobin becomes virtually zero in the presence of controls. (The effect of this biomarker is completely eliminated by a variable indicating whether the respondent reports having diabetes.)

The effects of three clinical measures differ significantly by sex. The ratios of total to HDL cholesterol and cortisol

to DHEA-S have negative effects among men, but virtually no effect for women, whereas the ε4 allele has a significant negative effect only among women.

A large number of self-reported measures are significantly related to self-rated health. For example, number of chronic diseases, mobility limitations, and level of pain are negatively related to the ratings as are the indexes of depression and stress. An auxiliary model (not shown) that included each of the 10 components of the CES-D revealed that the effect of the CES-D score is dominated by two components—sleeping poorly and being in a bad mood. A healthy diet and frequent

TABLE 3. Predicted proportions in each category of self-reported health status by selected covariates

Variable	Poor	Not so good	Average	Good	Excellent
BMI					
10 th percentile (20.1)	0.05	0.27	0.48	0.11	0.09
50 th percentile (24.1)	0.03	0.22	0.48	0.14	0.13
90 th percentile (28.9)	0.03	0.20	0.47	0.14	0.15
Waist-hip ratio					
10 th percentile (0.80)	0.03	0.21	0.48	0.14	0.14
50 th percentile (0.88)	0.04	0.23	0.48	0.13	0.12
90 th percentile (0.96)	0.04	0.25	0.48	0.12	0.11
Ratio of total to HDL cholesterol					
Males					
10 th percentile (2.79)	0.03	0.19	0.47	0.15	0.16
50 th percentile (4.32)	0.04	0.22	0.48	0.13	0.12
90 th percentile (6.17)	0.06	0.26	0.48	0.11	0.09
Females					
10 th percentile (2.86)	0.04	0.23	0.48	0.13	0.12
50 th percentile (4.09)	0.04	0.23	0.48	0.13	0.12
90 th percentile (6.05)	0.03	0.23	0.48	0.13	0.13
Ratio of cortisol to DHEA-S					
Males					
10 th percentile (0.07)	0.03	0.22	0.48	0.13	0.13
50 th percentile (0.20)	0.03	0.22	0.48	0.13	0.12
90 th percentile (0.82)	0.03	0.23	0.48	0.13	0.12
Females					
10 th percentile (0.16)	0.04	0.23	0.48	0.13	0.12
50 th percentile (0.43)	0.04	0.23	0.48	0.13	0.12
90 th percentile (2.51)	0.04	0.23	0.48	0.13	0.12
Epinephrine					
10 th percentile (0.0)	0.04	0.24	0.48	0.12	0.11
50 th percentile (2.10)	0.04	0.23	0.48	0.13	0.12
90 th percentile (5.62)	0.03	0.21	0.48	0.14	0.14
APOE ε4 allele					
Males					
No	0.04	0.22	0.48	0.13	0.12
Yes	0.05	0.24	0.48	0.12	0.11
Females					
No	0.03	0.22	0.48	0.14	0.13
Yes	0.05	0.29	0.48	0.10	0.08
Number of current illnesses/conditions					
10 th percentile (0)	0.02	0.19	0.49	0.15	0.15
50 th percentile (1)	0.03	0.22	0.50	0.13	0.12
90 th percentile (3)	0.04	0.28	0.50	0.11	0.08
Number of mobility limitations					
10 th percentile (0)	0.02	0.19	0.50	0.15	0.14
50 th percentile (1)	0.02	0.21	0.51	0.14	0.12
90 th percentile (6)	0.05	0.33	0.48	0.08	0.05
Fall or injury that caused problems					
No	0.03	0.23	0.48	0.13	0.12
Yes	0.05	0.29	0.48	0.10	0.08
Number of long-term medications					
10 th percentile (0)	0.02	0.20	0.49	0.14	0.14
50 th percentile (1)	0.03	0.24	0.50	0.12	0.11
90 th percentile (2)	0.04	0.28	0.49	0.11	0.08
Usual level of pain or discomfort					
10 th & 50 th percentile (none)	0.03	0.21	0.49	0.14	0.13
90 th percentile (medium)	0.04	0.28	0.49	0.11	0.08
Trouble with incontinence					
No	0.03	0.23	0.48	0.13	0.12
Yes	0.06	0.30	0.47	0.10	0.07
Number of hospital days in past year					
10 th & 50 th percentile (0)	0.03	0.23	0.48	0.13	0.12
90 th percentile (5)	0.04	0.24	0.48	0.13	0.11

Probabilities are based on model 2 of Table 2 and calculated assuming selected values of covariates leaving all other covariates at observed values.

exercise are associated with better health ratings. However, contrary to expectation, so is daily smoking, which may result from the relatively early deaths of the least healthy heavy smokers. Respondents with higher subjective assessments of their social position and women (but not men) who participate in more social activities also rate themselves as healthier than their counterparts (Table 2).

