

Do Biomarkers of Stress Mediate the Relationship  
between Socioeconomic Status and Health?

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**Summary:****Background:**

Psychosocial stress is posited as one of the primary pathways linking socioeconomic status (SES) to health outcomes, via sustained activation of stress-related autonomic and neuroendocrine responses, especially elevated levels of cortisol. To date, little population-level work has tested the relationship between SES and biological markers of these stress responses.

**Methods:**

We analyzed data from a national survey of 972 middle-aged and elderly respondents in Taiwan including survey, clinical, and biological measures. We tested the relationships between SES, as measured by education and income, and 13 biomarkers representing functioning of the neuroendocrine system, immune/inflammatory systems, and the cardiovascular system. We also examined whether these biomarkers account for the observed relationship between SES and self-reported health and mobility difficulties in our sample.

**Findings:**

Lower SES men have greater odds of falling into the highest risk quartile for only 2 of 13 biomarkers, and show a *lower* risk for 3 of the 13 biomarkers, with no association between SES and cortisol. Lower SES women have a higher risk for many of the cardiovascular risk factors, but a lower risk for elevated readings of epinephrine, norepinephrine, and cortisol. Inclusion of all 13 biological markers does not explain the relationship between SES and health outcomes in our sample.

**Interpretation:**

These data do not support the hypothesis that stress, via sustained activation of the body's stress response, is an important mediator in the relationship between socioeconomic status and health. Most notably, lower SES is not associated with higher levels of cortisol in either men or women.

## ***Introduction***

Much attention has been paid to the relationship between socioeconomic status (SES) and health, but the mechanisms linking the social and the physical are not well understood. With the seeming inadequacy of either differences in medical care or health-related behaviors to explain the gradient, psychosocial stress has emerged as a leading contender for translating low social status into poor health (1-3). It is postulated that lower status individuals are more likely to experience both chronic and acute stress in their lives, and numerous studies have provided empirical support for the idea that lower socioeconomic status is associated with more reported life stress (4-6). Studies of non-human primates have shown that lower status animals often show elevated basal cortisol levels, lower levels of HDL cholesterol, more signs of coronary heart disease, and more susceptibility to infection (7-10). Proponents of the idea that stress mediates the SES-health relationship suggest that the physical pathways through which low status harms health operate in much the same way in humans as they do in other primates. It is postulated that the experience of low social status elicits sustained activation of stress-related autonomic and neuroendocrine responses, with elevated levels of cortisol the most commonly mentioned mechanism through which low status damages health (11,12,13).

While substantial work has linked lower SES to several cardiovascular risk factors such as blood pressure, waist-to-hip ratio, cholesterol, and fibrinogen (14), much less work has related neuroendocrine markers of stress to SES in humans, and the results have been mixed. An analysis of 200 Whitehall participants found that resting blood pressure, heart rate, and salivary cortisol do not differ by employment grade, while average cortisol over the workday was significantly higher for lower grade men but significantly lower for lower grade women (15). A study of 767 adults in Germany found *positive* associations between morning salivary cortisol

concentrations and levels of education and occupational status (16), while a study of 150 men from Lithuania and Sweden found that low social class was related to high early morning levels of salivary cortisol (17). In a sample of 217 Canadian children, children of low socioeconomic status were found to have significantly higher morning salivary cortisol levels than children with high socioeconomic status (18).

Recent work has utilized the concept of allostatic load (AL), which refers to the cumulative physiological toll exacted on the body across multiple systems through repeated or chronic environmental challenges (19-21). Findings suggest that higher allostatic load is associated with lower levels of education in the MacArthur Studies of Successful Aging, and that this association accounts for roughly a third of the relationship between education and mortality in that sample. Importantly however, this relationship seems to be driven primarily by the cardiovascular risk components and inflammatory markers comprising allostatic load rather than the stress hormones (22, 23). Most of these studies have come from small or specially selected samples that may not be representative of the population at large, and all have been from Western populations. This paper will test the relationship between SES and a broad set of markers that have been proposed to reflect the physiological effects of stress in representative sample of middle-aged and elderly persons in Taiwan. We then examine whether these biomarkers can explain part or all of the observed relationship between SES and health outcomes. The biomarkers include measures of sympathetic nervous system (SNS) and hypothalamic pituitary–adrenal (HPA) axis functioning, immune/inflammatory markers, and cardiovascular disease risk factors.

