**Socioeconomic differentials in mortality by cause and age in the U.S.**

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The inverse relationship between socioeconomic status (SES) and mortality is well-known, but the mechanisms behind it are not well understood. This study compares differences in overall and cause-specific mortality by SES for adults over age 50 in the U.S. to see how the SES-mortality gradient varies by cause of death. Using Cox proportional hazard models, I find large all-cause mortality differentials by education. I find large differences in mortality by education for respiratory diseases, circulatory diseases, lung cancer, and endocrine, nutritional, and metabolic diseases. These mortality differences inform how SES affects health.

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## Background

Dating back to 1973 when Kitagawa and Hauser published their research on U.S. mortality in the 1960s, researchers have consistently found significant differences in mortality risks and rates by socioeconomic status (SES) in the U.S. (Christenson and Johnson 1995; Elo, Martikainen and Smith 2006; Kitagawa and Hauser 1973). SES differences in mortality also persist across most causes of death, particularly cardiovascular disease. Not all causes of death, however, have consistent patterns by SES. This paper focuses on six leading causes of death: cancer; circulatory diseases; lung cancer; respiratory diseases; nutritional, metabolic, and endocrine diseases; and Alzheimer's disease. These categories are used to determine how the SES-mortality gradient varies by cause.

There have been several explanations offered to explain this SES-mortality differential. First, the SES effect on health and mortality may operate directly through access to health care. Although the evidence is not conclusive, health care seems to have an inverse relationship with overall mortality risk. The better-off are more likely to have health insurance which makes health care more affordable and increases access to care. Studies have found that adults with chronic conditions who do not have health insurance are more likely to postpone or forgo needed care (Hoffman and Paradise 2008). Evidence also suggests that the uninsured have worse survival rates for cancer (Hoffman and Paradise 2008). If this is the underlying effect of SES on health, then the SES-mortality gradient should be largest for both chronic, treatable conditions, such as heart disease and diabetes, and diseases which benefit from early detection and treatment, such as cancer.

Another explanation for the SES-mortality gradient is differential ability to incorporate new health knowledge. This theory posits that the more educated are better able to digest health information and incorporate new health behaviors into their lives. They are also better able to follow complicated treatment regimes for chronic conditions such as diabetes or AIDS (Goldman and Smith 2002) and may more likely than their less well off peers to engage in salubrious behaviors, such as wearing seatbelts (Harwood et al. 2007), exercising (Harwood et al. 2007), maintaining a healthful body weight (Wray, Alwin and McCammon 2005), and not smoking (Harwood et al. 2007; Wray et al. 2005). If health behaviors are the mechanism linking SES and mortality, then the SES-mortality gradient should be largest for causes of death that are strongly associated with behavioral risk factors, such as lung cancer and respiratory diseases, as well as diabetes.

A third explanation identifies psychosocial support as the underlying cause behind health disparities. Psychosocial support can come from the family, particularly a spouse, work, or outside organizations. For example, those who work in jobs where they have more control over their work are less likely to develop coronary heart disease (Bosma et al. 1997). Additionally, controlling for psychological and social risk factors has been shown to reduce the mortality differential between high and low SES groups, for all cause mortality and mortality from diseases of the circulatory system (Lynch et al. 1996). Stress seems to affect the circulatory system the strongest, so if this is the mechanism linking SES and mortality, circulatory diseases would have the strongest gradient.

Cause of death is a particularly salient outcome to evaluate these theories. Death certificates provide high quality and reliable data. Cause of death does not suffer from the biases of self-reported health conditions; for example, respondents' reliability of self-report of

conditions depends in part on their knowledge of the conditions. Self-reported conditions also require a respondent to have recently seen a physician to obtain a diagnosis. Differences in frequency of medical contact by SES could lead to biases in reported prevalence of conditions, which is less of a problem when cause of death is the outcome.

In light of the different mechanisms through which SES may affect health, it is not surprising then that the effect of SES on mortality differs by cause of death. Cause of death incorporates different elements of health. It partially reflects incidence of a condition in a given population, but it also reflects how well a given set of people are able to fight off the condition. Previous research has shown how the relationship between mortality and SES varies for given causes of death: heart diseases; cancers; respiratory diseases; endocrine, nutritional, and metabolic diseases; and Alzheimer's disease and dementia.

