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Income inequality and weight status in US metropolitan areas

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Abstract

Prior empirical studies have demonstrated an association between income inequality and general health endpoints such as mortality and self-rated health, and findings have been taken as support for the hypothesis that inequality is detrimental to individual health. Unhealthy weight statuses may function as an intermediary link between inequality and more general heath endpoints. Using individual-level data from the 1996–98 Behavioral Risk Factor Surveillance System, we examine the relationship between individual weight status and income inequality in US metropolitan areas. Income inequality is calculated with data from the 1990 US Census 5% Public Use Microsample. In analyses stratified by race–sex groups, we do not find a positive association between income inequality and weight outcomes such as body mass index, the odds of being overweight, and the odds of being obese. Among white women, however, we do find a statistically significant *inverse* association between inequality and each of these weight outcomes, despite adjustments for individual-level covariates, metropolitan-level covariates, and census region. We also find that greater inequality is associated with higher odds for trying to lose weight among white women, even adjusting for current weight status. Although our findings are suggestive of a contextual effect of metropolitan area income inequality, we do not find an increased risk for unhealthy weight outcomes, adding to recent debates surrounding this topic.

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Introduction

A large number of empirical studies have suggested that, in addition to individual socioeconomic status, the extent of contextual social inequality may be relevant to health (Kawachi & Kennedy, 1999; Kawachi, Wilkinson, & Kennedy, 1999b; Kawachi, 2000; Subramanian, Blakely, & Kawachi, 2003). Proponents of the income inequality hypothesis argue that for a given

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locality, a greater degree of income inequality is detrimental to the health of its residents. In recent years, this hypothesis has been subject to a fair amount of criticism centered on issues such as confounding of results by individual incomes (Gravelle, 1998; Judge, Mulligan, & Benzeval, 1998; Gravelle, Wildman, & Sutton, 2002), racial composition (Deaton & Lubotsky, 2003), or regional differences (Mellor & Milyo, 2002, 2003); the possibility that inequality merely acts as a proxy for health-determining structural conditions with which it is correlated (Lynch, Smith, Kaplan, & House, 2000; House, 2001); and non-supportive empirical findings (e.g., Mellor & Milyo, 2001, 2002, 2003; Muller, 2002; Osler et al., 2002; Shibuya, Hashimoto, & Yano, 2002; Sturm & Gresenz, 2002). These arguments and findings, however, have been answered and critiqued in return (e.g., Kawachi & Blakely, 2001, 2002; Marmot & Wilkinson, 2001; Blakely & Kawachi, 2002; Blakely, Lochner, & Kawachi, 2002; Wilkinson, 2002; Subramanian et al., 2003).

We examine the relationship between income inequality in US metropolitan areas and three weight status measures, and also declared weight loss behavior. Our work accounts for prior methodological criticisms. The health outcomes commonly investigated are very general endpoints such as life expectancy, mortality, and self-rated health. To the degree that prior work supporting a detrimental effect of inequality on these outcomes is valid, weight status may function as an intermediary link between inequality and more general health measures. Overweight and obesity are well known to be associated with various morbidities and functional limitations (NHLBI Obesity Education Initiative Expert Panel, 1998; Must et al., 1999), and may also be associated with an increased risk of mortality (Harris et al., 1988; Stevens et al., 1992; Lee, Manson, Hennekens, & Paffenbarger, 1993; Manson et al., 1995).

Several pathways have been proposed with respect to the link between income inequality and health, and an extension of each to weight outcomes would predict that the higher the degree of income inequality in a community, the greater the burden of obesity (or overweight) and poor health habits leading to higher weight statuses in that community. Some have proposed that income inequality leads to a disinvestment in human capital and public services (Kaplan, Pamuk, Lynch, Cohen, & Balfour, 1996; Kawachi & Kennedy, 1999), an erosion of social capital or "social cohesion," (Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997; Kawachi & Kennedy, 1999), and a sense of relative deprivation (e.g., Wilkinson, 1992, 1996). In areas with lower social capital, e.g., persons may be at higher risk for being overweight because they lack appropriate health information and affective supports, or are less subject to normative social controls over unhealthy behaviors. It should be noted, however, that in the sociological literature, social capital has been shown to have both positive and negative effects on various socioeconomic attainments (Portes, 1998). In the case of relative deprivation, prolonged psychological stress and frustration may lead to chronic exposures to cortisol, which is associated with weight gain. Weight-promoting behaviors may also function as a coping response to stress and frustration. Lastly, we might expect an association between inequality and weight status to be greater at lower individual income levels, since it is primarily persons at the lower end of the income distribution that are adversely affected by pathways such as relative deprivation.

Few studies have considered the role of income inequality on weight outcomes. Kahn, Tatham, Pamuk, and Heath (1998) examine the effect of state-level income inequality on self-reported weight gain at the waist as opposed to other anatomic sites. The outcome assessed is location of weight gain, adjusting for weight status. The authors hypothesize that inequality induces psychological stress, which leads to weight gain in the abdominal region. They find that for men, inequality has a significant but modest positive effect on the odds of gaining weight at the waist. No significant effects are found among women. Our study differs in several respects. First, we focus on weight status itself, rather than area of weight gain, given a particular weight status. Second, we assess inequality at the metropolitan level rather than state level. As we discuss below, the metropolitan area is perhaps a better unit of analysis given the mechanisms being postulated. Third, Kahn et al.'s study is restricted to a specific age range (50-64), and does not include data on individual incomes. We include adults of all ages, and we assess the effect of inequality with adjustments for individual income and several other individual-level covariates.