## ***Methods***

### *Study Population*

Data for this study come from the 2000 Social Environment and Biomarkers Aging Study (SEBAS), made up of a random sub-sample of the participants in the on-going Survey of Health and Living Status of the Near Elderly and Elderly in Taiwan. This longitudinal survey began in 1989 with a nationally representative sample, including the institutionalized population, of persons 60 years and older. The survey was expanded in 1996 to include a new sample of middle-aged (aged 50 to 66) persons. In SEBAS, elderly respondents (71 and older in 2000) were over sampled relative to the near elderly (54 to 70 in 2000), as were persons in urban areas. The survey procedures were approved by the institutional review boards at Princeton University, Georgetown University, and the Bureau of Health Promotion, Department of Health, Taiwan, and conformed to the principles embodied in the Declaration of Helsinki. Among the 1713 respondents randomly selected, a total of 1497, constituting 92% of the survivors, were interviewed. Of this group, 1023 completed the physical exam portion of the survey that included physician evaluation and collection of blood and urine samples. Excluding respondents with missing data on one or more variables of interest leaves a final sample of 972. Older respondents were less likely to participate in the exam portion of the survey, but measures of socioeconomic status were not significantly related to participation. Because of higher non-participation rates among both the healthiest and the least healthy individuals, persons who received the medical exam reported the same general health status, on average, as those who did not. Each participant in the medical exam was asked to fast overnight, collect a 12-hour overnight urine sample (for integrated measures of neuroendocrine function), and proceed to the

medical exam the next morning at a nearby hospital where a physician or nurse drew blood from the participant and took blood pressure and other measures.

### *Measures*

As mentioned above, the concept of allostatic load underscores the notion that while mobilization of the body's stress response is necessary for short-term response to stimuli, excessive or overly frequent mobilization of these resources can damage the body's ability to regulate these systems and ultimately cause disease. Our biomarkers include the ten measures used in the first empirical implementation of allostatic load: serum dihydroepiandrosterone sulfate (DHEA-S, a functional HPA axis antagonist), urinary cortisol (an integrated measure of 12-hr HPA axis activity), urinary epinephrine and norepinephrine (integrated measures of 12-hr SNS activity), systolic and diastolic blood pressures (indices of cardiovascular activity), waist-hip ratio (an index of metabolism and adipose tissue deposition), serum HDL cholesterol, ratio of total to HDL serum cholesterol (indices of risk for cardiovascular disease), and blood plasma levels of glycosylated hemoglobin (HbA1c, an integrated measure of glucose metabolism over the previous 30-90 days). Recent work has expanded the empirical measure of allostatic load to include information on inflammatory markers and immune function (21). We include three additional markers meant to capture dysregulation in immune/inflammatory function: interleukin-6 (IL-6, a pro-inflammatory cytokine), insulin-like growth factor-1 (IGF-1, aids in muscle growth and bone repair), and albumin (low levels associated with inflammation). Measures of cortisol, epinephrine, and norepinephrine were derived from 12-hour overnight urine specimens and are reported as "micrograms per gram creatinine" to adjust for body size. Measures of DHEA-S, IGF-1, IL-6, albumin, ratio of total to HDL cholesterol, HDL cholesterol,

and glycosylated hemoglobin were taken from fasting blood specimens. Systolic and diastolic blood pressures were calculated using the average of two seated blood pressure readings taken about a minute apart, and waist-hip ratio was calculated based on waist circumference (measured at its narrowest point between the ribs and iliac crest) and hip circumference (measured at the maximal buttocks). Blood and urine samples were analyzed by Union Clinical Laboratories (UCL) in Taipei, with duplicate samples for a 10% subset of the specimens submitted to UCL and Quest Diagnostics in the U.S. for analysis. Intraclass correlations of .80 or higher were found for duplicates sent to UCL and interlaboratory correlations of 0.76 or higher for results sent to both laboratories (24).

