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**COMPOSITION AND DECOMPOSITION IN
U.S. GENDER-SPECIFIC SELF-REPORTED
HEALTH DISPARITIES, 1984-2007***

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Abstract

Variance function regression models and demographic decomposition methods are applied to identify two dimensions of changes in health disparities (SES-demographic effects vs. compositional effects, between-group disparities vs. within-group disparities) in the U.S. from 1984 to 2007. Using National Health Interview Survey data on self-reported health, we find that disparities in men's health increased, while those of women decreased, for the whole period. Widening men's health disparities are largely driven by increases in the effects of SES-demographic statuses on within-group disparities. These increases are moderated by increasing levels of men's college attainment. But decreasing middle and upper income attainment and a decreasing employment rate further increase men's health disparities. For women, the effects of SES-demographic statuses on health disparities also increased over time. This, however, was outweighed by increases in women's college attainment, middle and upper income attainment, and employment rate. The result is overall declining self-reported health disparities for women.

Keywords: Self-reported Health; Health Disparities; Gender; Variance Function Regression; Demographic Decomposition; SES-demographic Effects; Compositional Effects; Between-group Disparities; Within-group Disparities

COMPOSITION AND DECOMPOSITION IN U.S. GENDER-SPECIFIC SELF-REPORTED HEALTH DISPARITIES, 1984-2007

The growth of health disparities in the U.S. across the past two decades has been one of the most extensively researched topics in demography, medical sociology, epidemiology and public health (Williams and Collins 1995; Hummer et al. 1998). Disparities in health may occur across categories of socioeconomic status (SES, including income, education and occupation) and such sociodemographic characteristics as work status, marital status, race/ethnicity and gender. Researchers have examined health disparities by studying several different health outcomes, including mortality (e.g., Pappas et al. 1993), cause-specific mortality (e.g., Steenland et al. 2004; Jemal et al. 2008), morbidity (e.g., Schoeni et al. 2005), life expectancy (e.g., Crimmins and Saito 2001; Meara et al. 2008), and self-rated health (e.g., Goesling 2007).

Previous research, however, tended to focus on changes in gradients of SES and sociodemographic statuses (hereafter termed SES-demographic statuses) on health outcomes, that is, on the extent to which a health outcome varies as a function of each of a set of specific socioeconomic and demographic statuses. But health disparities can grow or decline due to two distinct mechanisms: the *gradient* or set of differential relationships of SES-demographic statuses to health outcomes can change and the *composition of the population* with respect to SES-demographic statuses itself can change. These two factors are confounded with each other. For example, growing health disparities can be caused not only by increasing gradients of the effects of SES-demographic statuses on health (i.e., a SES-demographic effect), but also can result from a decreasing probability for members of the population to be located at middle

statuses (i.e., a compositional effect). Separating SES-demographic effects from compositional effects then are essential in studying health disparities.

Moreover, health disparities are composed of between-group disparities and within-group disparities. *Between-group health disparities* represent variations in health across groups with different SES-demographic characteristics. *Within-group health disparities* describe health heterogeneity within each group with the same characteristics. Previous research has focused on between-group health disparities based on regression-based analysis, but within-group disparities in outcomes of social processes can far exceed between-group disparities (Western, Bloome, and Percheski 2008).

By applying a variance function regression model and standard decomposition methods to the analysis of self-reported health outcomes in the National Health Interview Survey, we are able to separate SES-demographic effects from compositional effects, between-group disparities from within-group disparities. Extant research suggests that temporal trends of health disparities in the past two decades have differed for men and women. Specifically, health disparities have consistently widened for men, while they have slightly increased, stagnated, contracted or significantly increased for women, depending on the time periods and health outcomes examined (Feldman et al. 1989; Preston and Elo 1995; Liu and Hummer 2008). Therefore, we examine gender-specific trends in self-rated health disparities in this paper.

We commence in the next section with a review of prior research on health disparities and trends therein over recent decades in the United States. We then review explanations of these trends. This is followed by a description of the data analyzed, the statistical models applied to the data, and the findings that result therefrom. We conclude with a discussion of the findings, limitations of the study, and directions for additional research.

TRENDS IN HEALTH DISPARITIES

Widening Health Disparities

Health disparities have increasingly become a major dimension of general social inequality in the United States. Socioeconomic statuses (SESs: along the dimensions of education, occupation, and income – a Weberian construct) are major social determinants of the distribution of health and disease in American society. Research has consistently documented an inverse relationship between SES and risks of diseases and death in the recent decades (e.g., Williams and Collins 1995; Link and Phelan 1995). The mechanisms accounting for this relationship include greater exposure to stress and hardship and more limited access to valuable resources (e.g., food, housing, health care, and medical knowledge), which can help prevent and cure disease, among lower SES individuals (George 2005; Adler and Newman 2002). In addition, health is not a singular condition and different components of SES (e.g., education, income and occupation) may have different impacts on different health outcomes. For example, some studies suggest that education exerts a stronger effect on the onset of disease and sickness, while income plays a more important role on the progression afterwards (e.g., Zimmer and House 2003).

In the U.S. context, race is another important predictor of health and disease; particularly, whites usually have better health than blacks. But research has shown divergent findings on the extent to which SES differences can account for race disparities in health. On the one hand, research suggests SES differences account for a large proportion of racial disparities in health (e.g., Rogers 1992), although it does not eliminate the disparities (e.g., Krieger and Fee 1994). On the other hand, some research finds even larger racial disparities in health at higher levels of

SES – that is, when SES increases, blacks do not achieve as much improvement in health as whites have (Farmer and Ferraro 2005). In spite of these divergent findings, most studies coherently demonstrate that race is a potent predictor of variations in health.

Many recent studies in demography, medical sociology and epidemiology have observed rising socioeconomic differentials in health in the U.S. in the past several decades, that is, socioeconomic status has increasingly exerted a stronger effect on health and diseases in the past several decades (e.g., Feldman et al. 1989; Pappas et al. 1993; Preston and Elo 1995; Hummer et al. 1998; Meara et al. 2008). For example, Pappas et al. (1993) found a sharper increase in socioeconomic disparity in mortality differentials since 1960 and this result holds across gender, race and marital status. Mortality differentials across socioeconomic statuses grew between 1990 and 2000 (Meara et al. 2008; Jemal et al. 2008). Besides mortality differentials, research has found growing educational disparities in old-age disability (Schoeni et al. 2005), disability-free life expectancy (Crimmins and Saito 2001), and total life expectancy (Meara et al. 2008) over recent decades. Similarly, a trend towards widening gaps in self-rated health by educational levels for middle-aged and older adults has continued since the early 1980s (Goesling 2007; Liu and Hummer 2008) or for all ages among younger cohorts (Lynch 2003).