Evidence for an SES-cause specific mortality gradient is strongest for heart diseases. This is particularly important because heart disease continues to be the leading cause of death in the U.S. (Heron et al. 2009). A gradient is found for all common measures of SES, for men and women, and in the U.S. and in Europe (Elo et al. 2006; Mackenbach et al. 1999; Martikainen et al. 2001; Weires et al. 2008; Zajacova 2006). Some studies point toward a steeper gradient for women than men for ischemic heart disease (Mackenbach et al. 1999; Martikainen et al. 2001), but the overall pattern is consistent, regardless of whether the outcome is cardiovascular disease, cerebrovascular disease, or ischemic heart disease.

Research on a relationship between SES and cancer mortality is mixed. Many studies have found only a small or non-existent relationship between SES and cancer mortality (Elo et al. 2006; Mackenbach et al. 1999; Martikainen et al. 2001; Pensola and Martikainen 2004; Zajacova 2006). Lung cancer mortality, however, displays a universally strong, inverse

relationship with SES due to differences in smoking behavior (Elo et al. 2006; Mackenbach et al. 1999; Martikainen et al. 2001a; Weires et al. 2008).

Respiratory disease (ICD10: J00-99) mortality is also inversely associated with SES. (Elo et al. 2006; Mackenbach et al. 1999; Martikainen et al. 2001a; Pensola and Martikainen 2004; Zajacova 2006). In fact, Martikainen et al. (2001), using data from the Finnish registry system, found the strongest association for this cause. The causes of most respiratory diseases are nearly all behavioral, so a strong gradient for this cause would be highly indicative of a behavioral explanation for SES-mortality differentials.

Alzheimer's Disease and dementia (ICD10: F01, F03, G30, R54) seem to be inversely associated with SES, or at least level of education (Ngandu et al. 2007; Wilson et al. 2009). Few other studies have examined mortality from Alzheimer's Disease and dementia alongside competing causes of death.

Previous studies on SES and mortality have limitations. Only limited information is available on decedents; the sole source of information is the limited information contained on the death certificate itself (*e.g.* (Steenland, Hu and Walker 2004). Other studies such as the National Longitudinal Mortality Study only interview respondents once, so any changes in their status over time are unknown (*e.g.* (Mackenbach et al. 1999). The literature would benefit from a study that had respondents under study for a continuous time and provided a wide range of information on respondents' health, SES, and other characteristics.

Also, less is known about how the relationship between SES and mortality changes with age. Kitagawa and Hauser (1973) found weak associations between education and mortality above age 65 and only for Whites. More recent studies have found similarly weaker effects for older ages (Backlund, Sorlie and Johnson 1996; Martikainen et al. 2001). One study looking at

the effects directly by age found the effect of SES (as measured by education) on mortality to decline with age in a non-linear fashion (Christenson and Johnson 1995). This could be a result of heterogeneity; perhaps only the most robust persons in low SES groups survive to old ages (Vaupel and Yashin 1985), although evidence suggests this is not the case (Beckett 2000).

This paper extends the literature on SES and cause-specific mortality by using the Health and Retirement Study (HRS). This unique dataset will help to show how SES and mortality are related in greater detail, for attributes that change over time, and for older ages. This paper seeks to find out in greater detail how the shape of the SES-mortality gradient differ by cause of death, and how the shape of the SES-mortality gradient change with age. I hypothesize that the strength of the SES-mortality gradient varies by cause. Chronic conditions, such as heart disease, will have a steeper gradient than other causes of mortality. Also, the strength of the SES-mortality gradient will decline with age.

## Data and Methods

The data are from the HRS, a prospective panel study of older adults in the U.S. The HRS is representative of the non-institutionalized adult population over the age of 50, starting in 1992. New cohorts of respondents aged 51-56 were added to the study in 1998 and 2004 to maintain a representative sample at ages 51 and older. Detailed information on respondents' socio-demographic characteristics is collected biennially. This research uses follow-up information through wave 8, which was conducted in 2006. The HRS also works with the National Center for Health Statistics to collect information on exact date of death and detailed cause of death using the National Death Index (NDI). Those with missing data on key variables are excluded. The total sample is 28,505.