Individual biomarkers are coded as 1 when the respondent falls in the highest risk quartile of the distribution of that biomarker (highest risk quartiles can be either high or low depending on the biomarker). Table 1 gives cut-off values for each biomarker. Allostatic load is scored as the number of risk factors for which the respondent falls in the highest risk quartile. Allostatic load, based on the original ten biomarkers, was found to be a strong predictor of new cardiovascular events, decline in cognitive and physical functioning, and mortality in a seven-year follow-up in a sample of the elderly from the MacArthur Successful Aging Study, even controlling for health status and other factors at baseline. This was true even when the risk factors were not individually predictive. Other criteria for calculating allostatic load, such as a stricter bottom 10% cut-off for scoring or the use of average z-scores for each parameter, yielded similar results in regard to health decline, with the quartile cut-off criteria showing the strongest effect (20, 25, 26). Three sub-scales will also be used to look at the separate effects of SNS and HPA axis functioning (cortisol, epinephrine, norepinephrine and DHEA-S);

immune/inflammatory markers (IL-6, IGF-1, and albumin); and cardiovascular risk factors (blood pressure and cholesterol measures, waist-hip ratio, HbA1c). As with the overall AL score, these sub-scales count the number of respective biomarkers for which the respondent is in the highest risk quartile.

Socioeconomic status (SES) is measured by education and income. Education is divided into three categories: no formal education, primary education, or secondary education. Income is measured as the respondent and spouse's reported income in 1999 and is divided into quartiles.

Health outcomes include self-reported health on a 5-point scale, with 1=excellent, 2=good, 3=average, 4=not so good, 5=poor, as well as the number (0-6) of mobility difficulties the respondent reports with regard to the following activities: squatting, walking up 2-3 flights of stairs, lifting or carrying 11-12kg, walking 200-300 meters, standing continuously for 15 minutes, and grasping or turning objects with fingers.

### *Statistical Analysis*

Analyses are run separately on men and women, due to potentially important sex differences in the biology of stress (27,28). For individual biomarkers, logistic regression models were used to calculate the odds ratios of falling into the highest risk quartile of each biomarker. For allostatic load, the three biomarker sub-scales, self-reported health status and the number of mobility difficulties, ordinal logit models were used to calculate the odds ratios of moving one point higher on each of the scales. Each model includes a control for the age of the respondent. Education and income are included jointly in all models to estimate their

independent effects. Running models with education and income included separately yielded identical substantive results on all but one biomarker for men (noted below). All analyses were performed using STATA version 8.0 (StataCorp, College Station, TX).

## ***Results***

Tables 2 and 3 show the associations between education, income and the physiological measures obtained during the examination and laboratory portions of SEBAS, separately for each biomarker and sex. Table 2, which presents estimates for the neuroendocrine and immune/inflammatory markers, indicates that education is significantly associated with more favorable readings for men for only one of the four indicators of SNS and HPA axis activity, DHEA-S, and one of the three immune/inflammatory markers, IL-6 (when education is included without income, higher education is associated with more favorable readings for IGF-1). Somewhat counter-intuitively, higher levels of education are significantly associated with higher readings for epinephrine. Income is not significantly related to any of the neuroendocrine or immune/inflammatory markers in men. Table 2 also reveals some unexpected results for women, with more education significantly associated with more favorable (lower) readings for DHEA-S but less favorable (higher) readings for epinephrine, and interestingly, cortisol. No significant associations are found for income.

Table 3, which examines the cardiovascular measures, reveals some unexpected findings for men. More education is significantly related to higher readings of glycosylated hemoglobin, while higher income is significantly associated with higher diastolic blood pressure. The results for women are more consistent with expectation: additional years of education are associated with a lower risk of unfavorable readings for waist-hip ratio, diastolic blood pressure, and glycosylated hemoglobin.

Table 4 presents odds ratios pertaining to the biomarker scales. The results indicate that lower education is significantly associated with worse scores in men only for the collective immune/inflammatory markers, with no relationship seen for neuroendocrine markers,

cardiovascular risk factors, or the overall allostatic load score. For women, less education is significantly associated with higher scores for the cardiovascular risk factors as well as allostatic load, but is not significantly associated with the stress hormone or immune/inflammation sub-scales by themselves, consistent with the findings for the individual biomarkers.