The magnitude of some of these effects can be gleaned from Table 3. These simulated proportions reveal the substantial impact of cholesterol for men (e.g., 31% of men at the 10th percentile of the total-to-HDL cholesterol ratio rate their health as good or excellent in contrast to only 20% of men at the 90th percentile). Large effects are also associated with the BMI: below the 90th percentile, higher values are associated with more favorable ratings, whereas above 29 (not shown), higher values are associated with progressively poorer ratings. The effect of the ϵ 4 allele is moderate among females, but virtually nil among men. Despite being statistically significant for men, the cortisol/DHEA-S ratio has little substantive effect.

The predicted proportions associated with self-reported measures of physical health generally reveal larger impacts than those for the clinical variables. For example, 29% of those at the 10th percentile of mobility limitations rate their health as good or excellent compared with 13% at the 90th percentile. Number of chronic conditions, a debilitating fall or injury in the prior year, medication use, level of pain, and incontinence also show substantial effects. Only the hospitalization variable appears to have a negligible impact.

DISCUSSION

Are clinical risk factors related to subjective health ratings? This analysis suggests that the simple answer is “yes.” More complex questions pertain to why and by how much these measures influence health assessments.

With regard to why, the answer almost certainly differs across measures. For example, poorer assessments associated with low BMI may be largely due to weight loss resulting from illness (not fully accounted for by the model), whereas those related to high values of BMI or waist-hip ratio probably emanate from the reverse mechanism (the effects of obesity on well-being). Obese individuals may also incorporate the health risks associated with obesity in their assessments, independent of symptoms and conditions.

Results for high cholesterol and hypertension further suggest that individuals incorporate information about risk into self-ratings. For high cholesterol, which in and of itself is typically asymptomatic, the strong association with self-rated health—even in the presence of controls for heart

disease—suggests that the diagnosis independently influences the self-ratings. This interpretation is supported by analyses of hypertension not presented here. The lack of a significant association between blood pressure and the self-ratings in Table 2 probably results from the finding that many Taiwanese with high blood pressure readings do not acknowledge their condition in the interview, most likely because they have not received a diagnosis (33). However, a variable denoting whether the respondent has been diagnosed with hypertension is significantly associated with self-rated health, even after controlling for heart disease.

The underlying mechanisms linking self-ratings to the presence of the ϵ 4 allele, the cortisol/DHEA-S ratio, and epinephrine are less obvious. Because respondents are unlikely to be aware of their measurement on these markers, the effects must operate through some other aspects of well-being. Yet, many of the major conditions associated with these variables are either controlled in the model (e.g., heart disease and depression) or were excluded because they had no significant effect (e.g., cognitive function). Thus, it is impossible to determine whether the apparent effects of these biomarkers reflect the inadequacy of self-reported measures, or whether these biomarkers affect health outcomes not included in the interview. The results for epinephrine and APOE are particularly intriguing. For epinephrine, low but not high values are associated with poor self-ratings. Although much of the literature linking stress and SNS activity identifies deleterious effects associated with high levels of norepinephrine and epinephrine (34, 53), low levels of epinephrine, which may denote an inadequate response to stressful challenges, are associated with depression and mortality (54, 55). Data not shown here indicate that epinephrine has a curvilinear relationship with the CES-D; both low and high values are associated with more depressive symptoms. Nonetheless, controlling for the CES-D did little to diminish the effect of epinephrine on self-ratings (model 2). Thus, either our measures have not completely captured the effects of epinephrine on mental health or epinephrine operates through factors not included in the model.

Our model also does little to explain the effect of APOE on self-assessed health. Thus, these results suggest that, at least among women, the negative impact of the ϵ 4 allele may extend beyond the two conditions (heart disease and Alzheimer’s disease) most frequently identified (56).