In another study, Diez-Roux, Link, and Northridge (2000) examine the relationship between income inequality and four cardiovascular disease risk factors, one of which is body mass index (BMI). Adjusting for individual-level income, they find that for women, inequality has a significant, positive association with BMI (among those with household incomes < \$25 K). Results for men are not significant. This study also measures inequality at the state level. Furthermore, the central findings do not adjust for race. Race is known to be significantly correlated with weight status, and is also associated with inequality. For example, black women are more likely than whites to be overweight (Flegal, Carroll, Ogden, & Johnson, 2002), and areas with higher inequality are associated with a higher proportion of black residents (Deaton & Lubotsky, 2003). As race is a potential confounder of the relationship of interest, we stratify all analyses by race as well as sex. $^{\rm 2}$

Methods

Many US studies on inequality and health utilize states as the unit of analysis (e.g., Kaplan et al., 1996; Kennedy, Kawachi, & Prothrow-Stith, 1996; Daly, Duncan, Kaplan, & Lynch, 1998; Kahn et al., 1998; Kennedy, Kawachi, Glass, & Prothrow-Stith, 1998; Diez-Roux et al., 2000; Kahn, Wise, Kennedy, & Kawachi, 2000; Lochner, Pamuk, Makuc, Kennedy, & Kawachi, 2001; Subramanian, Kawachi, & Kennedy, 2001; Muller, 2002). For hypotheses positing that inequality causes various social divisions, however, we would expect inequality measured more locally to have greater salience. It seems unlikely that persons enact status comparisons relative to others across an entire state, and personal or impersonal contact is certainly more likely across smaller areas. On the other hand, assessments of relative status based on mass media representations probably facilitate comparisons at a national or even broader level. In this sense, states may be too small. We assess income inequality at the level of the metropolitan statistical area (MSA). As psychosocial pathways such as relative deprivation and diminished social capital invoke, implicitly or explicitly, some process of individual comparisons, the MSA is perhaps a better choice than the state for a study comparing geographically bound units within the US. We do not use counties for two reasons. First, counties, unlike MSAs, are not defined to capture social and economic integration. Second, it is likely that many persons cross county boundaries in the course of their day-to-day activities. Smaller areas, such as the census tract, are unlikely to manifest any substantive heterogeneity of income.

We note that certain pathways between inequality and health may be less, or perhaps not, dependent on a process of individual comparisons and, furthermore, may operate more so at a state level. Kawachi and Kennedy (1999) have argued that high income disparity may lead to a disinvestment in social and public services because "the interests of the rich begin to diverge from those of the typical family," which engenders a demand for lower taxes and public services among the elite who have greater political clout or influence (p. 221). To the extent that such social spending and tax policies are relevant to health primarily at the state level, the MSA is less ideal as a unit of measurement for inequality. Accepting this limitation, however, there may also be some pertinent social and public policies determined at the metropolitan level and, as stated above, the metropolitan area seems more appropriate for pathways that suggest a process of individual comparisons.

Given the curvilinear or diminishing effects of individual income on individual health, an ecological correlation between income inequality and health outcomes may be driven by the known effects of individual income on health (Gravelle, 1998; Gravelle et al., 2002). In this sense, there may be a compositional effect from individual incomes rather than a contextual effect from inequality per se. We employ ecological measures such as income inequality but assess the weight outcomes of interest at the individual level, with adjustments for individual income and other individual-level characteristics to tease out compositional from contextual effects.

Data sources

1990 United Status Census, PUMS

Measures of income inequality and other aggregatelevel variables are generated with data from the 1990 US Census 5% Public Use Microdata Sample (US Department of Commerce, 1993). This is a 5% subsample of housing units that received the "long-form" of the 1990 Census questionnaire, which contained detailed questions about items such as income. There are individual records for over five million housing units with data on household income in 1989. Household income includes the incomes of the householder and all other persons 15 years or older living in the household. Income includes wages and salary, self-employment income, interests and dividends, net rental or royalty income, social security, public assistance, retirement or disability, unemployment compensation, and other forms of payments and earnings. We use household rather than individual income is used to remain consistent with the vast majority of previous studies on income inequality.

MSA boundaries are intended to identify a large population nucleus along with adjacent communities that have a high degree of economic and social integration with that nucleus. Metropolitan areas are

²In addition to racial composition, racial residential segregation, which is conceptually distinct from racial composition itself, may also relate to income inequality. First, segregation may be influenced by factors that also influence inequality. If, however, segregation leads to higher weight outcomes, one pathway would concern its effects on individual attainments such as education and income, factors which we adjust for in our analyses. A second connection concerns the fact that segregation likely contributes to income *inequality*. In this sense, inequality itself would function as a potential mediator between segregation and health, again suggesting that the relationship between inequality and health is worthy of investigation.

classified as either an MSA or a consolidated area (CMSA) that is divided into primary MSAs (PMSAs). PMSAs are defined when a metropolitan area has more than one million persons, and similarly reflect a cluster with strong internal social and economic links. All households are either in an MSA, in a PMSA, or not in a metropolitan area. We use MSAs and PMSAs (rather than CMSAs) as our level-two units, and we collectively refer to them as "MSAs" or "metropolitan areas."