Next, we test whether these biomarkers can account for all or part of the relationship between SES and health outcomes in our sample. Controlling only for age, we see a strong and consistent association between SES and health outcomes in the SEBAS sample (Model 1, Table 5). For men, higher education levels are associated with better self-reported health status as well as fewer mobility restrictions, while more income is independently associated with fewer mobility restrictions. For women, a higher level of income is significantly related to both health measures, and more education is associated with better self-reported health. Our sample is thus consistent with the large literature in many countries finding significant relationships between various measures of socioeconomic status and health outcomes.

Model 2 in Table 5 shows results adjusting for the three biomarker sub-scales, whereas Model 3 adjusts for the summary index of allostatic load (the estimates in Model 2 are similar to those controlling for each individual biomarker). We see that inclusion of the biomarker scales does not diminish the observed effect of education and income on these health outcomes for either men or women. These results are not surprising given the weak associations between education, income, and the biomarkers presented in our previous tables.

## *Discussion*

Overall, these results provide only weak evidence for the idea that stress, particularly through sustained activation of the autonomic and neuroendocrine responses, is one of the primary pathways linking socioeconomic status to health outcomes. While lower education is significantly related to several risk factors for women, these are not the stress hormones through which the story of psychosocial stress is so often told. Associations between SES and cardiovascular measures in women could be confounded by other risk factors including poor nutrition, inactivity and smoking, which are known to increase cardiovascular risk. While the relationships between education and IL-6 and DHEA-S are intriguing, the lack of associations in the expected direction between SES and the markers of HPA-axis and SNS activity leaves us without a smoking gun linking education or income to what are deemed the primary mediators of the body's stress response.

Most notably, lower SES is not significantly related to higher levels of cortisol, the stress hormone most commonly implicated in the literature on SES, stress and health. For the stress hypothesis to remain viable, researchers would need to observe consistent evidence of sustained activation of the HPA-axis and SNS in low SES individuals. The current study finds very mixed evidence of increased physiological dysregulation due to stress in lower SES individuals, and no evidence that biological markers of stress explain the SES gradient in health. These results are partially inconsistent with those of Seeman and colleagues, whose analysis of the MacArthur Study of Successful Aging suggests that allostatic load explains roughly a third of the association between education and seven-year mortality in their sample (20). While their study examines the relationship between education and the summary measure of allostatic load, it does not test education's association with individual biomarkers or categories of biomarkers. Their results for

mortality reveal that the attenuation of the effect of education on mortality is largely driven by the effects of the cardiovascular risk factors and immune/inflammatory markers, with the neuroendocrine markers accounting for only a 4% attenuation. Thus while there is mounting evidence that lower socioeconomic status is associated with biological dysregulation in several systems including the cardiovascular system, metabolic functions, and inflammation processes, evidence linking SES to dysregulation in the SNS and HPA axis is scarce.

Potential limitations to this work include the fact that this is a middle-aged and elderly sample, representing cohorts with low average education and comprised of many retirees. If the relationship between psychosocial stress and SES occurs primarily through characteristics of one's job (29, 30), we may not see strong links between neuroendocrine markers and SES in our sample. Additionally, income may be a less reliable measure of social status in a retired population. Despite these potential problems, we do see the typical health gradients by education and income in our sample, suggesting that education and income are measuring important differences in SES. Furthermore, since income is correlated over time and the effects of SNS and HPA-axis dysregulation are cumulative, we should expect these relationships to be present even in non-working populations if they are indeed the primary driver of SES gradients in health.

It is also possible that while the SES gradients in health seen here are similar to those in other countries, the pathways linking SES and health may be different in Taiwan, where cultural norms regarding social relationships and status are distinct from Western countries (31). There is also recent work suggesting that both very high and very low values of the biological parameters comprising allostatic load, including cortisol, are important for health outcomes (24). Future work would benefit from looking in more detail at the relationship between SES and both

extremes of the distribution of neuroendocrine markers. Another potential limitation of this work is the contemporaneous measurement of biomarkers and health outcomes, which makes it difficult to identify the direction of the relationship between the two. Availability of follow-up health and mortality data for SEBAS will mitigate this problem in the future. For the present analysis, however, the first-stage relationships between education, income, and the biomarkers are so weak that it would be very surprising for these biomarkers to explain the relationship between SES and future health outcomes. Finally, research into the dynamic and complex interactions comprising the body's stress response is on-going; there is much we do not know, or do not know how to measure. Future research should incorporate new knowledge of the physiological processes and measurement of stress, so that a better consensus regarding the role of stress in the SES-health gradient can be found.