Besides a growing health disparity by SES, disparities also have widened across marital status, race and ethnicity. With regard to marital status, studies found that in the past several decades male mortality differences between the married and unmarried statuses have widened (Smith 1996; Hummer et al. 1998), and women's self-rated health has improved more for the married than for the widowed, divorced and separated (Liu and Umberson 2008). And the racial gap in mortality and life expectancy continues widening, which is largely explained by a slower rate of decline among blacks than whites for heart disease, while HIV infection, diabetes,

pneumonia and homicide are major causes of decreasing life expectancy for blacks (Kochanek et al. 1994).

Trends in Gender-Specific Health Disparities

Although enlarging health disparities have become a widespread phenomenon in the U.S. that holds across gender (Pappas et al. 1993), some research actually suggests different trends of health disparities by sex. For example, Feldman et al. (1989) compared the mortality rate in the 1960 Matched Records Study data and in 1971-1984 data from the first National Health and Nutrition Examination Survey (NHANES I) Epidemiologic Followup Study (NHEFS). They found that educational differentials in men's death rates widened between 1960 and 1984, because death rates declined more rapidly among the more educated than the less educated. In contrast, women's death rates declined at similar rates across levels of educational attainment, which produces similar magnitudes of mortality disparities between 1960 and 1984 for women. NCHS (1994) reports that the racial gap in life expectancy widened much more for men (from 6.9 years to 8.3 years) than for women (from 5.6 years to 5.8 years) between 1980 and 1991. Moreover, Preston and Elo (1995) found that educational disparities in adult mortality widened for men but contracted for working-age women. In contrast, most recent research suggests that educational disparities in self-rated health and life expectancy have widened among women in the two most recent decades (Liu and Hummer 2008; Meara et al. 2008), which may be due to the increasing importance of education not only for labor market and income outcomes (Hamil-Luker 2005), but also for a higher probability of marriage, a higher standard of living and insurance against poverty among women than ever before (DiPrete and Buchmann 2006). In sum, these studies generally have found that health disparities have widened for men, while only

slightly increasing, stagnating, contracting, or significantly increasing for women – but the findings for women also depend on the time periods and health outcomes examined. Nonetheless, there is substantial evidence of major gender differences in trends in health disparities. Accordingly, in the analyses reported below, we examine *gender-specific self-rated health disparities trends*, with the recognition that findings may not generalize to health disparity trends associated with other health outcomes.

EXPLAINING TRENDS IN HEALTH DISPARITIES

Changing Gradients and Changing Population Composition

Much the prior research on health disparities has focused on health differences across individual-level characteristics (e.g., income, education, occupation, work status, race, ethnicity, gender, marital status) and how these differences change over time. This line of research is more concerned with *changing gradients* of the relationship of individual-level characteristics to health. However, changing health disparities can also be attributed to *changing population composition*, which has been neglected in previous research.

Enlarging health disparities can be caused, for example, by increasing gradients of the relationship of SES-demographic characteristics to health (e.g., a SES-demographic effect) or a decreasing probability for lower SES groups to enter middle or higher statuses (i.e., a compositional effect), and vice versa for declining health disparities. For instance, health disparities can increase due to the increasing importance of education on earnings (DiPrete and Buchmann 2006), access to advanced health care and medical technology innovation (Glied and Lleras-Muney 2008), or others beneficial for health (i.e., a socioeconomic effect), but also can be

reduced by increasing higher education attainment and labor force participation among lower income families (i.e., a compositional effect).

Therefore, although health disparity has increased among women in recent years due to the increasing importance of education for labor force participation, income and health (Liu and Hummer 2008) (i.e., a socioeconomic effect), this increase is possibly offset by increasing higher education achievement in lower status families (Buchmann and DiPrete 2006) and by increasing labor force participation (Juhn and Potter 2006) among women (i.e., a compositional effect). In contrast, health outcomes for men may be worsened by the double effect of the increasing importance of education for earnings and standard of living (i.e., a socioeconomic effect), and by declining labor force participation (Juhn and Potter 2006) among men (i.e., a compositional effect). Therefore, in order to attain a complete picture of changing health disparities, it is very important to separate SES-demographic effects from compositional effects.

Between-Group and Within-Group Disparities

Recent research suggests increases in income inequality can occur both between groups and within groups (Western, Bloome, and Percheski 2008; Western and Bloome 2009; Lemieux 2006). This research proceeds by decomposing increases in income inequality into between-group disparities and within-group disparities. The same logic can be applied to health disparities. Between-group health disparities represent variation in health across groups with different characteristics, for example, health difference between less educated persons and better educated persons. Previous research has focused on between-group health disparities based on regression-based analysis, but within-group disparities also can be substantial and should be studied.

Within-group health disparities describe health heterogeneity within each group with the same characteristics – for example, within a group defined by low levels of educational attainment – which can be estimated by residual variances from regression analyses. These residuals usually are neglected by scholars. Within-group health disparities may be due to variations in health within groups, differing variances of measurement error, and genetic effects. Research suggests that genetic factors can account for as much as 50% of frailty and mortality differences among individuals (Yashin et al. 1999; Iachine et al. 1998). Added to variations in health within groups, it follows that within-group disparities may contribute more to total health disparities than between-group disparities. However, we are more interested in explaining how *changes* in health disparities are associated with *changes* in between-group disparities and *changes* in within-group disparities. In this case, *changes* in health disparities are not necessarily largely driven by *changes* in within-group disparities. Assuming group-specific variances of measurement error are relatively constant over time (Lemieux 2006), and noting that the time scale of genetic changes involves many generations, changes in within-group health disparities are then mostly driven by changes in variations in health within groups.

The previous section has described how changes in between-group health disparities can be explained by changing gradients of SES-demographic effects on health and by the changing SES-demographic composition of the population, which also contribute to changes in within-group health disparities. In other words, changes in within-group disparities can be caused by changes in variations in health within each socioeconomic-demographic group (e.g., a SES-demographic effect) and/or changes in population distribution in groups with different within-group health variations (e.g., a compositional effect). For example, increase in within-group health disparities can be associated with increasing within-group health variations in college

graduates and/or with an increasing population proportion of college graduates who may have higher within-group health variations than non-college graduates.