Some studies (e.g., Fiscella & Franks, 1997; Kennedy et al., 1998) use a measure of inequality based on incomes from the same dataset that captures the health outcome of interest. In contrast, the use of census data greatly improves the assessment of inequality. Census data for income is far more detailed, offers the precision of census data collection techniques, and provides sample sizes several orders of magnitude larger than would be feasible in any independent health survey. Our approach also improves on prior studies that use census data. These studies typically use census data from the summary tape files, which present data in a summarized format of counts of households that fall into various income intervals. Given the interval nature of the data and top-coding at the upper end (\$125,000 in 1990), interpolation procedures are necessary to calculate certain measures of income inequality (e.g., Gini coefficient, Robin Hood index). Using data from the PUMS, we have access to individual household incomes as a continuous variable, and there is no top-coding present in the 5% sample, with a large number of incomes in excess of \$800,000.

Behavioral risk factor surveillance system

Data for individual-level variables are drawn from the Behavioral Risk Factor Surveillance System (BRFSS) survey (Centers for Disease Control and Prevention, 1996-1998). The BRFSS is nationally representative, cross-sectional data collected on a yearly basis by the Centers for Disease Control and Prevention, and it is designed to monitor a wide array of health-related practices and outcomes in the adult population. Data are collected from a probability sample of noninstitutionalized adults for each state through randomdigit-dial telephone surveys. The survey includes data on self-reported height and weight, which are used to calculate BMI, and standard sociodemographic information such as age, sex, race/ethnicity, education, and income. BMI is equal to weight in kilograms divided by the square of height in meters. Respondents are also asked, "Are you now trying to lose weight?" Lastly, there are FIPS codes available for each respondent's county of residence. County information is suppressed for counties with fewer than 50 respondents. Surveys from the years 1996-98 are combined to allow for larger sample sizes in stratified analyses.

MSA identification

County FIPS codes for respondents in the BRFSS are used to identify their metropolitan area of residence (according the 1990 Census MSA definitions). To achieve such identification, a cross-walk file, or custom correlation list, identifying the relation between counties and metropolitan areas was generated from the US Census Bureau 1998 MARBLE geographic database with the Geocorr 3.0 application. Using this dataset, we assign each county to either a specific MSA or the general category of non-MSA. While most county boundaries do not cross MSA boundaries, 33 counties are not contained within one single MSA. If at least 50% of the population in these counties resides in one MSA, the county is assigned to that MSA. Five counties are too dispersed among metropolitan areas to be reasonably assigned to a single MSA, and BRFSS respondents residing in these counties are not included in the analyses.

Excluding residents of Puerto Rico, there are 402,581 respondents in the BRFSS 1996-98. We exclude persons who could not be identified to a metropolitan or nonmetropolitan area: 5518 (1%) persons residing in counties that are not reasonably assigned to one MSA in the cross-walk, 8271 (2%) persons listed as "Don't know/Not sure" or "Refused" for county code, and 107,740 (27%) persons with suppressed county identifiers. Given that suppression is based on a scant number of respondents for the county, it is likely that this group consists predominately of persons who reside in nonmetropolitan areas, thereby attenuating the impact of these missing data as our analyses concern only those who reside in metropolitan areas. Exclusion of all such "unidentified" persons leaves a sample size of 281,052. Of these "identified" respondents, 62,232 (22%) are excluded because they do not live in a metropolitan area, and 19,375 (7%) are excluded because they reside in an MSA that is not represented in the PUMS.³ This leaves a sample size of 199,445 respondents (from 392 counties) identified into 226 different metropolitan areas, with an average of 883 persons per MSA (range of 51-8586).

Income inequality and other MSA-level variables

Income inequality measures are generated from household income in the PUMS data, which contains an average of 12,347 households per MSA in the 226 metropolitan areas of interest. We use the Gini coefficient as the main measure of income inequality to remain consistent with a large number of previous

³The 5% PUMS does not capture every metropolitan area in the US, so some respondents from the BRFSS could not be matched to the PUMS-derived MSA data.

studies on income inequality and health (e.g., Kennedy et al., 1996, 1998; Lynch et al., 1998; Kawachi, Kennedy, & Glass, 1999a; Soobader & LeClere, 1999; Lochner et al., 2001; Subramanian et al., 2001; Muller, 2002; Shibuya et al., 2002; Sturm & Gresenz, 2002). The Gini coefficient ranges from 0 at perfect equality to 1 at maximal inequality. A Gini coefficient based on household income is computed for each of the 226 metropolitan areas, with census household weights incorporated into the calculation. The mean and median of household income are also computed as indices of overall or absolute income status. Population counts for MSAs are computed from the cross-walk file by summing the county (or fraction of county) population counts in each MSA. We also generate other measures of income inequality-the Robin Hood Index (Kennedy et al., 1996), the coefficient of variation, and ratios for percentiles of income-to allow for an assessment of whether or not results are discrepant when other measures are used.