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**Table 1: Cut-off points for highest risk quartile of individual biomarkers in 2000 SEBAS**

<b>Biomarker</b>	<b>Cut-point</b>	<b>Mean (SD)</b>
<b><i>Stress Hormones</i></b>		
Cortisol (µg/g creatinine)	≥ 30.0	28.63 (53.05)
Epinephrine (µg/g creatinine)	≥ 3.7	2.65 (2.64)
Norepinephrine (µg/g creatinine)	≥ 27.1	21.83 (9.88)
DHEA-S (µg/dL)	≤ 40.8	81.17 (59.14)
<b><i>Immune/Inflammatory Markers</i></b>		
IGF-1 (ng/mL)	≤ 69.5	105.14 (48.3)
IL-6 (pg/mL)	≥ 1.41	1.84 ( 8.3)
Albumin (mg/dl)	≤ 4.4	4.48 (0.29)
<b><i>Cardiovascular Risk Factors</i></b>		
Systolic blood pressure (mmHG)	≥ 150	138.45 (20.68)
Diastolic blood pressure (mmHG)	≥ 90	82.14 (11.08)
Ratio of total cholesterol to HDL	≥ 5.1	4.37 (1.44)
HDL cholesterol (mg/dL)	≤ 38	49.10 (13.71)
Glycosylated Hemoglobin (%)	≥ 5.8	5.75 (1.34)
Waist-hip ratio	≥ 0.93	0.88 (0.07)

*Notes:* For IL-6 and epinephrine, a large number (approximately 33% and 20%, respectively) of readings fell below assay sensitivity, but our analysis looks at the top quartile for each of those measures.

**Table 2: Odds ratio for falling in highest risk quartile of neuroendocrine and immune/inflammatory markers (95% CI)**

<b>Men</b>							
<b>SES Variables</b>	<b>Cortisol</b>	<b>Norepinephrine</b>	<b>Epinephrine</b>	<b>DHEA-S</b>	<b>IGF-1</b>	<b>IL-6</b>	<b>Albumin</b>
No Formal Education	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Primary Education	1.1 (0.61 - 1.98)	0.74 (0.41 - 1.36)	1.13 (0.61 - 2.07)	0.87 (0.49 - 1.53)	0.85 (0.50 - 1.44)	0.70 (0.42 - 1.16)	1.0 (0.61 - 1.63)
Secondary Education	1.01 (0.53 - 1.94)	0.83 (0.43 - 1.57)	1.97 (1.04 - 3.74)*	0.33 (0.16 - 0.69)*	0.61 (0.33 - 1.12)	0.48 (0.27 - 0.85)*	0.78 (0.45 - 1.34)
Lowest Income Quartile	1.0	1.0	1.0	1.0	1.0	1.0	1.0
2nd Income Quartile	1.49 (0.77 - 2.88)	0.99 (0.48 - 2.06)	0.59 (0.31 - 1.13)	0.99 (0.53 - 1.86)	0.71 (0.39 - 1.28)	1.65 (0.91 - 2.99)	1.35 (0.78 - 2.34)
3rd Income Quartile	1.08 (0.55 - 2.10)	1.51 (0.77 - 2.98)	0.69 (0.37 - 1.28)	0.55 (0.28 - 1.09)	0.74 (0.42 - 1.32)	1.44 (0.80 - 2.61)	0.99 (0.58 - 1.72)
Highest Income Quartile	1.32 (0.67 - 2.60)	1.62 (0.80 - 3.27)	0.85 (0.46 - 1.58)	0.66 (0.32 - 1.35)	0.59 (0.31 - 1.10)	1.04 (0.54 - 1.98)	1.08 (0.61 - 1.91)