Consistent with previous research suggesting that men and women may use different criteria for evaluating their overall health (2), our results identify several large and significant sex differences. However, we can only speculate about their underlying causes. For some biomarkers, these results may reflect a sex difference in knowledge about one’s level of risk or in a perception of the likelihood of disease (e.g., cholesterol). For example, men may place more importance

on cholesterol levels than women, perhaps because of the perception that cardiovascular disease is predominately a male illness. The greater effect of the cortisol/DHEA-S ratio among men may result from larger negative effects of cortisol or larger positive effects of DHEA-S on health among men than women. The latter is consistent with the literature on DHEA-S (57). Moreover, our results indicate that in the presence of controls for health measures, the effects of the cortisol/DHEA-S ratio are greatly diminished.

Although this analysis has demonstrated the importance of clinical measures for self-rated health, the results also suggest that the clinical measures are less powerful predictors than self-reports about diverse aspects of well-being. Similarly, another study found that self-rated morbidity was more important than physician-rated morbidity in predicting self-rated health (30). This result is not altogether surprising given that self-rated health status is also subjective: we are trying to account for a person's *perception* of their own health rather than some objective measure of their health status. Thus, rather than enhance our efforts to collect so-called objective measures through physician's reports and biomarkers, we may need to focus on aspects of well-being that are notoriously difficult to measure, such as: mental and emotional health; limitations imposed by health conditions; personality characteristics, including optimism, perseverance, and coping strategies; and the quality of social relationships. As noted by Ferraro and Farmer, although researchers need to "remain vigilant about issues of data quality," "self-reported data should not axiomatically be characterized as inferior solely because they come from respondents" (30).

An important limitation of this study is the cross-sectional nature of the data, which limits inferences about how respondents use clinical and other criteria to assess their overall health. Recent efforts to incorporate biological measurements into large-scale social and health surveys may soon provide *longitudinal* information on clinical measures, self-reported indicators of physical and psychological well-being, and relevant environmental variables that will enable the analyst to further elucidate the processes by which respondents evaluate their own health. Such future research may also help to solve the puzzles raised in this article, namely, what are the mechanisms that give rise to: 1) the large sex differences in the effects of the clinical measures on self-assessed health; and 2) the influence of clinical measures about which respondents are unlikely to have information regarding their measurements.

We gratefully acknowledge support for this project from the Demography and Epidemiology Unit of the Behavioral and Social Research Program of the National Institute of Aging (grants R01AG16790 and R01AG16661) and the National Institute of Child Health and Human Development (grant 5P30HD32030). We would also like to thank Maxine

Weinstein, Teresa Seeman, Marion Carter, and Germán Rodríguez for their helpful comments.

REFERENCES

1. Mossey JM, Shapiro E. Self-rated health: a predictor of mortality among the elderly. *Am J Public Health*. 1982;72(8):800–808.
2. Idler EL, Benyamini Y. Self-rated health and mortality: a review of twenty-seven community studies. *J Health Soc Behav*. 1997;38:21–37.
3. Benyamini Y, Idler EL. Community studies reporting association between self-rated health and mortality. *Res Aging*. 1999;21(3):392–401.
4. Grand A, Grosclaude P, Bocquet H, Pous J, Albaredo JL. Predictive value of life events, psychological factors and self-rated health on disability in an elderly rural French population. *Soc Sci Med*. 1988;27(12):1337–1342.
5. Jagger C, Spiers NA, Clarke M. Factors associated with decline in function, institutionalization and mortality of elderly people. *Age Ageing*. 1993; 22(3):190–197.
6. Kaplan GA, Strawbridge WJ, Camacho T, Cohen RD. Factors associated with change in physical functioning in the elderly: a six-year prospective study. *J of Aging and Health*. 1993;5:40–53.
7. Mor V, Wilcox V, Rakowski W, Hiris J. Functional transitions among the elderly: patterns, predictors, and related hospital use. *Am J Public Health*. 1994;84(8):1274–1280.
8. Haga H, Shibata H, Suyama Y, Suzuki K, Iwasaki K, Suzuki T, et al. Self-rated health as a predictor of active life in the community elderly. *Japanese Journal of Epidemiology*. 1995;5:11–15.
9. Idler EL, Kasl S. Self-ratings of health: do they also predict change in functional ability? *J Gerontol B Psychol Sci Soc Sci*. 1995;50(6):S344–S353.
10. Ferraro KF, Farmer MM, Wybraniec JA. Health trajectories: long-term dynamics among black and white adults. *J Health Soc Behav*. 1997; 38(1):38–54.
11. Shadbolt B. Some correlates of self-rated health for Australian women. *Am J Public Health*. 1997;87(6):951–956.
12. Møller L, Kristensen TS, Hallnagel H. Self-rated health as a predictor of coronary heart disease in Copenhagen, Denmark. *J Epidemiol Community Health*. 1996;50(4):423–428.
13. Idler EL. Age differences in self-assessments of health: age changes, cohort differences, or survivorship? *J Gerontol*. 1993;48(6):S289–S300.
14. Wolinsky FD, Stump TE, Johnson RJ. Hospital utilization profiles among older adults over time: consistency and volume among survivors and decedents. *J Gerontol B Psychol Sci Soc Sci*. 1995;50(2):S88–100.
15. Benyamini Y, Idler EL, Leventhal H, Leventhal EA. Positive affect and function as influences on self-assessments of health: expanding our view beyond illness and disability. *J Gerontol B Psychol Sci Soc Sci*. 2000; 55(2):P107–P116.
16. Krause NM, Jay GM. What do global self-rated health items measure? *Med Care*. 1994;32(9):930–942.
17. Carlson P. Risk behaviours and self-rated health in Russia 1998. *J Epidemiol Community Health*. 2001;55(11):806–817.
18. Fylkesnes K, Førde OH. Determinants and dimensions involved in self-evaluation of health. *Soc Sci Med*. 1992;35(3):271–279.
19. Manor O, Matthews S, Power C. Self-rated health and limiting longstanding illness: inter-relationships with morbidity in early adulthood. *Int J Epidemiol*. 2001;30(3):600–607.
20. Kawachi I, Kennedy BP, Glass R. Social capital and self-rated health: a contextual analysis. *Am J Public Health*. 1999;89(8):1187–1193.
21. Manderbacka K, Lahelma E, Martikainen P. Examining the continuity of self-rated health. *Int J Epidemiol*. 1998;27(2):208–213.
22. Linn BS, Linn MW. Objective and self-assessed health in the old and very old. *Soc Sci Med*. 1980;14A(4):311–315.