Analyses

Given concerns for confounding by race as well as variation in weight, weight-related behaviors, and preferences by gender and race, we stratify analyses by race-sex groups. Furthermore, preliminary analyses revealed multiple significant interactions between race, sex, and other covariates. Although stratified analyses do not permit an examination of racial discrepancies in weight outcomes, this is not the focus of our study. The investigation is restricted to non-Hispanic whites and non-Hispanic blacks, the two groups in the BRFSS with larger sample sizes. Of the 199,445 respondents the BRFSS successfully linked to an MSA in the PUMS, 174.858 are either non-Hispanic black or white. Of this group, 1998 women (1%) are excluded because they were pregnant at the time of the survey, and 6055 persons (3%) are excluded because they are missing data for the calculation of BMI. Additionally, 211 persons (<1%) are excluded because there are missing data on education, followed by 22,663 persons (13%) missing data on household income. This leaves an overall sample size of 143, 931, consisting of 68,545 white women, 56,263 white men, 12,200 black women, and 6,923 black men.

We model BMI as a dependent variable with two-level hierarchical linear models using HLM 5.05 software. Here, individuals at level one are nested within metropolitan areas at level two, and metropolitan area characteristics are modeled as level-two variables. With BMI as a level-one dependent variable, level-one predictors are centered on their grand means, and the variance components of their slopes are fixed at level two. This allows for the assessment and modeling of the variation in BMI between MSAs, adjusted for differences between MSAs in individual-level variables (or compositional effects), which is reflected in the variation of the level-one intercept. MSA-level variables (as contextual effects) are then modeled as predictors for the level-one intercept. BRFSS sampling weights, which adjust for unequal coverage and response rates among segments of the population, are included in these analyses.

MSA Gini, median income, and population are modeled as continuous variables. MSA population size is logged because its distribution is heavily skewed to the right. Individual age is modeled as a continuous variable and includes a squared term, as preliminary analyses indicate significant non-linear effects on BMI. Individual household income is also modeled as a continuous variable. In the BRFSS, data on household income is provided in the form of eight intervals of income, with the highest category listed as "\$75,000 or more". Respondents are assigned an income corresponding to the midpoint of their income interval. For respondents in the last, open-ended category, a Pareto estimate for the median of this category (\$104,789) is used (Parker & Fenwick, 1983). Income is also logged. Education and the four census regions of residence are modeled as categorical variables with dichotomous indicators. We include census regions as fixed effects to adjust for potential confounding of an association between metropolitan income inequality and weight by unmeasured regional factors. Mellor and Milyo (2002, 2003) have argued that it is important to control for region since determinants of both health outcomes and inequality vary across regions in the US. Hence, we examine variation between MSAs adjusted for region.

In addition to modeling BMI as a dependent variable, we examine the effect of income inequality on whether or not respondents are in fact overweight (BMI \geq 25), and whether or not they are obese (BMI \geq 30). Lastly, we examine the effect of inequality on whether or not respondents stated that they were currently trying to lose weight, adjusting for current weight status (BMI). In doing so, we examine not only objective weight outcomes, but also weight-related behavior, net of actual weight status. The question on trying to lose weight, however, is not part of the fixed, core component of the BRFSS; while all states included the question in 1996 and 1998, only five states included it in 1997. Thus, these analyses are restricted to a total sample of 99,847 persons, which also reflects the exclusion of 236 persons who refused to answer the question or answered with "Don't know/Not sure." These three dichotomous outcomes are analyzed with multivariate logistic regression models conducted in STATA 8.0 software rather than HLM, because current HLM software cannot accommodate survey sampling weights for generalized

Table 1		
BRFSS sample characteristics	by subgroup	$(N = 143,931)^{a}$

Variable	Mean (SD) or %			
	White women	White men	Black women	Black men
BMI	24.9 (5.5)	26.4 (4.0)	27.7 (6.6)	27.0 (4.1)
Overweight (BMI≥25)	39.9	61.5	63.0	65.0
Obese (BMI≥30)	14.3	16.0	29.1	20.7
Age (years)	46.8 (18.6)	44.5 (15.7)	42.4 (16.5)	41.1 (13.4)
Household income (\$1000)	46.9 (32.5)	52.5 (29.0)	32.5 (26.3)	37.9 (23.0)
Education				
Some HS or less	7.5	6.9	14.6	13.7
HS grad.	31.1	27.2	33.2	34.4
Some college or tech.	30.3	28.4	31.7	31.2
College grad.	31.2	37.5	20.4	20.7
Census region				
Northeast	22.1	21.8	19.4	19.2
Midwest	25.1	24.5	23.6	22.6
South	27.0	28.0	43.3	45.1
West	25.8	25.7	13.7	13.1
Subgroup N	68,545	56,263	12,200	6923

^aData reflect sampling weights.

linear models. For these models, however, we report robust standard errors adjusted for clustering, or potential non-independence, within MSAs.⁴

Given the expectation that a detrimental effect of inequality would be strongest among those who are most disadvantaged, we test for an interaction between inequality and individual income. Given the concern for confounding of the effect of inequality by individual income, results are checked for sensitivity to alternative specifications of the income variable, such as including higher-order terms and modeling it as a categorical variable. The potential of bias from excluding persons missing on the income variable is checked by ensuring that the results are robust to assigning all such persons either the lowest or the highest income category. Models are also assessed with alternative measures of income inequality such as the Robin Hood Index, the coefficient of variation, and ratios of percentiles of income.