<b>Women</b>							
<b>SES Variables</b>	<b>Cortisol</b>	<b>Norepinephrine</b>	<b>Epinephrine</b>	<b>DHEA-S</b>	<b>IGF-1</b>	<b>IL-6</b>	<b>Albumin</b>
No Formal Education	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Primary Education	1.18 (0.73 - 1.91)	1.07 (0.67 - 1.72)	1.42 (0.87 - 2.33)	0.99 (0.62 - 1.56)	0.76 (0.45 - 1.27)	0.84 (0.50 - 1.41)	0.6 (0.35 - 1.04)
Secondary Education	1.98 (1.02 - 3.85)*	0.98 (0.49 - 1.96)	2.93 (1.48 - 5.79)*	0.30 (0.13 - 0.67)*	0.89 (0.43 - 1.86)	0.94 (0.44 - 1.98)	0.70 (0.32 - 1.52)
Lowest Income Quartile	1.0	1.0	1.0	1.0	1.0	1.0	1.0
2nd Income Quartile	1.09 (0.61 - 1.95)	1.24 (0.70 - 2.23)	1.06 (0.58 - 1.93)	0.79 (0.45 - 1.38)	0.86 (0.47 - 1.56)	0.83 (0.45 - 1.53)	0.64 (0.34 - 1.22)
3rd Income Quartile	0.80 (0.42 - 1.52)	1.82 (0.98 - 3.39)	1.01 (0.53 - 1.94)	0.81 (0.44 - 1.50)	0.88 (0.46 - 1.68)	0.89 (0.46 - 1.73)	1.15 (0.59 - 2.22)
Highest Income Quartile	0.90 (0.45 - 1.81)	1.12 (0.55 - 2.27)	0.73 (0.35 - 1.54)	0.91 (0.46 - 1.82)	0.76 (0.36 - 1.60)	0.74 (0.34 - 1.58)	0.85 (0.39 - 1.84)

95% confidence intervals in parentheses

\* p < 0.05

All models control for age

**Table 3: Odds ratio for falling in highest risk quartile of cardiovascular markers (95% CI)**

<b>Men</b>						
<b>SES Variables</b>	<b>Waist-Hip Ratio</b>	<b>Systolic BP</b>	<b>Diastolic BP</b>	<b>HDL Chol</b>	<b>Ratio Chol/HDL</b>	<b>HbA1c</b>
No Formal Education	1.0	1.0	1.0	1.0	1.0	1.0
Primary Education	0.86 (0.53 - 1.41)	1.31 (0.74 - 2.30)	0.96 (0.57 - 1.62)	1.40 (0.80 - 2.44)	1.23 (0.70 - 2.18)	1.33 (0.70 - 2.54)
Secondary Education	0.96 (0.56 - 1.65)	1.26 (0.68 - 2.33)	0.68 (0.38 - 1.23)	1.63 (0.89 - 2.97)	1.82 (0.99 - 3.35)	2.24 (1.14 - 4.42)*
Lowest Income Quartile	1.0	1.0	1.0	1.0	1.0	1.0
2nd Income Quartile	0.63 (0.36 - 1.08)	1.0 (0.53 - 1.87)	1.38 (0.75 - 2.57)	0.97 (0.54 - 1.74)	0.83 (0.46 - 1.50)	1.33 (0.68 - 2.59)
3rd Income Quartile	0.86 (0.51 - 1.45)	1.21 (0.67 - 2.21)	1.95 (1.09 - 3.51)*	0.79 (0.45 - 1.40)	0.70 (0.39 - 1.25)	1.0 (0.52 - 1.92)
Highest Income Quartile	0.61 (0.35 - 1.06)	1.06 (0.57 - 2.00)	1.32 (0.70 - 2.47)	0.93 (0.52 - 1.67)	0.75 (0.42 - 1.37)	0.99 (0.51 - 1.95)

