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To cite this article: Celia C. Lo, William Ash-Houchen, Heather M. Gerling & Tyrone C. Cheng (2018): Data spanning three decades illustrate racial disparities in likelihood of obesity, Ethnicity & Health, DOI: 10.1080/13557858.2018.1447650

To link to this article: https://doi.org/10.1080/13557858.2018.1447650

Published online: 04 Mar 2018.
Data spanning three decades illustrate racial disparities in likelihood of obesity

Celia C. Lo\textsuperscript{a}, William Ash-Houchen\textsuperscript{a}, Heather M. Gerling\textsuperscript{a} and Tyrone C. Cheng\textsuperscript{b}

\textsuperscript{a}Department of Sociology and Social Work, Texas Woman’s University, Denton, TX, USA; \textsuperscript{b}Department of Social Work and Child Advocacy, Montclair State University, Montclair, NJ, USA

\textbf{ABSTRACT}

Obesity rates have risen significantly in recent decades, with underprivileged Americans associated with higher rates of the condition. Risks associated with obesity, furthermore, appear unequally distributed across different racial/ethnic groups, according to the literature. The present study examined racial disparities in obesity as a function of socioeconomic factors, using a sample of American adults from a 32-year longitudinal study. We accounted for the time factor as we evaluated obesity’s associations with selected socioeconomic factors; we also examined race/ethnicity’s moderating role in obesity–socioeconomic status associations over time. We used data from the National Longitudinal Survey of Youth (NLSY) to obtain a final sample of 118,749 person-waves for analysis. A subsample of person-waves numbering 65,702 represented data from White respondents; one numbering 31,618 represented data from Black respondents; and one numbering 21,429 represented data from Hispanic respondents. Needing to consider repeated measures of the same variables over time, we chose generalized estimated equations (GEE) for use in the data analysis. Speaking generally, the obtained results suggested that for the two smaller subsamples, minority race/ethnicity could have introduced disadvantages that helped explain links between obesity and race/ethnicity. Results also showed that White–Black disparities in obesity have widened slightly in the past three decades, while White–Hispanic disparities have stabilized during the same time period.

\textbf{Introduction}

Health disparities have characterized the United States for decades, and race/ethnicity plays a role in health disparities – even those that may not immediately appear related to race (Williams and Jackson 2005). Non-Hispanic Blacks have been associated with higher likelihood (versus non-Hispanic Whites) of dying from chronic illnesses such as heart disease and cancer, the top causes of death in the United States (Heron 2016; Heron and Anderson 2016). Hispanics, in turn, have been associated with lower likelihood (versus Whites or Blacks) of dying from either disease (National Center for Health...
Obesity has long been described as a health-risk behavior contributing to chronic conditions like heart disease and cancer (Lamon-Fava, Wilson, and Schaefer 1996; Bell, Adair, and Popkin 2002; Flegal et al. 2007); it often results from sedentary lifestyles and poor food choices (McCracken and Blanck 2007). Generally, obesity is more prevalent among Blacks and Hispanics than Whites, and obesity is also associated with lower as opposed to higher socioeconomic status (SES) (Kahng 2010; Fang, Shaw, and Keenan 2011; National Center for Health Statistics 2016).

The present study drew on a prospective longitudinal survey launched in 1979 that captured a national sample of Americans born 1957–1964, describing respondents’ likelihood of obesity as well as socioeconomic status (among other things). The present study sought to understand whether and how, for these respondents, obesity’s associations with poverty, education, family assets, and other status factors persisted over three decades. Previous research with national samples observed socioeconomic factors to interact with race/ethnicity in explaining likelihood of illness and likelihood of health. Specifically, an observed link between family income and level of glycosylated hemoglobin, an indicator of type 2 diabetes, was significantly stronger among Whites than Blacks (Lo, Lara, and Cheng 2017). Additionally, in a prior longitudinal study, increasing education promoted higher self-ratings of health among Whites but did not change such ratings among Blacks (Farmer and Ferraro 2005). Race/ethnicity is a marker for exposure to health-related disadvantages; moreover, the socioeconomic patterns that help explain various racial/ethnic groups’ obesity rates may be quite distinct (Williams and Jackson 2005). For these reasons, social patterns of obesity needed to be evaluated separately for Whites, Blacks, and Hispanics in our study. Race/ethnicity’s role in such patterns over time also required separate evaluation by ethnicity. Often in the social sciences, Black is considered a race and Hispanic an ethnicity. In the present study, Black and Hispanic subsamples, as well as a White subsample, were analyzed. The phrase ‘racial/ethnic group(s)’ is used throughout this manuscript to indicate one or more of the three analyzed subsamples.