Results

Sample characteristics

Table 1 summarizes individual characteristics for each subgroup in the BRFSS sample. White women have the lowest mean BMI (24.9), as well as the lowest fraction of overweight (39.9%) and obese (14.3%). Black women have the highest mean BMI (27.7), and the highest fraction of obese (29.1%). Black men, however, have the highest percentage of overweight (65.0%). Region of residence is fairly evenly distributed for whites, but blacks show disproportionately large numbers residing in the South. Table 2 summarizes characteristics of the 226 metropolitan areas. The mean Gini coefficient is 0.41 with a range of 0.35–0.47. Joliet (IL) and York (PA) are examples of metropolitan areas with the lowest coefficients. New Orleans (LA), New York (NY), and McAllen-Edinburg-Mission (TX) are examples of areas with the highest coefficients.

Body mass index

For all four subgroups, a one-way ANOVA model with BMI as the dependent variable and a random effect for the intercept demonstrates significant (p < 0.001)

⁴We also checked all linear and generalized linear models for sensitivity to adjustments for survey design elements using the SVY_ commands in STATA. As standard errors and conclusions were not altered in any meaningful fashion, we present results from the main models as described.

Table 2 Metropolitan area characteristics (N = 226)

Variable	Mean (SD)	Range
Median household income (\$1000)	29.7 (5.7)	16.9-51.0
Mean household income (\$1000)	36.2 (6.4)	24.0-60.6
Population size (100,000)	7.8 (11.4)	1.1-88.6
Gini coefficient (household income)	0.41 (0.02)	0.35-0.47

Table 3 Coefficient for MSA Gini from models for body mass index

	Fixed effect ^a (SE)		
	(1) ^b	(2) ^c	(3) ^d
White women $(N = 68,545)$	-1.322	-1.000	-0.851
	(0.197)**	(0.178)**	(0.202)**
White men $(N = 56, 263)$	-0.235	-0.142	-0.156
	(0.150)	(0.144)	(0.172)
Black women ($N = 12,200$)	-0.684	-0.701	-0.273
	(0.483)	(0.467)	(0.541)
Black men (N = 6923)	-0.123	-0.098	0.107
	(0.449)	(0.453)	(0.514)

**P<0.01.

 ${}^{a}\beta$ = change in BMI estimated for a 0.1 unit increase in the Gini coefficient.

^bModel adjusts for MSA median income and age.

^cModel adjusts for MSA median income, age, individual household income, and education.

^dModel adjusts for MSA median income, age, individual household income, education, MSA population, and region.

variation between metropolitan areas in BMI, and the estimated variance components are as follows: white women 0.320, white men 0.174, black women 0.780, and black men 0.473. As expected, the proportion of total variance that is between metropolitan areas is small (1-2%) relative to the proportion that is within metropolitan areas. Table 3 displays the coefficient or fixed effect for MSA Gini from models with BMI as the dependent variable and varying combinations of covariates. Model 1 examines the effect of MSA inequality on BMI, adjusting only for MSA median income and individual age. Model 2 additionally adjusts for individual-level income and education, and model 3 further adjusts for MSA population size and census region. The Gini coefficient is scaled so that its regression coefficient represents the change in BMI estimated for a 0.1 unit increase in Gini. For white women, a 0.1 unit increase in Gini is associated with a -1.322 unit decrease in BMI in model 1. Thus, higher income inequality at the MSA level actually predicts lower, rather than higher, BMI at the individual level. Additional adjustment for individual household income and education in Model 2 results in an attenuation of the effect of Gini to -1.000, and further adjustment for MSA population size and census region in Model 3 attenuates the effect of Gini to -0.851 (p < 0.01). For white men, higher inequality also predicts lower weight status, but the estimates are not significant (p > 0.05), even in Model 1. For black women, an increase in inequality is again associated with lower weight status, but the estimates are not significant. The attenuation in moving from model 2 to model 3 is primarily due to the inclusion of regional controls. For black men, higher inequality is inversely associated with BMI in models 2 and 3, but positively associated in model 3. The estimates, however, are not significant. Table 4 displays all variables in model 3 for each subgroup. For white women, the effect (on BMI) of a 0.1 unit increase in Gini is close in magnitude to the effect of graduating from college versus having only a high-school diploma (or having some college or technical school). In sum, increased metropolitan income inequality is associated with lower BMI in three of the four subgroups, but is only significant among white women.