<b>Women</b>						
<b>SES Variables</b>	<b>Waist-Hip Ratio</b>	<b>Systolic BP</b>	<b>Diastolic BP</b>	<b>HDL Chol</b>	<b>Ratio Chol/HDL</b>	<b>HbA1c</b>
No Formal Education	1.0	1.0	1.0	1.0	1.0	1.0
Primary Education	1.10 (0.55 - 2.20)	0.93 (0.55 - 1.57)	0.85 (0.52 - 1.41)	1.05 (0.59 - 1.87)	0.91 (0.54 - 1.54)	0.61 (0.37 - 0.99)*
Secondary Education	0.08 (0.01 - 0.63)*	0.65 (0.29 - 1.45)	0.34 (0.14 - 0.83)*	0.57 (0.20 - 1.61)	0.68 (0.30 - 1.56)	0.45 (0.21 - 0.94)*
Lowest Income Quartile	1.0	1.0	1.0	1.0	1.0	1.0
2nd Income Quartile	0.48 (0.20 - 1.19)	0.41 (0.21 - 0.78)*	0.73 (0.39 - 1.38)	1.1 (0.54 - 2.22)	1.41 (0.74 - 2.68)	1.21 (0.68 - 2.17)
3rd Income Quartile	0.99 (0.40 - 2.43)	0.96 (0.50 - 1.82)	1.29 (0.67 - 2.48)	1.43 (0.69 - 2.99)	1.30 (0.64 - 2.61)	1.52 (0.81 - 2.86)
Highest Income Quartile	1.87 (0.71 - 4.92)	0.64 (0.30 - 1.38)	1.02 (0.48 - 2.15)	0.39 (0.14 - 1.11)	0.86 (0.38 - 1.96)	1.08 (0.52 - 2.21)

95% confidence intervals in parentheses

\* p <0.05

All models control for age

**Table 4: Odds ratio for having more risk factors in highest quartile for each index (95% CI)**

<b>Men</b>				
<b>SES Variables</b>	<b>Neuroendocrine</b>	<b>Cardiovascular</b>	<b>Immune/Inflammatory</b>	<b>Allostatic Load</b>
No Formal Education	1.0	1.0	1.0	1.0
Primary Education	0.97 (0.62 - 1.50)	1.17 (0.77 - 1.77)	0.78 (0.50 - 1.21)	0.97 (0.65 - 1.46)
Secondary Education	0.93 (0.57 - 1.51)	1.38 (0.87 - 2.18)	0.53 (0.33 - 0.86)*	0.95 (0.61 - 1.49)
Lowest Income Quartile	1.0	1.0	1.0	1.0
2nd Income Quartile	1.12 (0.68 - 1.85)	0.92 (0.58 - 1.46)	1.25 (0.76 - 2.04)	1.02 (0.64 - 1.60)
3rd Income Quartile	1.05 (0.65 - 1.70)	1.0 (0.64 - 1.56)	1.03 (0.64 - 1.65)	0.91 (0.59 - 1.42)
Highest Income Quartile	1.24 (0.75 - 2.07)	0.85 (0.53 - 1.35)	0.89 (0.55 - 1.46)	0.82 (0.52 - 1.30)

<b>Women</b>				
<b>SES Variables</b>	<b>Neuroendocrine</b>	<b>Cardiovascular</b>	<b>Immune/Inflammatory</b>	<b>Allostatic Load</b>
No Formal Education	1.0	1.0	1.0	1.0
Primary Education	1.3 (0.87 - 1.95)	0.77 (0.52 - 1.15)	0.72 (0.47 - 1.10)	0.84 (0.57 - 1.24)
Secondary Education	1.39 (0.76 - 2.52)	0.33 (0.18 - 0.61)*	0.94 (0.52 - 1.71)	0.55 (0.31 - 0.96)*
Lowest Income Quartile	1.0	1.0	1.0	1.0
2nd Income Quartile	1.06 (0.65 - 1.72)	0.83 (0.50 - 1.37)	0.71 (0.43 - 1.19)	0.66 (0.40 - 1.07)
3rd Income Quartile	1.04 (0.61 - 1.79)	1.46 (0.86 - 2.51)	0.89 (0.51 - 1.56)	1.17 (0.69 - 1.99)
Highest Income Quartile	0.86 (0.48 - 1.56)	0.90 (0.50 - 1.63)	0.69 (0.37 - 1.29)	0.69 (0.39 - 1.23)