DATA

Our analysis is based on annual data from the National Health Interview Survey (NHIS) for the 24-year period 1984 to 2007. NHIS is a repeated cross-sectional survey of civilian non-institutionalized US population conducted by National Center for Health Statistics. NHIS collects health information for each member of a family or household sampled, as reported by one primary respondent. In order to reduce reporting/measurement errors, we limit our analysis to the primary respondent. The sample size for men is about 16,837 each year (in total $16,837 \times 24 = 404,098$), and for women is about 12,439 each year (in total $12,439 \times 24 = 298,546$).

The sample frame for the NHIS is redesigned every ten years. Nonetheless, the fundamental design of the 1995-2007 NHIS is similar to that of the 1985-1994 NHIS. Two changes in the sampling design are notable. First, the number of primary sampling locations has increased from 198 to 358 since 1995. Second, both black and Hispanic populations were oversampled in the 1995-2007 NHIS, while only blacks were oversampled in the 1985-1994 NHIS. These two redesigns potentially increase the variances (health disparities in our paper) among samples. As discussed in the Results section, we study trends in health disparities by adjusting for population compositional changes, which encompasses both real changes in the society and sampling changes in the survey.

The question and response structure for the key outcome variable analyzed here, *self-rated health*, has remained largely unchanged across periodic revisions of the NHIS

questionnaires, which facilitates the analysis of trends. It has five response categories: poor, fair, good, very good, and excellent. Self-rated health is a widely used measure of general health status that has been found to be very predictive of mortality and strongly correlated with objective assessments of health, including physician diagnoses (Idler and Benyamini, 1997; Case and Paxson, 2005). Close relationships between self-rated health and objective health indicators also hold across population subgroups (Bosworth et al., 1999; Kennedy, Kasl, and Vaccarino, 2001). In order to differentiate respondents with very good health from those with average or poor health and get a better (less skewed) distribution of the outcome variable, we recoded the measure into a dichotomous variable coded 1 for people reporting “very good” or “excellent” health and 0 for people reporting “poor”, “fair” or “good” health¹.

To facilitate application of variance function regressions and decomposition methods (described in the Research Strategy and Models section), we also recoded all explanatory variables into categorical variables. In the NHIS, income was measured by several income categories. We first calculated the mid-point of each income category and then converted the mid-points to 2007 U.S. dollars. We recoded family income to three income levels: below the 20th percentile, 20th to 50th percentile, and above the 50th percentile.² A detailed description of explanatory variables is given in Table 1. In brief, we grouped the explanatory variables for each gender into three income levels, three education categories, two employment statuses, six age groups, two race categories, and two marital statuses. This yields $3*3*2*6*2*2 = 432$ groups for each gender.

¹ We also recoded the measure into a dichotomous variable coded 1 for people reporting “good”, “very good” or “excellent” health and 0 for people reporting “poor”, or “fair” health. Overall findings for this difference in coding are similar.

² We could not identify whether people are above the 80th or 90th percentile due to questionnaire limitations in the NHIS. For example, in the 2007 NHIS, the highest income category is \$75,000 and above.

[Insert Table 1 About Here]

RESEARCH STRATEGY AND MODELS

We applied variance function regression and decomposition methods to study gender-specific health disparity trends in the U.S. from 1984 to 2007. By using these methods, we can separate SES-demographic effects from compositional effects, between-group disparities from within-group disparities.

Variance Function Regression

We measure health disparity by the variance in probabilities of reporting very good or excellent health. By using variance function regression, we can distinguish between-group differences from within-group differences (Western, Bloome, and Percheski 2008; Western and Bloome 2009; Lemieux 2006). Sociologists often emphasize between-group inequality (or disparity) based on regression-based analyses of inequality. But within-group or residual inequality (or disparity) may far exceed between-group differences (Western, Bloome, and Percheski 2008). By using variance function regression, we can analyze the extent to which increasing or decreasing health disparity in the U.S. can be attributed to increasing or decreasing between-group differences as compared to increasing or decreasing within-group differences.

Variance function regression has two parts, including a regression for conditional mean, y_i , and a regression for logarithm of the residual variances, $\log(\sigma_i^2)$ (Western and Bloome 2009):

$$\hat{y}_i = x_i' \beta \tag{1}$$

$$\log(\hat{\sigma}_i^2) = z_i' \lambda, \quad (2)$$

where observations on individual sample members are indexed by i , x_i' is a vector of covariates for mean or expected value \hat{y}_i , and z_i' is a vector of covariates (possibly equal to x_i') for the conditional expected value of logarithm of the residual variance $\log(\hat{\sigma}_i^2)$. The quantity σ_i^2 is the squared residuals, \hat{e}_i^2 , from the first regression, where $\hat{e}_i = y_i - x_i' \hat{\beta}$. From a substantive viewpoint, the first regression describes how covariates that define the 432 cells described above affect the y_i response variable and account for the deviations of the within-cell sample means from the average or grand mean \bar{y} (which can be termed the between-group inequality), while the second regression explains how covariates affect the within-cell variability of the response variable around the cell means, that is, the unpredictability of y_i within the 432 cells (which can be termed the within-group inequality).

The models are estimated by maximum likelihood, using an iterative two stage method (Western and Bloome 2009; Aitkin 1987). In the iteration process, we repeat a weighted linear regression of y_i on x_i , with weights, $1/\hat{\sigma}_i^2$, which is estimated from the gamma regression on the log of the squared OLS residuals (i.e., the second regression). This method can generate unbiased, consistent and efficient estimations when sample size is large. Although the outcome variable, self-rated health is coded as a binary variable, we use a linear probability model rather than a binary logistic model for the first regression because the sample size is large enough to generate accurate estimates with the linear probability model.³

³ Specifically, (1) all of the predicted probabilities of reporting very good or excellent health are between 0 and 1 for all 432 groups for each year (1984, ..., 2007); (2) the linear probability model is much easier to interpret than is the binary logistic model; and (3) without modification, the binary logistic model does not permit adjustment for weights $1/\hat{\sigma}_i^2$ in the two stage estimation method (Western and Bloome 2009).