Table 4				
Models	for	body	mass	index

	Fixed effect (SE)			
	White women	White men	Black women	Black men
MSA Gini ^a	-0.851	-0.156	-0.273	0.107
	(0.202)**	(0.172)	(0.541)	(0.514)
MSA median income ^b	-0.295	-0.183	-0.082	-0.117
	(0.086)**	(0.076)*	(0.249)	(0.241)
MSA population ^c	0.021	-0.027	-0.127	-0.029
	(0.043)	(0.036)	(0.112)	(0.117)
Age	0.368	0.324	0.378	0.205
	(0.009)**	(0.008)**	(0.029)**	(0.030)**
(Age) ²	-0.003	-0.003	-0.004	-0.002
	(<0.001)**	(<0.001)**	(<0.001)**	(<0.001)**
Individual income ^d	-0.776	0.122	-0.606	0.361
	(0.055)**	(0.040)**	(0.103)**	(0.155)*
Education: HS or less	1.781	0.874	2.468	0.498
	(0.160)**	(0.139)**	(0.268)**	(0.274)
HS graduate	0.937	0.677	1.384	0.130
	(0.086)**	(0.059)**	(0.233)**	(0.199)
Some college or tech.	0.817	0.570	0.935	0.093
	(0.072)**	(0.057)**	(0.235)**	(0.210)
College graduate	—	—	—	—
Region: Northeast	0.189	0.389	0.483	0.090
	(0.112)	(0.105)**	(0.413)	(0.304)
Midwest	0.367	0.251	1.279	0.763
	(0.110)**	(0.097)*	(0.422)**	(0.231)**
South	-0.156	0.137	0.652	0.606
	(0.110)	(0.100)	(0.398)	(0.216)**
West		—		—
N	68,545	56,263	12,200	6923

P*<0.05; *P*<0.01.

 ${}^{a}\beta$ = change in BMI estimated for a 0.1 unit increase in the Gini coefficient.

^b\$median income/10,000.

^cln (population/100,000).

^dln (\$household income).

Overweight, obese, and trying to lose weight

Table 5 shows the results of multivariate logistic regression models for the odds of being overweight $(BMI \ge 25)$ and being obese $(BMI \ge 30)$. In contrast to the models for BMI, these models assess whether or not persons are actually in weight categories defined, according to clinical and public health standards, as problematic for health. For white women, a 0.1 unit increase in Gini is associated with 0.71 times lower odds of being overweight and 0.73 times lower odds for being obese. For white men, black women and black men, estimates for effect of inequality on being overweight and obese are not significant (p > 0.05). Table 5 also displays the results of logistic regression models for the odds of currently trying to lose weight, adjusting for current weight status (in addition to the other covariates). For white women and men, MSA Gini

is positively associated with the odds of trying to lose weight. For women, a 0.1 unit increase in Gini is associated with 1.40 times higher odds of trying to lose weight, and for white men, the odds are increased by a factor of 1.39. For blacks, estimates for the effect of inequality are not significant.

As lower weight status may reflect ill health or an increase in unhealthy but potentially weight-reducing behaviors such as smoking, the results for BMI, overweight, and obese are checked with adjustments for self-reported health status and smoking status. The inclusion of these controls does not alter findings in any substantive manner. All models are also tested for an interaction between the Gini coefficient and individual household income. No significant interactions are found among any of the subgroups. Further inspection by stratifying regressions on levels of income does not reveal any consistent trends

	Odds ratio [95% CI]		
	Overweight	Obese	Lose weight ^b
White women	0.71 [0.61–0.84]**	0.73 [0.59–0.90]**	1.40 [1.14–1.71]**
Ν	68,545	68,545	47,767
White men	0.87 [0.72–1.06]	0.95 [0.77–1.19]	1.39 [1.08–1.79]*
Ν	56,263	56,263	39,095
Black women	1.12 [0.82–1.54]	0.92 [0.61–1.40]	1.03 [0.63–1.69]
Ν	12,200	12,200	8242
Black men	0.94 [0.55–1.61]	0.83 [0.49–1.40]	1.00 [0.47–2.12]
Ν	6923	6923	4743

Table 5 Odds ratio for MSA Gini from logit models for overweight, obese, and currently trying to lose weight^a

P*<0.05; *P*<0.01.

^aAll models control for age, individual household income, education, MSA median income, MSA population, and region. ^bModel additionally adjusts for body mass index.

with respect to the effect of inequality on weight outcomes.

Table 6 Models for BMI using different measures of inequality^a white women (N = 68.545)

Measure of inequality	Fixed effect ^b	(SE)
Gini coefficient	-0.197	(0.047)**
Robin Hood index	-0.185	(0.049)**
Coefficient of variation	-0.237	(0.046)**
90th:50th percentile	-0.197	(0.045)**
90th:10th percentile	-0.082	(0.050)

***P*<0.01.

^aAll models control for age, individual household income, education, MSA median income, MSA population, and region.

^bScaled to represent change in BMI estimated for a one standard deviation increase in each measure of inequality.

ment. Given the potential for confounding by individual income, results are also checked for sensitivity to alternative specifications of the individual income variable, modeling it as a polynomial function and as a categorical variable. Point estimates for the effect of income inequality are minimally affected by these alternative specifications, and conclusions with respect to the direction of association and significance testing remain the same.

Discussion

Most studies on inequality and health have examined general endpoints such as mortality and self-rated health

Sensitivity analyses

The results are checked for sensitivity to alternative measures of income inequality such as the Robin Hood Index, the coefficient of variation, and ratios of percentiles of income. Correlations between the various inequality measures are generally quite high, ranging from 0.71 to 0.99. Table 6 displays hierarchical linear models for BMI (in white women) using different measures of inequality, with each scaled so that its fixed effect represents the change in BMI estimated for a one standard deviation increase in the inequality measure. Like the Gini coefficient, the Robin Hood Index, the coefficient of variation, and the 90th:50th percentile ratio all show a significant inverse association with BMI. Compared to the 90th:10th ratio, the 90th:50th ratio may be more "affluence sensitive," reflecting dispersion at the high end (Daly et al., 1998).