95% confidence intervals in parentheses

\* p<0.05

All models control for age

**Table 5: Odds ratio for worse health or worse mobility (95% CI)**

SES Variables	Men		Women	
	Self-Rated Health	Mobility Difficulty	Self-Rated Health	Mobility Difficulty
<b>Model 1</b>				
No Formal Education	1.0	1.0	1.0	1.0
Primary Education	0.77 (0.50 - 1.19)	0.73 (0.46 - 1.15)	0.71 (0.47 - 1.08)	0.68 (0.44 - 1.03)
Secondary Education	0.56 (0.35 - 0.91)*	0.40 (0.23 - 0.68)*	0.49 (0.27 - 0.92)*	1.02 (0.57 - 1.84)
Lowest Income Quartile	1.0	1.0	1.0	1.0
2nd Income Quartile	1.09 (0.68 - 1.73)	0.96 (0.59 - 1.58)	0.64 (0.40 - 1.05)	0.55 (0.34 - 0.89)*
3rd Income Quartile	0.86 (0.55 - 1.36)	0.52 (0.31 - 0.88)*	0.8 (0.47 - 1.38)	0.55 (0.32 - 0.95)*
Highest Income Quartile	0.62 (0.39 - 1.01)	0.79 (0.46 - 1.38)	0.5 (0.27 - 0.92)*	0.34 (0.18 - 0.63)*
<b>Model 2</b>				
No Formal Education	1.0	1.0	1.0	1.0
Primary Education	0.72 (0.47 - 1.12)	0.72 (0.46 - 1.15)	0.71 (0.47 - 1.09)	0.66 (0.43 - 1.00)
Secondary Education	0.50 (0.31 - 0.82)**	0.39 (0.22 - 0.67)**	0.51 (0.27 - 0.95)*	1.04 (0.57 - 1.88)
Lowest Income Quartile	1.0	1.0	1.0	1.0
2nd Income Quartile	1.1 (0.69 - 1.76)	0.97 (0.59 - 1.60)	0.64 (0.39 - 1.04)	0.52 (0.32 - 0.85)*
3rd Income Quartile	0.84 (0.53 - 1.32)	0.53 (0.31 - 0.90)*	0.8 (0.46 - 1.37)	0.52 (0.30 - 0.90)*
Highest Income Quartile	0.60 (0.37 - 0.98)*	0.81 (0.47 - 1.42)	0.5 (0.27 - 0.93)*	0.34 (0.18 - 0.63)*
<b>Model 3</b>				
No Formal Education	1.0	1.0	1.0	1.0
Primary Education	0.77 (0.50 - 1.19)	0.72 (0.46 - 1.14)	0.72 (0.47 - 1.09)	0.7 (0.46 - 1.06)
Secondary Education	0.56 (0.35 - 0.91)*	0.39 (0.22 - 0.66)*	0.5 (0.27 - 0.93)*	1.11 (0.61 - 2.00)
Lowest Income Quartile	1.0	1.0	1.0	1.0
2nd Income Quartile	1.09 (0.68 - 1.73)	0.96 (0.58 - 1.58)	0.64 (0.39 - 1.05)	0.54 (0.33 - 0.88)*
3rd Income Quartile	0.86 (0.55 - 1.36)	0.52 (0.31 - 0.88)*	0.8 (0.46 - 1.37)	0.52 (0.30 - 0.90)*
Highest Income Quartile	0.63 (0.39 - 1.01)	0.83 (0.48 - 1.45)	0.5 (0.27 - 0.93)*	0.35 (0.19 - 0.65)*

Notes:

\*  $p < 0.05$

Model 1: adjusted for age

Model 2: adjusted for age and 3 sub-scales for stress, immune, and cardiovascular markers

Model 3: adjusted for age and allostatic load index

Self-reported health: 1=excellent, 2=good, 3=average, 4=not so good, 5=poor

Mobility difficulty: the # (0-6) of mobility difficulties the respondent reports with regards to the following activities: squatting, walking up 2-3 flights of stairs, lifting or carrying 11-12kg, walking 200-300 meters, standing continuously for 15 minutes, and grasping or turning things with fingers