Obesity’s associations with race/ethnicity and SES are well-studied (Nguyen, Moser, and Chou 2014; Wong, Chou, and Ahmed 2014). Nevertheless, the present study brings three significant contributions. The first arises from our focus on how SES relates to obesity for Whites, Blacks, and Hispanics discretely. This unusual focus guided us to try to see whether and how SES and race/ethnicity intersect and together exert influences on obesity risk (for men and for women), ultimately seeking race/ethnicity’s moderating role in obesity, if such exists. The second significant contribution involves our choice to let availability of family assets (wealth) indicate SES in our analyses, an approach seldom encountered in the extensive literature documenting the SES–obesity association. Measuring such availability was significant because greater family wealth more often characterizes Whites than it does minority Americans (Williams and Collins 1995), making family assets a potentially important indicator of obesity likelihood. Our study’s third significant contribution is its longitudinal data. With few exceptions (Gustafsson, Persson, and Hammarstrom 2012; Wong, Chou, and Ahmed 2014), research on obesity’s associations with SES and race/ethnicity has been of cross-sectional nature (Nguyen, Moser, and Chou 2014). Our longitudinal data from a 30-year period permitted an atypical study that could test these associations across decades when the prevalence of obesity in the United States was steadily rising.
Literature review and hypotheses

We undertook this research because we want to see a discussion of health’s fundamental causes brought to the forefront of the health-risk literature. Health’s fundamental causes are socioeconomic status and other social status factors; from our perspective, they are the key factors in Americans’ health (Link and Phelan 1995). Individuals who occupy the bottom social strata for extended periods are exposed to resource deprivation – including deprivation of preventive-care access – and the accompanying chronic stress, which fosters worse health compared to that of those in higher strata (Kessler, Mickelson, and Williams 1999; Phelan and Link 2005; Virtanen et al. 2008; Pavlova and Silbereisen 2012). Furthermore, lower SES (typically associated with little education, menial work, and low income) is linked to relatively slow acquisition of information that can change the beliefs, attitudes, and lifestyles that tend to diminish health (Link and Phelan 1995).

In the United States, having minority ethnicity – being Black, Hispanic, or a non-White American of other race/ethnicity – is linked to many social, economic, and health disadvantages (Lo and Cheng 2012; Lo, Cheng, and Howell 2014a). The sources of these disadvantages have been institutionalized and patterned through social structural factors (e.g. education, job loss) that are distributed unequally across racial/ethnic groups; minority Americans are, for example, more likely than White counterparts to have low SES and lack social resources (Williams and Jackson 2005). Decades of structural and institutionalized racism in our society mean minority Americans, especially Blacks, live with three disadvantages: (1) They are confined to residential areas that are physically segregated from gainful employment and from full access to health care (Wilson 1987; Williams and Collins 2001); (2) They are exposed to socially disorganized neighborhoods offering relatively little access to physical-activity opportunities or healthy grocery shopping (Zick et al. 2009; Christian et al. 2017); (3) They have learned mistrust of ‘the system,’ to include the health-care system in place to maintain and improve health (Williams and Mohammed 2009; Williams and Sternthal 2010). Residential segregation, social disorganization, and mistrust of institutions all feed racial disparities in health-risk behaviors and illness-health.