23. Baron-Epel O, Kaplan G. General subjective health status or age-related subjective health status: does it make a difference? *Soc Sci Med*. 2001; 53(10):1373–1381.
24. Bobak M, Pikhart H, Hertzman C, Rose R, Marmot M. Socioeconomic factors, perceived control and self-reported health in Russia. A cross sectional study. *Soc Sci Med*. 1998;47(2):269–279.
25. Zimmer Z, Natividad J, Lin HS, Chayovan N. A cross-national examination of the determinants of self-assessed health. *J Health Soc Behav*. 2000;41(4): 465–481.
26. Ostrove JM, Adler NE, Kupperman M, Washington AE. Objective and subjective assessments of socioeconomic status and their relationship to self-rated health in an ethnically diverse sample of pregnant women. *Health Psychol*. 2000;19(6):613–618.
27. Idler EL, Angel RJ. Self-rated health and mortality in the NHANES-I Epidemiologic Follow-up Study. *Am J Public Health*. 1990;80(4):446–452.
28. Harlow SD, Linet MS. Agreement between questionnaire data and medical records. The evidence for accuracy of recall. *Am J Epidemiol*. 1989;129(2):233–248.
29. Martin LM, Leff M, Calonge N, Garrett C, Nelson DE. Validation of self-reported chronic conditions and health services in a managed care population. *Am J Prev Med*. 2000;18(3):215–218.
30. Ferraro KF, Farmer MM. Utility of health data from social surveys: is there a gold standard for measuring morbidity? *Am Sociol Rev*. 1999;64:303–315.
31. Leinonen R, Heikkinen E, Jylhä M. Predictors of decline in self-assessments of health among older people – a 5-year longitudinal study. *Soc Sci Med*. 2001;52(9):1329–1341.
32. Hermalin AI, Liang J, Chang M-C. 1989 survey of health and living status of the elderly in Taiwan: questionnaire and survey design. Comparative study of the elderly in Asia research report 89-1. Ann Arbor, MI: PSC Publications, University of Michigan; 1989.
33. Goldman N, Lin IF, Weinstein M, Lin YH. Evaluating the quality of self-reports of hypertension and diabetes. Princeton, NJ: Princeton University; 2002, OPR Working Paper 2002–03.
34. Seeman TE, Singer BH, Rowe JW, Horwitz RI, McEwen BS. Price of adaptation—allostatic load and its health consequences. *MacArthur studies of successful aging*. *Arch Intern Med*. 1997;157(19):2259–2268.
35. Singer B, Ryff CD. Hierarchies of life histories and associated health risks. *Ann N Y Acad Sci*. 1999;896:96–115.
36. Bjorntorp P. The associations between obesity, adipose tissue distribution and disease. *Acta Med Scand*. 1988;723:121–134.
37. Koenig RJ, Peterson CM, Jones RL, Saudek C, Lehrman M, Cerami A. Correlation of glucose regulation and hemoglobin A_{1c} in diabetes mellitus. *N Engl J Med*. 1976;295:417–420.
38. Dunn PJ, Cole RA, Soeldner JS, Gleason RE, Kwa E, Firoozabadi H, et al. Temporal relationship of glycosylated haemoglobin concentrations to glucose control in diabetics. *Diabetologia*. 1979;17:213–220.
39. Davidson M, Schriger D, Peters A, Lorber B. Relationship between fasting plasma glucose and glycosylated hemoglobin. Potential for false-positive diagnoses of type 2 diabetes using new diagnostic criteria. *JAMA*. 1999;281:1203–1210.