SES is measured as education and wealth. Education is measured in years and ranges from 0 – 17+. Household wealth is measured as the non-housing wealth of the household. It is calculated as the sum of the appropriate wealth components, minus debts, at the time a respondent entered into the study. Because debts are included, wealth can be a negative amount. Wealth is standardized to 2000 dollars. Wealth is included in models as a categorical variable. Respondents are split roughly into quartiles: lowest (zero or negative wealth), low ($1-7,320), high ($7,321-50,713), and highest ($50,714+). Other explanatory variables are age, sex, race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic, and Other), and marital status (married/partnered, divorced/separated, widowed, and never married)[1]. Descriptive statistics of the sample are shown in Table 1.

Mortality differentials by SES are estimated using Cox proportional-hazards models (Allison 1995). Respondents enter the analysis at the date of their first interview or 50th birthday, whichever comes later, and continue until their death or the date of their last interview. For cause-specific mortality models, respondents who die from a cause other than the one under investigation are censored at the date of death (Allison 1995).

Cause of death is coded into five categories: neoplasms (except lung cancer) (ICD-10 codes C00-D48 minus C32-C34), lung cancer (ICD-10 codes C32-C34), diseases of the circulatory system (ICD-10 codes I00-I99), respiratory diseases (ICD-10 codes J00-J99), endocrine, nutritional, and metabolic diseases (mostly diabetes) (ICD10- E00-E90), and Alzheimer's disease and dementia (ICD-10 codes F01, F03, G30, R54).

[1] A future draft will include changes in marital status.

## Results

Table 2 shows the results of the analysis for all-cause mortality. Model 1 shows the relationship between the demographic controls (age, gender, race/ethnicity, and marital status) and mortality. Adding in education, as in Model 2, mitigates the effect of some of these controls but does not substantively alter them, with the exception of the Hispanic group, which has a significantly lower hazard ratio relative to Whites once education is included in the model. As expected, education shows a clear inverse relationship with mortality, with those in lowest education category (less than high school) having the highest mortality. Model 3 includes wealth instead of education as an SES control. Wealth further mitigates the racial and marital status differences in mortality. Again, mortality is highest for the lowest wealth group and decreases at each level of wealth. Model 4 includes wealth and education together. Including both education and wealth makes the direct effect of each smaller, but both retain significant effects. The categorical variables for wealth and education are moderately correlated (R= 0.3440).

### Cause-Specific Mortality

Table 3 shows the results of the cause-specific mortality models. The SES-mortality gradient shows up clearly for circulatory diseases, as expected. Circulatory diseases account for nearly 40% of all deaths so it is not surprising the SES gradient for circulatory diseases is similar to that for all-cause mortality. Consistent with other studies, cancer shows only a small, insignificant gradient. However, the most educated and wealthiest have a significantly lower hazard of cancer mortality than the least educated and least wealthy. Lung cancer and respiratory diseases both show the predicted strong gradient with education and wealth being associated with lower hazards of mortality. Mortality from endocrine, nutritional, and metabolic diseases, a less frequently studied outcome, show a gradient that is even more pronounced than lung cancer and respiratory disease, with the most educated having a 70% lower hazard of death than the

least educated. Lastly, Alzheimer's disease shows a weak gradient by education but no gradient by wealth. A test of coefficients (results not shown) showed the effect of being in the most highly educated group (relative to lowest educated group) differed significantly for lung cancer and respiratory diseases relative to all other causes of death. The effect of being in the wealthiest group (relative to the least wealthy) differed for respiratory diseases relative to all other causes of death.

## Conclusions

In accordance with previous studies, I find an inverse association between SES and mortality among adults over age 50 in the U.S. I also find a gradient for specific causes of death: respiratory diseases, circulatory diseases, and lung cancer. The gradient for these causes is well known and for respiratory diseases and lung cancer is mostly attributable to differential smoking behaviors. Other cancers have a small gradient but significant protective effects are only found in the most educated and wealthiest categories. This is also in line with previous research. Endocrine, metabolic, and nutritional diseases show a strong wealth gradient and also protective effects for the most educated. This gradient is less studied. Alzheimer's disease showed a weak education gradient but no wealth gradient. Future research will more fully investigate the age pattern of the SES-cause mortality gradient. I also hope to model differences in mortality upon onset of a disease by SES.