Decomposition and Standardization

Because we are interested in how health disparities (variances in probabilities of reporting very good or excellent health) changed from 1984 to 2007, we estimated the variance function regressions for each year instead of pooling all 24 years together. The variance function regressions can be denoted:

$$\hat{y}_{it} = x_{it}'\beta_t$$

$$\log(\hat{\sigma}_{it}^2) = z_{it}'\lambda_t,$$

where t demotes year, ranged from 1984 to 2007. After estimating all the β_t and λ_t coefficients for each year, we predict the probabilities and residuals of reporting very good or excellent health for each group in each year, that is, the \hat{y}_{ij} and $\hat{\sigma}_{ij}^2$, where j demotes group, ranged from 1 to 432. Each group j has a compositional weight or cell proportions, π_{ij} , giving the fraction of individuals falling into this group. Then the total variances in probabilities of reporting very good or excellent health can be written (Western and Bloome 2009):

$$V_t = B_t + W_t = \sum_{j=1}^J \pi_{ij} \hat{r}_{ij}^2 + \sum_{j=1}^J \pi_{ij} \hat{\sigma}_{ij}^2, \quad (3)$$

where B_t is between-group component (weighted sum of squared group deviation or between-group variance \hat{r}_{ij}^2), and W_t is within-group component (weighted sum of residual variance or within-group variance $\hat{\sigma}_{ij}^2$). Group deviation \hat{r}_{ij} is the deviation of the group mean from grand mean, that is $\hat{r}_{ij} = \hat{y}_{ij} - \bar{y}_t$.

If we have estimates at two time points, $t=0, 1$, we then can write changes in total variance in the following way:

$$\begin{aligned}
V_1 - V_0 &= B_1 - B_0 + W_1 - W_0 \\
&= \underbrace{\sum_{j=1}^J (\pi_{1j} - \pi_{0j}) \hat{r}_{1j}^2 + \sum_{j=1}^J (\hat{r}_{1j}^2 - \hat{r}_{0j}^2) \pi_{0j}}_{B_1 - B_0} + \underbrace{\sum_{j=1}^J (\pi_{1j} - \pi_{0j}) \hat{\sigma}_{1j}^2 + \sum_{j=1}^J (\hat{\sigma}_{1j}^2 - \hat{\sigma}_{0j}^2) \pi_{0j}}_{W_1 - W_0} \\
&= \underbrace{\sum_{j=1}^J (\pi_{1j} - \pi_{0j}) (\hat{r}_{1j}^2 + \hat{\sigma}_{1j}^2)}_{E_C} + \underbrace{\sum_{j=1}^J (\hat{r}_{1j}^2 - \hat{r}_{0j}^2) \pi_{0j}}_{E_B} + \underbrace{\sum_{j=1}^J (\hat{\sigma}_{1j}^2 - \hat{\sigma}_{0j}^2) \pi_{0j}}_{E_W} \\
&= E_C + E_B + E_W
\end{aligned} \tag{4}$$

The change in the between-group variance $B_1 - B_0$ is associated with a compositional effect

($\sum_{j=1}^J (\pi_{1j} - \pi_{0j}) \hat{r}_{1j}^2$) and a socioeconomic effect ($\sum_{j=1}^J (\hat{r}_{1j}^2 - \hat{r}_{0j}^2) \pi_{0j}$). The change in the within-group

variance $W_1 - W_0$ is associated with a compositional effect ($\sum_{j=1}^J (\pi_{1j} - \pi_{0j}) \hat{\sigma}_{1j}^2$) and a SES-

demographic effect ($\sum_{j=1}^J (\hat{\sigma}_{1j}^2 - \hat{\sigma}_{0j}^2) \pi_{0j}$). In sum, Equation 4 indicates that changes in health

disparity V_t can be decomposed into (1) a compositional effect that changes the distribution of

population across groups (i.e., E_C , or changes in π_{ij}), (2) a SES-demographic effect that changes

the gradient of SES-demographic status on between-group disparities (i.e., E_B , or changes in \hat{r}_{ij}^2),

and (3) a SES-demographic effect that changes the gradient of SES-demographic statuses on

within-group disparities (i.e., E_w , or changes in $\hat{\sigma}_{ij}^2$).

We also can standardize adjusted variances by fixing π_{ij} , \hat{r}_{ij}^2 , or $\hat{\sigma}_{ij}^2$ at baseline time point, $t = 0$. Adjusted variances can be interpreted as the variance we could observe if population compositions, between-group variances, or within-group variances remained unchanged at their $t = 0$ values. Additionally, we can calculate an explanatory variable's (e.g., income or education) socioeconomic effect or compositional effect by fixing its regression coefficients or weights at t

= 0. Due to space limits, we cannot describe these standardization techniques in more details; they, however, can be found in Western, Bloome, and Percheski (2008), Western and Bloome (2009), and Lemieux (2006).

RESULTS

Disparities in Men's Self-Report Health, 1984-2007

Figure 1 displays the estimated trend of self-reported health disparities for men from 1984 to 2007. The trajectory over time of predicted total variance from the variance function regression tracks very closely with the trajectory of observed total variance, which suggests that the variance function regression does a good job in capturing the trend of self-reported health disparities and the linear probability model generates very accurate estimations. Overall, self-reported health disparity for men increased from 1984 to 2007. But there were variations in the trend within this overall period – specifically, disparity decreased from 1984 to 1990, rose until around 1995, decreased afterwards, and then rose again after 2000.⁴

[Insert Figure 1 About Here]

Figure 2 further decomposes the total variance into between-group variance and within-group variance. The left vertical axis is for total variance and within-group variance. The right one is for between-group variance. There are two main findings in Figure 2. First, within-group variance accounts much more than between-group variance for total variance. In 1984, within-

⁴ Considering the possible increase in variance caused by a sampling redesign of the NHIS in 1995, the “real” disparity may decrease even more after 1995. So the decrease in health disparity between 1995 and 2000 is not artificial, but may be underestimated.

group variance and between-group variance contribute about 81% and 19% to total variance, respectively. In 2007, the corresponding numbers are 84% and 16%. This implies that many unobserved factors are not captured in between-group studies that control only for observed explanatory variables such as those in the present analysis. Life style, health habits, genetic differences, and measurement errors may contribute to within-group variances. Second, across the entire period from 1984 to 2007, within-group variance increases, which drives total variance up, while between-group variance decreases and offsets the effect of increases in within-group variance to some extent.