40. Goodyer IM, Herbert J, Altham PM. Adrenal steroid secretion and major depression in 8- to 16-year olds, III. Influence of cortisol/DHEA ratio at presentation on subsequent rates of disappointing life events and persistent major depression. *Psychol Med*. 1998;28(2):265–273.
41. Lupien SJ, Nair NP, Briere S, Maheu F, Tu MT, Lemay M, et al. Increased cortisol levels and impaired cognition in human aging: implication for depression and dementia in later life. *Rev Neurosci*. 1999;10(2):117–139.
42. Vanitallie TB. Stress: a risk factor for serious illness. *Metabolism*. 2002;51(6 suppl. 1):40–45.
43. Ewbank D. The genetic make-up of population and its implications for mortality by cause of death: links between Alzheimer's and ischaemic heart disease. In: *Health and Mortality: Issues of Global Concern*. New York: United Nations, Department of Economic and Social Affairs, Population Division; 1999:344–356.
44. Boey KW. Cross-validation of a short form of the CES-D in Chinese elderly. *Int J Geriatr Psychiatry*. 1999;14(8):608–617.
45. Rouch-Leroyer I, Sourgen C, Barberger-Gateau P, Fuhrer R, Dartigues JF. Detection of depressive symptomatology in elderly people: a short version of the CES-D scale. *Aging (Milano)*. 2000;12(3):228–233.
46. Turvey CL, Wallace RB, Herzog R. A revised CES-D measure of depressive symptoms and a DSM-based measure of major depressive episodes in the elderly. *Int Psychogeriatr*. 1999;11(2):139–148.
47. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psych Meas*. 1977;1:385–401.
48. Adler NE, Epel ES, Castellazzo G, Ickovics JR. Relationship of subjective and objective social status with psychological and physiological functioning: preliminary data in healthy white women. *Health Psychol*. 2000;19(6): 586–592.
49. Goodman E, Adler NE, Kawachi I, Frazier AL, Huang B, Colditz GA. Adolescents' perceptions of social status: development and evaluation of a new indicator. *Pediatrics*. 2001;108(2):E31(1–8).
50. StataCorp. Stata Statistical Software: Release 7.0, Reference H-P. College Station, TX: Stata Corporation; 2001;469–473.
51. Allison DB, Faith MS, Hao M, Koler DP. Hypothesis concerning the U-shaped relation between body mass index and mortality. *Am J Epidemiol*. 1997;146(4):339–349.
52. Lee IM, Manson JE, Hennekens CH, Paffenbarger RS Jr. Body weight and mortality. A 27-year follow-up of middle-aged men. *JAMA*. 1993;270(23): 2823–2828.
53. Weiner H. *Perturbing the organism: the biology of stressful experience*. Chicago: The University of Chicago Press; 1992.
54. Christensen NJ, Jensen EW. Sympathoadrenal activity and psychosocial stress. The significance of aging, long-term smoking, and stress models. *Ann N Y Acad Sci*. 1995;771:640–647.
55. Gjerris A, Rafaelsen OJ, Christensen NJ. CSF-adrenaline—low in somatizing depression. *Acta Psychiatr Scand*. 1987;75(5):516–520.
56. Schmidt S, Barcellos LF, DeSombre K, Rimmler JB, Lincoln RR, Bucher P, et al. Association of polymorphisms in the apolipoprotein E region with susceptibility to and progression of multiple sclerosis. *Am J Hum Genet*. 2002;70(3):708–717.
57. Kroboth PD, Salek FS, Pittenger AL, Fabian TJ, Frye RF. DHEA and DHEA-S: A review. *J Clin Pharmacol*. 1999;39:327–348.