A notable number of persons are excluded because there are missing data on household income in the BRFSS. Results are checked for the extent of potential bias from this exclusion by analyzing two additional datasets. In one, all persons missing on income are assigned to the highest income level. In the other, all such persons are assigned to the lowest income level. All models for all subgroups are then re-executed on each of these datasets. For the predictor of interest, MSA inequality, estimates are quite similar and conclusions remain unchanged at each extreme of income assignstatus. In this study, we narrow the scope to weight status as a specific outcome and find that, at least for non-Hispanic white women, living in a metropolitan area with greater income inequality is associated with lower BMI, lower odds for being overweight, and lower odds for being obese. It is also associated with greater odds for trying to lose weight, adjusting for current weight status. We find no significant effect of income inequality on weight status in the other race-sex groups. In recent years there has been a fair amount of debate on the relationship between inequality and health, with some critics charging that income inequality is not in fact associated with individual health outcomes (Mellor & Milvo, 2001, 2002; Mackenbach, 2002). Our findings suggest that this is not unequivocally the case. Among proponents of the income inequality hypothesis, however, it is generally thought that inequality results in worsened average health (Kawachi & Kennedy, 1999; Kawachi et al., 1999b). Intermediary mechanisms such as disinvestments in human capital, the erosion of social capital, and relative deprivation largely suggest that inequality leads to unhealthy behaviors and poor health. We do not find a positive association between inequality and the likelihood of clinically relevant outcomes such as overweight and obesity. Indeed, the direction of association between inequality and weight status is generally negative among subgroups (though significant only for white women). Furthermore, the negative association does not appear to reflect reduced weight from ill health or unhealthy behavior such as smoking. Hence, income inequality may benefit (or be protective for) white women, providing that it is not associated with an excess of underweight.

Thus, in contrast to previous work reporting that income inequality has an adverse effect on outcomes such as mortality and general health status, we do not find evidence for a health-impairing effect on weight status. One source for this difference may be that previous work measures inequality at the state level (e.g., Kaplan et al., 1996; Kennedy et al., 1996, 1998; Kawachi & Kennedy, 1997; Kahn et al., 2000; Lochner et al., 2001), while this study looks across metropolitan areas. As previously discussed, however, conventionally proposed pathways directly or indirectly imply a process of mutual, social comparison in terms of income or other features associated with income. As such, exposure (or contact) is critical for this process, and it seems more likely to occur among coresidents within the same metropolitan area than among persons more remotely scattered across an entire state. A small number of other studies have measured inequality across metropolitan areas. Some find that inequality has a detrimental effect on mortality but are ecological in design (Lynch et al., 1998; Ross et al., 2000; Sanmartin et al., 2003). Our findings suggest that weight-related morbidity may not function as a mediator in this association. Consistent

with our findings, other multi-level studies on metropolitan areas have found no significant risk increase for poor health status (Blakely et al., 2002; Mellor & Milyo, 2002) or chronic medical conditions (Sturm & Gresenz, 2002). The fact that negative health effects tend to appear at the state rather than more local levels has prompted the suggestion that state-level associations may be confounded by state-wide policies towards the poor, policies which contribute to both health outcomes and income inequality (Wagstaff & van Doorslaer, 2000).

In a multi-level study of income inequality and cardiovascular risk factors, Diez-Roux et al. (2000) find that for women, higher inequality, as measured by the Robin Hood Index, predicts higher BMI. This contrasts with our study, which finds the opposite relationship in white women, regardless of whether the Gini coefficient or the Robin Hood Index is used. There are key differences, however, between the two studies. First, Diez-Roux, Link and Northridge measure inequality at the state level rather than the metropolitan level. Second, their finding is not adjusted for race. We stratify our analyses by race because it is associated with both inequality and weight status-areas of high inequality have a higher proportion of black residents (Deaton & Lubotsky, 2003), and black women have a higher weight status on average than white women (Flegal, Carroll, Kuczmarski, & Johnson, 1998; Flegal et al., 2002). Although the authors chose to present unadjusted estimates in their tables, they do note that when they adjust for race, inequality predicts lower BMI for women, which is more consistent with what we find at the metropolitan level.

As pathways from the inequality and health literature are predominantly formulated to account for adversity in health outcomes, our findings for white women, which were robust to several analytical checks, require a different framework for interpretation. Here, we merely speculate on one manner by which these findings might be interpreted. In conceptualizing health-relevant behaviors, conventional medical and public health models often focus on the pursuit of health benefits subject to resource and other instrumental-type constraints (Chang & Christakis, 2002). This conceptualization however, overlooks the potentially important role that body size and appearance play in broader regimes of social status. In the context of a cultural economy that values thinness is over fatness along moral and aesthetic dimensions the body can serve as a repository for individual differentiation and status distinctions. Body size is not just a matter of health; it is also a salient, and often conspicuous, source of social status. Moreover, bodily form can affect life chances, operating, like cultural capital, as an informal basis for contemporary social closure practices, which function to delimit individual attainments. Preliminary data from economic studies have shown that weight status can affect attainments ranging from educational level to wages and household income for white women (Register & Williams, 1990; Gortmaker, Must, Perrin, Sobal, & Dietz, 1993; Sargent & Blanchflower, 1994; Averett & Korenman, 1996; Cawley, 2000). Given these considerations, one could speculate that in areas of greater income inequality, women are more likely to use the body as an alternative source of social status. With higher inequality, any form of status competition (or emulation) on the basis of income is relatively more difficult for persons who are not at the upper end of the distribution. As suggested by previously proposed mechanisms for the effect of inequality, there may be perceptions of relative deficits, but rather than assuming that it uniformly leads to ill health, we might also consider how it renders the body more salient as a compensatory means to social status. While the standard literature on inequality and health considers the role of social positioning via individual income, it does not consider the body itself as a form of status positioning.