[Insert Figure 2 About Here]

Panel A of Table 2 further separates SES-demographic and compositional effects on the growth of men's self-reported health disparities – both for the entire 24-year period from 1984 to 2007 and for four sub-periods thereof. In each period, change in variance is decomposed into between-group variance and within-group variance, both of which are further decomposed into two sub-components: SES-demographic effects and compositional effects. The next two rows sum up the SES-demographic effects and compositional effects. The numbers without parentheses are amounts of change in variance associated with each specific component. The numbers in parentheses are percents of change explained by the corresponding component. For example, the amount of change in variance from 1984 to 1990 associated with between-group SES-demographic effects is -0.394, which contributes about 55% $(-0.394/-0.718)$ to the total change in variance (i.e., -0.718) from 1984 to 1990.

There are four main findings from Panel A. First, change in between-group variance accounts for much more than change in within-group variance as components of change in total variance when total variance decreases, while change in within-group variance is dominant when total variance increases. Increasing within-group variance accounts for 159% increase in total variance from 1984 to 2007, which is offset by decreasing between-group variance to some extent. Second, overall, SES-demographic effects on between-group variance decreased from 1984 to 2007, which, however, contribute only about 10% (-0.059/-0.574) to the declines of between-group health disparity. In contrast, compositional effects account for about 90% (-0.515/-0.574) of the declines of between-group health disparity. Third, SES-demographic effects on within-group variance dramatically increased from 1984 to 2007, which contributed 112% (1.739/1.546) to the increase of within-group health disparities. Compositional effects counterbalance the SES-demographic effects to some extent. Fourth, all the growth of health disparities from 1984 to 2007 results from increases in the gradients of the relationship of SES-demographic statuses on within-group health disparities (the contribution is 179%), while decreases in between-group variance and changes in population compositions offset the former effect to some extent.⁵

[Insert Table 2 About Here]

Panel B of Table 2 further portrays how different segments of SES-demographic status (e.g., income and education) affect total variance by examining the changes in adjusted variances when a particular factor was fixed at the 1984 level. The numbers without parentheses are counterfactual changes in variance estimated by fixing a particular component at the 1984 level.

⁵ The “real” compositional effect may be underestimated as explained in notes below Table 2.

The difference of observed change in variance and adjusted change in variance is the amount of change in observed variance associated with each specific component. The numbers in parentheses are percents of change explained by the corresponding component. For example, by fixing the college education attainment effect on between-group variance β at the 1984 level, the adjusted change in variance is 0.932. Thus, the amount of change in observed variance associated with this effect is 0.039 ($=0.971-0.932$), which means this effect accounts about 4.1% ($0.039/0.971$) of the increase in variance from 1984 to 2007. Overall, the changing gradients of SES-demographic effects on within-group health disparities contribute much more to change in total disparities than changing gradients of their effects on between-group health disparities, e.g., 108.4% vs. 4.1% for the college education effect, 37.3% vs. 17.9% for the middle and upper income effect, 27.6% vs. -4.9% for the employment status effect, 91.3% vs. 0.7% for the race effect.

This means that increasing gradients of SES-demographic effects on with-group disparities are the main engines of widening health disparities for men. More specifically, education plays a more important role than income. College effects contribute about 112.5% ($(0.971+0.121)/0.971$) to the increase of total disparities, while middle and upper income effects account for about 55.2% ($(0.971-0.435)/0.971$). Health disparities between employed and unemployed men decrease, while disparities within each employment status increase, which may be due to an increasing underemployment rate within employed status⁶. Surprisingly, health disparities between races (i.e., white and non-white) do not increase much after controlling for other SES-demographic factors, but disparities within each race increase dramatically. This may be related to increasing diversity among the non-white race category across the years from 1984 to 2007 as a consequence of immigration to the U.S., as this category includes not only blacks,

⁶ In this paper, employed status includes both full-time job and part-time job.

but also Hispanics and Asians. The latter two groups have been found to have similar or even better health than whites, due to migration selectivity and the healthy immigrant effect (Antecol and Bedard 2006; McDonald and Kennedy 2004). As for the contributions of population composition to change in total variance, although increasing college attainment slows down the increase in health disparities, decreasing middle and upper income attainments and a decreasing employment rate further increase the health disparities.

Disparities in Women's Self-Reported Health, 1984-2007

Figure 3 displays the trend in self-reported health disparities for women from 1984 to 2007. Overall, total health disparity for women decreased from 1984 to 2007, but, as with men, this overall trend contains periods of increases and decreases. Specifically, disparity decreased from 1984 to 1990, rose until around 1995, decreased afterwards, and then rose again after 2000.⁷ These period-specific trends are similar to those for men, but men's total health disparity increased overall from 1984 to 2007 while women's shows a slight decline. For women, in addition, both between-group health disparity and within-group health disparity decreased overall from 1984 to 2007. Similarly to men, within-group variance accounts much more than between-group variance as a percentage of total variance, 84% and 16% on average, respectively. The dotted lines represent the trends of variances adjusted for compositional changes which are induced by population composition changes and possibly by sampling design changes in the NHIS surveys as well. If population compositions were fixed at 1984 levels, all the three variances would have increased over time. This suggests compositional changes made a substantial contribution to the declining health disparities.

⁷ See footnote 4.

[Insert Figure 3 About Here]

Panel A of Table 3 further separates SES-demographic and compositional effects on the growth of health disparities from 1984 to 2007. There are four main findings from Panel A. First, generally, changes in within-group variance account for more of changes in total variance than changes in between-group variance in all periods except 1984-1990. Second, different from men, SES-demographic effects on women's overall between-group variance significantly increased from 1984 to 2007, which, however, was outweighed by compositional effects, leading to overall decreased between-group health disparity. Third, similar to men, SES-demographic effects on women's within-group variance increased from 1984 to 2007, but to a much smaller extent than for men. Compositional effects far outweigh the SES-demographic effects, which results in overall decreased within-group health disparity for women. Fourth, all of the reduction of women health disparities from 1984 to 2007 results from changes in population composition (as indicated by dotted lines in Figure 3), while changes in gradients of socioeconomic statuses on between-group and within-group variance offset compositional effects to some extent⁸.