## Future Steps for this Project

I have several additional plans for this paper. First, I will analyze the age pattern of the mortality differential. I will do this first by allowing the effect of age to vary over with time over

cause. I will also do this by breaking the sample into age groups and seeing how the effect of age varies by age group.

Additionally, I will introduce time-varying covariates. Specifically, I will include marital status changes, health behavior changes (particularly smoking status), and other key attributes that affect mortality, vary by SES, and change over time.

# Tables & Figures

## Table 1: Preliminary Descriptive Statistics, HRS Sample

| | Proportion or mean (s.d.) |
|---|---|
| Male | 43.97 |
| | |
| Year of birth | 1932.4 (12.6) |
| | |
| Race/Ethnicity | |
| White | 74.35 |
| Black | 14.83 |
| Missing/other | 2.33 |
| Hispanic | 8.49 |
| | |
| Marital Status (at entry) | |
| Married/partnered | 71.81 |
| Divorced/separated | 10.12 |
| Widowed | 14.84 |
| Never married | 3.24 |
| | |
| SES | |
| Years of school | 11.9 (3.5) |
| Wealth ($ 2000) | 66,769 (238,079) |
| | |
| | Number |
| Vital Status 2006 | |
| Alive | 21,150 |
| Dead | 8,406 |
| Circulatory diseases | 3,325 |
| Cancer (except lung) | 1,382 |
| Lung cancer | 603 |
| Respiratory illnesses | 805 |
| Endocrine/Nut/Met. | 330 |
| Alzheimer's Disease | 288 |
| Other cause | 1,368 |
| Cause missing | 316 |
| | |
| N | 28,505 |

## Table 2: Results, Hazard Ratios Mortality (Std. Err.), HRS Sample

| | Model 1 | Model 2 | Model 3 | Model 4 |
|---|---|---|---|---|
| Female | 0.61***(0.01) | 0.61***(0.01) | 0.61***(0.01) | 0.61***(0.01) |
| | | | | |
| **Race/Ethnicity** | | | | |
| (ref = White) | | | | |
| Black | 1.25***(0.04) | 1.15***(0.04) | 1.08* (0.03) | 1.05 (0.03) |
| Missing/ other | 1.12 (0.90) | 1.11 (0.09) | 1.00 (0.08) | 1.02 (0.09) |
| Hispanic | 0.97 (0.04) | 0.86***(0.04) | 0.82 (0.04) | 0.78***(0.04) |
| | | | | |
| **Marital Status** | | | | |
| (ref = Married) | | | | |
| Sep/Div | 1.41***(0.06) | 1.40***(0.06) | 1.32***(0.05) | 1.34***(0.05) |
| Wid | 1.21***(0.04) | 1.18***(0.03) | 1.15***(0.03) | 1.14***(0.03) |
| Never | 1.43***(0.09) | 1.43***(0.09) | 1.37***(0.08) | 1.39***(0.08) |
| | | | | |
| **SES** | | | | |
| Education | | | | |
| <HS | | Ref | | Ref |
| HS/GED | | 0.85***(0.02) | | 0.90***(0.03) |
| Some coll | | 0.79***(0.03) | | 0.86***(0.03) |
| Coll+ | | 0.61***(0.02) | | 0.69***(0.03) |
| | | | | |
| Wealth | | | | |
| Lowest | | | Ref | Ref |
| Low | | | 0.91** (0.03) | 0.92** (0.03) |
| High | | | 0.74***(0.02) | 0.77***(0.02) |
| Highest | | | 0.63***(0.02) | 0.70***(0.02) |
| | | | | |
| N | 28,505 | 28,505 | 28,505 | 28,505 |
| Generalized $R^2$ | 0.0187 | 0.0250 | 0.0263 | 0.0294 |