We have suggested merely one approach to interpreting the findings of this study. We do not mean suggest that it is necessary to embrace a social status model for the relationship between inequality and health as a whole, and there are, no doubt, other ways to interpret these findings. For example, they may reflect spillover effects from the wealthy. Daly et al. (1998) have argued that an increase in the number of affluent families in a given area may in fact have positive health impacts for all residents. In this sense, it is not inequality per se that leads to better health, but the fact that these areas have a greater proportion of highly wealthy persons who affect or contribute to the community in terms of the quality and quantity of public resources that are relevant to health. Alternatively, these findings may reflect differences between metropolitan areas in ethnic composition. Areas with a high degree of inequality may have a relatively high percentage of new immigrants who are less likely to be overweight or obese. Adjusting for the percentage of foreign-born residents in each MSA, however, did not have any substantive effect on the association between inequality and weight status. Income inequality may also function as a proxy for geographically patterned cultural differences in dietary and other lifestyle preferences, or bodily aesthetic standards that do not derive from inequality per se. We adjust for census region, but there may be residual influences within region. Lastly, it may be the case that persons of lower weight status or a certain disposition towards bodily appearance are more likely to move to areas of high inequality.

The notion of relative deprivation and a process of status comparisons suggest that the impact of inequality should be stronger for those who are more disadvantaged. In this study we do not find a significant interaction between income inequality and individual income, despite multiple model specifications. To some extent, this constitutes evidence against the contribution of such processes to these results. Alternatively, it is possible that all but those at the very highest tiers of income experience comparable status deficits, with all upholding the highest tier as the point of reference. If this is true, interaction effects may not be detectable using the BRFSS data, wherein information on individual household income is top-coded at \$75 K, precluding any discrimination between income groups above this level.

We do not find a significant association between inequality and weight outcomes among blacks. Coefficient estimates for the inequality variable are largely attenuated and rendered non-significant with the inclusion of regional controls, indicating strong confounding effects from regional differences. It may be the case that persons tend to judge income inequality relative to persons within their own race/ethnic group. Despite high overall inequality in some metropolitan areas, there may be relatively few high-income blacks. There might also be an effect from racial segregation at the neighborhood level. High-inequality areas may have greater segregation, with many blacks living in racially and economically segregated neighborhoods where they have relatively little contact with high-income persons. Lastly, the sample size is much smaller for blacks than it is for whites, perhaps contributing, to some extent, to wider confidence intervals.

There are several limitations to this study. First, height and weight are self-reported rather than directly measured in the BRFSS, allowing for the possibility of misreporting at the extremes of BMI. Many, however, have concluded that self-reports are an excellent approximation for actual values, with self-reports showing extremely high correlations with measured values (Stunkard & Albaum, 1981; Stewart, 1987; Jeffrey, 1996). Others have shown that self-reports may bias analyses when weight status is assessed as a categorical variable, but bear little effect on analyses when weight status is modeled as a continuous variable (Kuskowska-Wolk, Bergstron, & Bostrom, 1992). Furthermore, this is the same dataset used for recent estimates of the prevalence of overweight and obesity in the US (Mokdad et al., 1999, 2001). Other data such as the National Health and Nutrition Examination Survey offer a nationally representative sample with measured weight status but do not permit the identification of respondents into a sufficient number of metropolitan areas. Second, there is more than a half-decade of time difference between the measure of inequality and the measure of weight status. As such, it is not known how inequality may have changed in the intervening years in a manner that may influence results. We did, however, wish to preserve the temporal ordering of inequality as a

causal factor and allow for a substantive time lag between inequality and the measure of health. It should also be noted that individual characteristics such as income, race, and perhaps even preferences relating to weight, may influence place of residence. Lastly, the BRFSS data do not allow for the differentiation of household incomes above \$75 K.

In conclusion, we find that the distribution of income in one's area of residence, a contextual factor, can be associated with individual weight-related health outcomes above and beyond the effects of other individual characteristics. This is an important supplement to conventional medical models of risk assessment, which focus primarily on individual-level factors. In contrast, however, to advocates of the income inequality hypothesis, we do not find that inequality leads to worsened or poor health. Hence, if it is the case that inequality is associated with higher mortality and lower overall health status, then unhealthy weights likely do not play an intermediary role. Contrary to the tendency to suppose that inequality is detrimental to health in a uniform fashion, our findings suggest that further work is needed to assess how and why some outcomes may differ from others, and, furthermore, the potential role of cultural and symbolic dimensions.

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