[Insert Table 3 About Here]

Panel B of Table 3 further explains how different dimensions of SES-demographic effects and population compositions affect trends in health disparities for women. As for men, an increasing gradient of the effect of college education on health increases health disparities for women, which accounts for about -14% of the decrease in health disparities. After controlling for other SES-demographic factors, however, the gradients of middle and upper income and

⁸ The "real" compositional effect may be underestimated as explained in notes below Table 3.

employment status effects on women's health variance decrease over time, which thus contribute about 29% and 22% to the decrease in health disparities, respectively. An articulation of the causes of the decreases of the gradients of these two factors over time is beyond the scope of this paper and merits further research. Enlarging racial health disparities significantly slowed down the decrease in total disparities for women. As expected, increasing college attainment, middle and upper income attainments, and employment rate reduce the total variance in probabilities of reporting very good/excellent health. They account for about 52.6%, 96.9%, and 22.2% of the decline of total health disparities, respectively.

DISCUSSION AND CONCLUSIONS

Health disparities have grown considerably in recent decades, at least for men. The sources of these increases have not been clear, however. Much previous research focuses on widening health disparities across socioeconomic status levels by looking at how the gradients of effects of SES on health changes over time. But population-wide health disparities are affected by changes in population composition as well. Previous research also focused on health disparities across groups with different SES-demographic characteristics (i.e., between-group disparities), but health disparities within each group with the same SES-demographic characteristics (i.e., within-group disparities) can exceed between-group disparities. By applying variance function regression models and demographic decomposition methods to the analysis of health outcomes, we are able to separate SES-demographic effects from compositional effects and between-group disparities from within-group disparities.

Applied to gender-specific self-rated health in the NHIS data from 1984 to 2007, these methods identify different health disparities trends for men and women. Both men's and women's health disparities decreased from 1984 to 1990, rose until around 1995, decreased afterwards, and then rose again after 2000. But, across the whole time period from 1984 to 2007, men's health disparities increased, while women's disparities decreased. Widening men's health disparities are largely driven by rising gradients of SES-demographic statuses (e.g., income, education and race) on within-group disparities, which are moderated by the equalizing effects of increasing rates of college attainment to some extent. However, decreasing middle and upper income attainment and a decreasing employment rate further increases men's disparities.

For women, the gradients of SES-demographic statuses (e.g., education and race) on between-group and within-group health disparities also increased over time. This, however, was outweighed by changing population composition, e.g., increasing college attainment, middle and upper income attainment, and employment rate. This leads to overall declining health disparities between 1984 and 2007 for women. But, if increases in college attainment and labor participation rates slow down in the future, women's health disparities may also increase like those of men in the past two decades.

This study proposes a new concept, within-group health disparities – disparities in health outcomes within each group defined by the same SES-demographic characteristics. Results suggest the gradient of SES-demographic effects on within-group health disparities has dramatically increased over the time period studied, especially for men. This poses a question: why did the health disparities among people with the same SES-demographic characteristics increase? Now that this phenomenon of increasing within-group health disparities has been identified, its full study and explanation can become a major topic of future research.

This study pertains to trends in self-rated health disparities. Although self-rated health has been shown to be a valid measurement of overall objective health, it should be emphasized that health is not a singular condition. Thus, further research is needed to examine trends in health disparities associated with other health outcomes, like functional health status (e.g., as defined by limitations in activities of daily living), morbidity (including the presence of specific diseases), and mortality. The conceptual framework developed herein – which emphasizes two dimensions to explore health disparities: SES-demographic effects vs. compositional effects, between-group health disparities vs. within-group health disparities – can fruitfully guide such future studies.

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Table 1. Description of Explanatory Variables in the NHIS, 1984 to 2007.

Variable	Description
Family income	1=below 20 th , 2=20 th to 50 th , and 3=above 50 th .
Education	1=without high school degree, 2=high school graduates, and 3=college graduates
Employment status	1=employed, and 2=unemployed
Age	1=18/29, 2=30/39, 3=40/49, 4=50/59, 5=60/69, and 6=70/85.
Race	1=whites, and 2=non-whites
Marital status	1=married, and 2=unmarried

Table 2. Decomposition and Standardization of the Change in Variance in Probabilities of Reporting Very Good/Excellent Health, U.S. Men, 1984-2007.

	1984 to 1990		1990 to 1995		1995 to 2000		2000 to 2007		1984 to 2007	
Panel A. Decomposition										
Change in variance	-0.718		0.771		-0.280		1.198		0.971	
Between-group	-0.623	(86.7)	0.167	(21.7)	-0.246	(87.7)	0.127	(10.6)	-0.574	(-59.1)
SES-Demographic effect	-0.394	(54.9)	0.440	(57.0)	-0.399	(142.5)	0.248	(20.7)	-0.059	(-6.1)
Compositional effect	-0.228	(31.8)	-0.273	(-35.3)	0.154	(-54.9)	-0.121	(-10.1)	-0.515	(-53.0)
Within-group	-0.096	(13.3)	0.604	(78.3)	-0.034	(12.3)	1.072	(89.4)	1.546	(159.1)
SES-Demographic effect	-0.192	(26.8)	0.546	(70.7)	0.014	(-4.9)	1.302	(108.7)	1.739	(179.1)
Compositional effect	0.097	(-13.5)	0.058	(7.6)	-0.048	(17.2)	-0.230	(-19.2)	-0.194	(-19.9)
Sum of SES-Demographic effects	-0.587	(81.7)	0.985	(127.8)	-0.386	(137.7)	1.550	(129.3)	1.680	(172.9)
Sum of compositional effects	-0.131	(18.3)	-0.214	(-27.8)	0.105	(-37.7)	-0.351	(-29.3)	-0.708	(-72.9)
Panel B. Adjusted change in variance, fixing at 1984										
College effect, β									0.932	(4.1)
College effect, λ									-0.082	(108.4)
College effect, β and λ									-0.121	(112.5)
Middle and upper income effect, β									0.797	(17.9)
Middle and upper income effect, λ									0.609	(37.3)
Middle and upper income effect, β and λ									0.435	(55.2)
Employment status effect, β									1.019	(-4.9)
Employment status effect, λ									0.703	(27.6)
Employment status effect, β and λ									0.751	(22.7)
Race effect, β									0.965	(0.7)
Race effect, λ									0.085	(91.3)
Race effect, β and λ									0.078	(92.0)
College attainment									1.148	(-18.2)
Middle and upper income attainment									0.87	(10.4)
Employment status composition									0.697	(28.2)

Note: 1. Numbers in parentheses are percent of change explained by corresponding factors.