The multiple disadvantage model views minority race/ethnicity as a disadvantage to health, and was developed to illustrate the importance of social location and social integration factors in the explanation of lifestyles and mental health – specifically, for roles that lifestyle and mental health may hold in homicide victims’ age at demise (Lo, Howell, and Cheng 2013b). Research has used the model to explain racial/ethnic differences in lifestyles, especially concerning substance use; the research has identified both additive and multiplicative impacts of social structural and resource factors on lifestyle and health outcomes (Lo, Howell, and Cheng 2013a; Lo, Cheng, and Howell 2014b). Previous research demonstrates the need to consider racial differences in health simultaneously with gender and other ascribed statuses that are intersectional; that is, to consider the effect of minority race/ethnicity alongside other, potentially multiplicative, effects such as those of gender and SES (Etherington 2015). Minority Americans’ health may be further disadvantaged by their exposure to structural and perceived discrimination (Williams and Sternthal 2010), which seems to reduce returns on investments like education (Farmer and Ferraro 2005). The literature demonstrates that the health benefits
of having relatively much education are diminished for minority Americans compared to Whites (Williams and Collins 1995; Farmer and Ferraro 2005).

Link and Phelan (1995) outlined how advancing knowledge and technology related to health and disease should, initially, promote the health of individuals with plentiful resources like money, power, prestige, knowledge, and privileged social networks: that is, of Whites and those with higher SES. Eventually, though, the health of less ‘resourced,’ less powerful Americans should also benefit: that is, Blacks and those with lower SES. Link and Phelan’s fundamental cause perspective suggests how, despite contemporary advances in the prevention and treatment of heart disease, White–Black disparities in the associated mortality rates have widened steadily for five decades now (Phelan and Link 2005). The perspective also explains changing trends in health risks such as cigarette smoking – which rather evenhandedly killed people in all socioeconomic groups, until science could inform consumers (some better than others) of cigarettes’ role in lung cancer (Phelan and Link 2005). Information on the harm associated with cigarettes led first to behavioral change at higher socioeconomic statuses (Phelan and Link 2005). As understanding of such harm has begun to reach broader populations, behavioral change among those in lower socioeconomic strata has begun as well.

Concerning obesity in recent decades, as rates of cigarette use have fallen, obesity rates have risen, across racial/ethnic groups (Remington and Brownson 2011). Like smoking, obesity is both dangerous and modifiable, but since the start of the 2000s, the rate of obesity has been rising so precipitously that obesity may soon outstrip smoking when it comes to culpability for mortality (Mokdad et al. 2004), unless – as knowledge of obesity’s role in heart and other ailments continues spreading, gradually finding reflection in Americans’ beliefs and attitudes – rates of obesity taper off (Hubert et al. 1983; Danaei et al. 2009). The fundamental cause perspective argues that racial disparities in obesity could first increase, declining only as related health information slowly, with time, extends to low-SES Americans.

Public-health research often finds lifestyle and diet to be the proximal risk factors in chronic disease and health (Knoops et al. 2004; Van Dam et al. 2008), neglecting to look to social conditions as health’s fundamental cause (Link and Phelan 1995). In contrast, the present study was interested in social conditions as health’s ultimate source. Obesity, which, on one level, is indeed a health-risk behavior and lifestyle indicator, was our outcome variable. We viewed likelihood of being obese in contexts of social structure and time, exploring the role of race/ethnicity in obesity rates of American adults across 32 years (1981–2012). We set out to test the following three hypotheses: (1) that, among adults of various ages, the health risk represented by likelihood of obesity is affected by socioeconomic factors including education, employment, and poverty and wealth (Pavlova and Silbereisen 2012); (2) that race/ethnicity moderates associations between obesity and the social structural factors measured to test Hypothesis 1, which leads to further obesity-related disadvantage for respondents of minority race/ethnicity (Charles and DeCicca 2008); and (3) that over time, racial disparities in obesity likelihood will gradually decrease (Link and Phelan 1995). The model that we tested is illustrated by Figure 1 (Theoretical Model Linking SES and Other Social Status Factors with Obesity).