*** p<0.001
** p<0.01
*p<0.05

## Table 3: Hazard Ratios by Cause of Death, HRS Sample

| | All cause | Circulatory disease | Cancer (no lung) | Lung cancer | Respiratory disease | Endocrine disease | Alzheimer's disease | All other causes |
|---|---|---|---|---|---|---|---|---|
| Female | 0.61***(0.01) | 0.61*** (0.02) | 0.61 (0.04)*** | 0.44*** (0.04) | 0.45***(0.04) | 0.67** (0.08) | 1.39* (0.20) | 0.64***(0.04) |
| Race/Ethnicity | | | | | | | | |
| White (ref) | | | | | | | | |
| Black | 1.05 (0.03) | 1.02 (0.05) | 1.06 (0.09) | 0.77* (0.10) | 0.64***(0.07) | 1.50** (0.22) | 0.83 (0.16) | 1.51(0.11)*** |
| Missing/other | 1.02 (0.09) | 0.88 (0.13) | 0.99 (0.20) | 0.97 (0.29) | 0.67 (0.21) | 1.51 (0.55) | 1.29 (0.59) | 1.04 (0.22) |
| Hispanic | 0.78***(0.04) | 0.81** (0.06) | 0.47*** (0.07) | 0.45***(0.09) | 0.37***(0.07) | 1.41 (0.26) | 0.62 (0.20) | 1.11 (0.12) |
| Marital Status | | | | | | | | |
| Married (ref) | | | | | | | | |
| Sep/Div | 1.34***(0.05) | 1.32*** (0.09) | 1.20 (0.12) | 1.20 (0.18) | 1.75***(0.22) | 1.51* (0.28) | 1.01 (0.28) | 1.48***(0.14) |
| Wid | 1.14***(0.03) | 1.18***(0.05) | 1.00 (0.08) | 1.47***(0.18) | 1.13 (0.11) | 1.18 (0.18) | 0.87 (0.12) | 1.16* (0.09) |
| Never | 1.39***(0.08) | 1.43 (0.14) | 0.96 (0.17) | 0.85 (0.25) | 1.677**(0.31) | 1.35 (0.41) | 0.95 (0.34) | 1.84***(0.24) |
| SES | | | | | | | | |
| Education | | | | | | | | |
| <HS (ref) | Ref | | | | | | | |
| HS/GED | 0.90***(0.03) | 0.94 (0.04) | 0.90 (0.12) | 0.95 (0.10) | 0.73***(0.06) | 0.77 (0.11) | 1.09 (0.16) | 0.95 (0.07) |
| Some coll | 0.86***(0.03) | 0.85** (0.05) | 0.89 (0.07) | 0.89(0.11) | 0.81* (0.09) | 0.75 (0.13) | 1.00 (0.18) | 0.87 (0.08) |
| Coll+ | 0.69***(0.03) | 0.73*** (0.05) | 0.80* (0.17) | 0.40***(0.07) | 0.49***(0.07) | 0.30***(0.09) | 0.78 (0.18) | 0.84 (0.08) |
| Wealth | | | | | | | | |
| Lowest (ref) | Ref | | | | | | | |
| Low | 0.92** (0.03) | 0.90 (0.04) | 0.91 (0.25) | 1.11 (0.12) | 0.97 (0.09) | 0.63** (0.09) | 1.13 (0.20) | 1.01 (0.94) |
| High | 0.77***(0.02) | 0.76***(0.04) | 0.86 (0.07) | 0.74* (0.09) | 0.65***(0.07) | 0.57***(0.09) | 1.16 (0.20) | 0.86 (0.07) |
| Highest | 0.70***(0.02) | 0.71***(0.04) | 0.79** (0.07) | 0.66** (0.09) | 0.60***(0.07) | 0.50***(0.09) | 0.94 (0.18) | 0.71*** (0.06) |
| N | 28,505 | 28505 | 28505 | 28505 | 28505 | 28505 | 28505 | 28505 |
| Generalized $R^2$ | 0.0294 | 0.0103 | 0.0046 | 0.0058 | 0.0079 | 0.0043 | 0.0005 | 0.0006 |

***p<0.000
**p<0.01
*p<0.05

## References


Allison, P.D. 1995. *Survival analysis using SAS: A practical guide*. Cary, NC: SAS Institute.

Backlund, E., P.D. Sorlie, and N.J. Johnson. 1996. "The shape of the relationship between income and mortality in the United States - Evidence from the national longitudinal mortality study." *Annals of Epidemiology* 6(1):12-20.

Beckett, M. 2000. "Converging health inequalities in later life - An artifact of mortality selection?" *Journal of Health and Social Behavior* 41(1):106-119.

Bosma, H., M.G. Marmot, H. Hemingway, A.C. Nicholson, E. Brunner, and S.A. Stansfeld. 1997. "Low job control and risk of coronary heart disease in whitehall ii (prospective cohort) study." *BMJ* 314(7080):558-.