2. Compositional effects here include compositional changes induced by population composition changes in the reality and sampling design changes in the NHIS surveys. Since sampling redesign may increase variance, the real compositional effects should be more negative. In other words, the compositional effects here may underestimate the real compositional effects.

Table 3. Decomposition and Standardization of the Change in Variance in Probabilities of Reporting Very Good/Excellent Health, U.S. Women, 1984-2007.

	1984 to 1990		1990 to 1995		1995 to 2000		2000 to 2007		1984 to 2007	
Panel A. Decomposition										
Change in variance	-0.578		0.481		-1.000		0.563		-0.535	
Between-group	-0.329	(57.0)	0.176	(36.7)	-0.137	(13.7)	0.241	(42.8)	-0.049	(9.2)
SES-Demographic effect	-0.060	(10.4)	0.353	(73.4)	-0.028	(2.8)	0.470	(83.6)	0.942	(-176.2)
Compositional effect	-0.269	(46.6)	-0.176	(-36.7)	-0.109	(10.9)	-0.229	(-40.8)	-0.991	(185.3)
Within-group	-0.248	(43.0)	0.304	(63.3)	-0.863	(86.3)	0.322	(57.2)	-0.486	(90.8)
SES-Demographic effect	-0.128	(22.1)	0.304	(63.2)	-0.110	(11.0)	0.349	(62.0)	0.162	(-30.3)
Compositional effect	-0.121	(20.9)	0.001	(0.1)	-0.753	(75.3)	-0.027	(-4.9)	-0.648	(121.1)
Sum of SES-Demographic effects	-0.188	(32.5)	0.656	(136.6)	-0.138	(13.8)	0.819	(145.7)	1.104	(-206.5)
Sum of compositional effects	-0.390	(67.5)	-0.176	(-36.6)	-0.862	(86.2)	-0.257	(-45.7)	-1.639	(306.5)
Panel B. Adjusted change in variance, fixing at 1984										
College effect									-0.611	(-14.2)
Middle and upper income effect									-0.378	(29.4)
Employment status effect									-0.416	(22.1)
Race effect									-1.124	(-110.2)
College attainment									-0.254	(52.6)
Middle and upper income attainment									-0.017	(96.9)
Employment status composition									-0.416	(22.2)

Note: 1. Numbers in parentheses are percent of change explained by corresponding factors.

2. Compositional effects here include compositional changes induced by population composition changes in the reality and sampling design changes in the NHIS surveys. Since sampling redesign may increase variance, the real compositional effects should be more negative. In other words, the compositional effects here may underestimate the real compositional effects.

Figure 1. Comparison between observed variance distribution and predicted variance from variance function regression, U.S. Men, 1984-2007

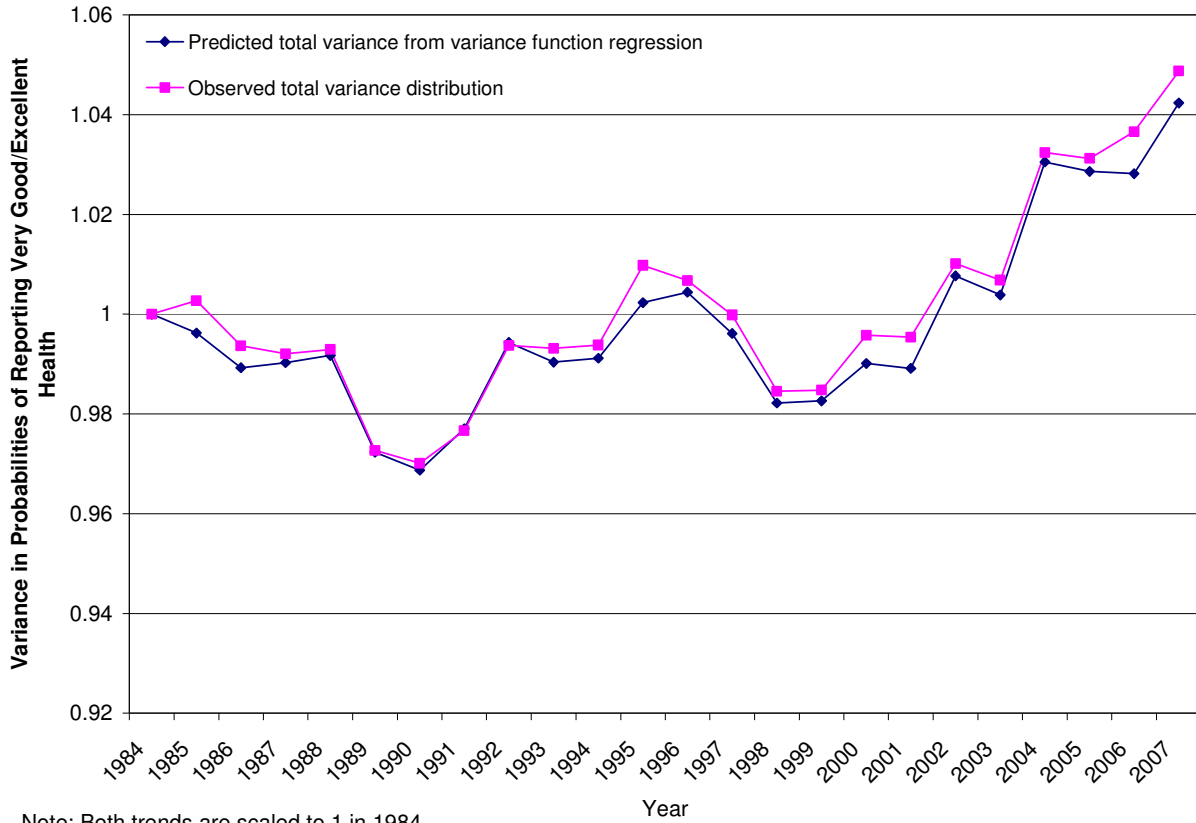


Figure 2. Decomposition of Total Variance into Between-Group Variance and Within-Group Variance, U.S. Men, 1984-2007

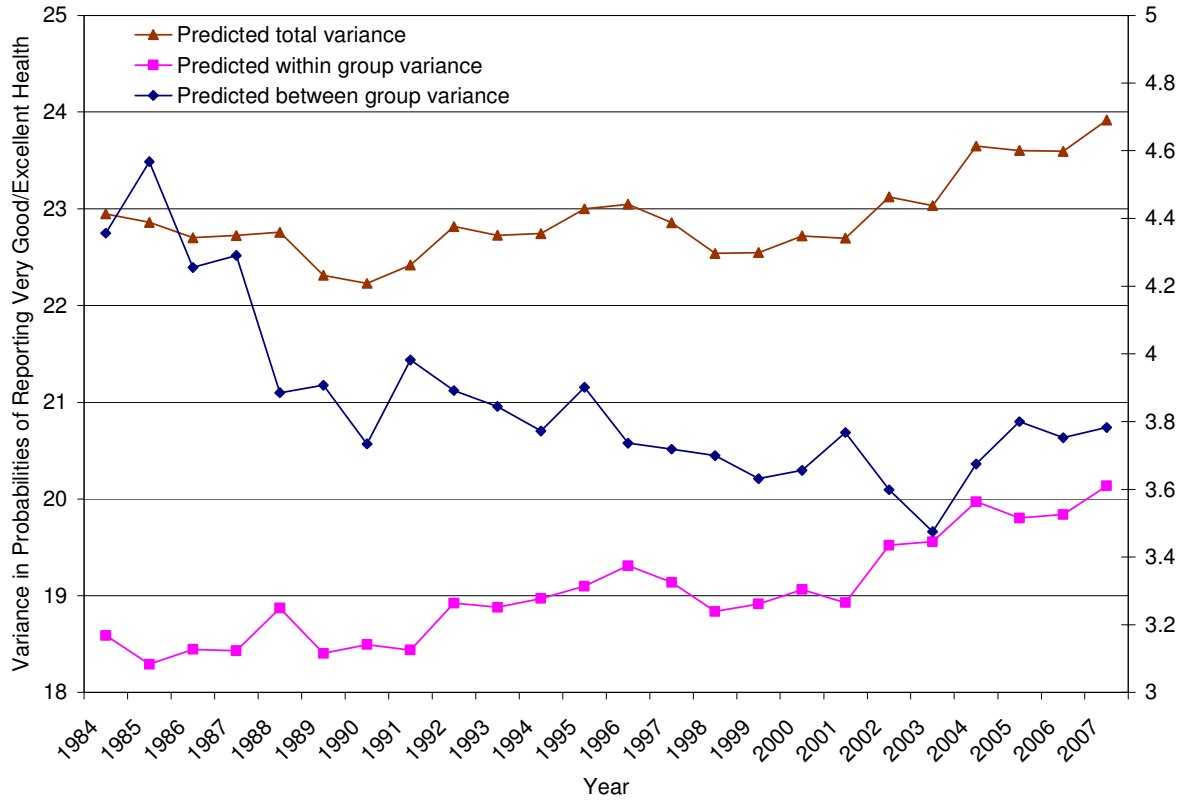
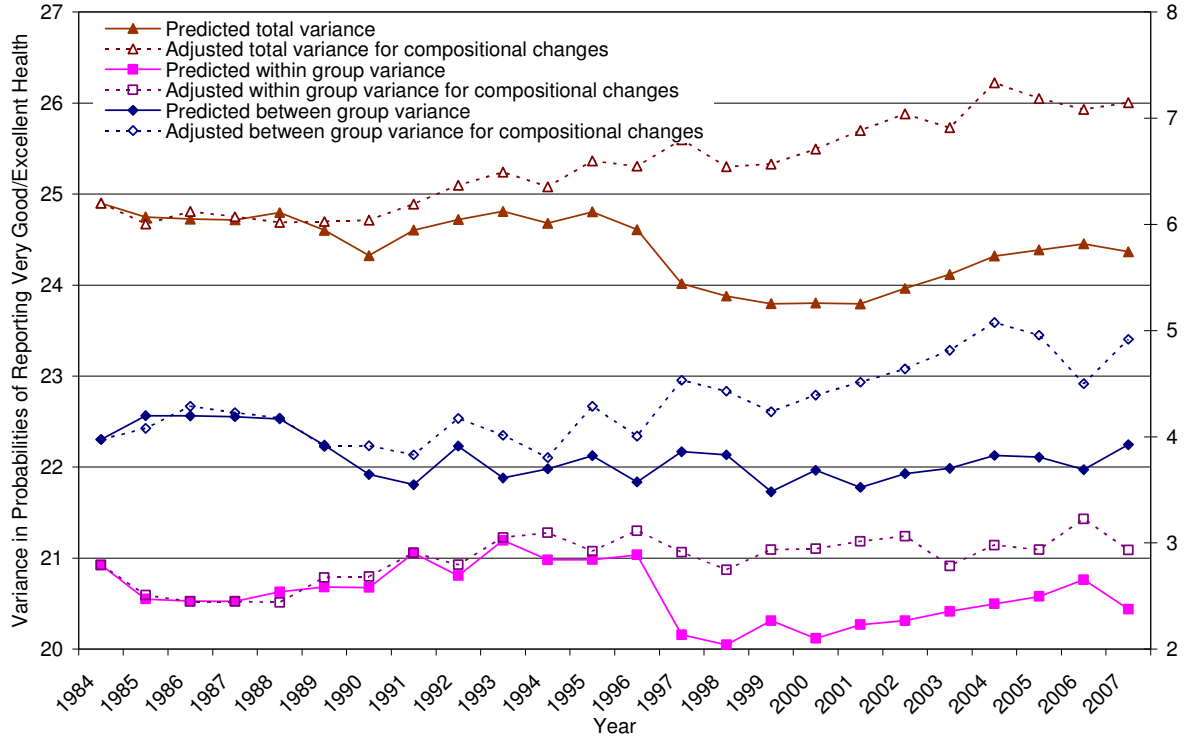


Figure 3. Decomposition of Total Variance into Between-Group Variance and Within-Group Variance, U.S. Women, 1984-2007



1. Adjusted for compositional changes: population compositions fixed at 1984 level.
2. Taking into account the possible increase in variance caused by sampling redesign after 1995, the sharp decrease in within-group disparity from 1996 to 1997 would be smoother since disparity should be lower in 1995 and 1996 than as shown in above figure.