Christenson, B.A.and N.E. Johnson. 1995. "Educational inequality in adult mortality: An assessment with death certificate data from Michigan." *Demography* 32(2):215-229.

Elo, I.T., P. Martikainen, and K.P. Smith. 2006. "Socioeconomic differentials in mortality in Finland and the United States: the role of education and income." *European Journal of Population-Revue Europeenne De Demographie* 22(2):179-203.

Goldman, D.P.and J.P. Smith. 2002. "Can patient self-management help explain the SES health gradient?" *Proceedings of the National Academy of Sciences of the United States of America* 99(16):10929-10934.

Harwood, G.A., P. Salsberry, A.K. Ferketich, and A.E. Wewers. 2007. "Cigarette smoking, socioeconomic status, and psychosocial factors: Examining a conceptual framework." *Public Health Nursing* 24(4):361-371.

Heron, M., D.L. Hoyert, S.L. Murphy, J. Xu, K.D. Kochanek, and B. Tejada-Vera. 2009. "Deaths: Final Data for 2006." Hyattsville, MD: U.S. Department of Health and Human Services.

Hoffman, C.and J. Paradise. 2008. "Health insurance and access to health care in the United States." Pp. 149-160 in *Reducing the Impact of Poverty on Health and Human Development: Scientific Approaches*. Oxford: Blackwell Publishing.

Kitagawa, E.M.and P.M. Hauser. 1973. *Differential mortality in the United States, a study in socioeconomic epidemiology*. Cambridge, Mass.,: Harvard University Press.

Lynch, J.W., G.A. Kaplan, R.D. Cohen, J. Tuomilehto, and J.T. Salonen. 1996. "Do cardiovascular risk factors explain the relation between socioeconomic status, risk of all-cause mortality, cardiovascular mortality, and acute myocardial infarction?" *American Journal of Epidemiology* 144(10):934-942.

Mackenbach, J.P., A.E. Kunst, F. Groenhof, J.K. Borgan, G. Costa, F. Faggiano, P. Jozan, M. Leinsalu, P. Martikainen, J. Rychtarikova, and T. Valkonen. 1999. "Socioeconomic inequalities in mortality among women and among men: An international study." *American Journal of Public Health* 89(12):1800-1806.

Martikainen, P., P. Makela, S. Koskinen, and T. Valkonen. 2001. "Income differences in mortality: a register-based follow-up study of three million men and women." *International Journal of Epidemiology* 30(6):1397-1405.

Ngandu, T., E. von Strauss, E.L. Helkala, B. Winblad, A. Nissinen, J. Tuomilehto, H. Soininen, and M. Kivipelto. 2007. "Education and dementia - What lies behind the association?" *Neurology* 69(14):1442-1450.

Pensola, T.and P. Martikainen. 2004. "Life-course experiences and mortality by adult social class among young men." *Social Science & Medicine* 58(11):2149-2170.

Steenland, K., S. Hu, and J. Walker. 2004. "All-cause and cause-specific mortality by socioeconomic status among employed persons in 27 US states, 1984-4997." *American Journal of Public Health* 94(6):1037-1042.

Vaupel, J.W.and A.I. Yashin. 1985. "Heterogeneity ruses - Some surprising effects of selection on population-dynamics." *American Statistician* 39(3):176-185.

Weires, M., J.L. Bermejo, K. Sundquist, J. Sundquist, and K. Hemminki. 2008. "Socio-economic status and overall and cause-specific mortality in Sweden." *Bmc Public Health* 8.

Wilson, R.S., L.E. Hebert, P.A. Scherr, L.L. Barnes, C.F.M. de Leon, and D.A. Evans. 2009. "Educational attainment and cognitive decline in old age." *Neurology* 72(5):460-465.

Wray, L.A., D.F. Alwin, and R.J. McCammon. 2005. "Social status and risky health behaviors: Results from the health and retirement study." *Journals of Gerontology Series B-Psychological Sciences and Social Sciences* 60:85-92.

Zajacova, A. 2006. "Education, gender, and mortality: Does schooling have the same effect on mortality for men and women in the US?" *Social Science & Medicine* 63(8):2176-2190.