The literature contains numerous examinations of the role in obesity that other social status factors (gender, age, marital status, etc.) may play. However, these studies’ results have been inconsistent for minority groups (Cutler, Glaeser, and Shapiro 2003; National Center for Health Statistics 2016), as when elevated likelihood of obesity was observed for
Black females, but not Black males (National Center for Health Statistics 2016). We examined gender differences within racial groups closely, since such differences can signal intersectional differences relating to both race and gender that analyses of race alone may obscure (Bauer 2014).

Methods

Our study drew on data derived from the National Longitudinal Survey of Youth (NLSY). The Ohio State University started NLSY in 1979 with funding from the U. S. Department of Labor. The university conducted the survey annually until 1994 and then collected data biennially through 2012. NLSY sought to capture individuals’ life experiences over time: family, schooling, job experiences, crime, and substance abuse. Its national sample comprised civilian, non-institutionalized people born between 1957 and 1964, who were interviewed in person and by telephone. Some interviews concurrently employed computer-assisted formats or paper-and-pencil surveys. In 1979, 12,686 persons ages 14–22 were interviewed. In order to capture our outcome variable, obesity, we focused on NLSY years that included data collection concerning respondents’ body weight, 18 years in all, ranging 1981–2012. For our analyses, we linked each respondent’s data in a longitudinal record based on exactly 18 interviews.

Applying the discrete-time method to analyze this data (Singer and Willett 2003), we divided each longitudinal record into person-waves, our study’s units of analysis. A total of 118,749 person-waves – our sample – were constructed, with a subsample numbering 65,702 representing White respondents’ data; another subsample numbering 31,618 representing Black respondents’ data; and a final subsample numbering 21,429 representing Hispanic respondents’ data. Using person-waves, we evaluated associations between our explanatory variables and likelihood of obesity, the outcome.
**Measures**

For our longitudinal study, we needed to employ likelihood of being obese as a time-varying variable. The 18 survey years in which NLSY had collected data on respondent body weight were 1981, 1982, 1985, 1986, 1988, 1990, 1992, 1993, 1994, 1996, 1998, 2000, 2002, 2004, 2006, 2008, 2010, and 2012. Data from only these years made up the longitudinal records for respondents in our final sample. Respondent height was not reported in each of the 18 years, but because respondents were adults, we felt it was reasonable to assume height data would be roughly stable. We let height reported in 1982 give our height data for 1981 and 1982. Similarly, we let height reported in 1985 give our height data for any year between 1985 and 2004 (inclusive). Height reported in 2006, 2008, 2010, and 2012 gave our height data for those years. To clean up the self-reported height and weight data, we first pinpointed extreme values: height above 7 feet, weight below 70 pounds or above 600 pounds. Next, we considered extreme values in a context of respondent height/weight history, correcting values that appeared to be erroneous based on values recorded for the previous survey year and subsequent survey year. An example is the pair of respondents indicating weight of 50 pounds, when recorded weight had been 150 pounds in the two nearest survey years; we corrected their reports of 50 pounds to 150 pounds. Next, we calculated BMI as weight (lb) / [height (in)] 2 * 703, taking BMI of 30 or more to indicate presence of obesity. Obesity was a dichotomous variable in our study, with 0 indicating the reference category.

We measured our six explanatory factors – poverty, highest grade completed, weeks out of the labor force, weeks of unemployment, family assets, marital status – as variables that varied across the 18 years which figured in our outcome. We measured poverty dichotomously, 1 indicating presence of poverty (i.e. in preceding calendar year, total net family income below Poverty Income Guidelines for family of given size), and 0 indicating absence of poverty. We measured highest grade completed continuously, the variable stating the number of years of formal schooling completed, with offered responses of 0 (no grade completed), 1 (1st grade completed) … 12 (12th grade completed), 13 (1st year of college completed), 14 (2nd year of college completed) … 20 (8th year of college completed or more). In 2012, respondents were not asked about highest grade completed, so we let values reported for the variable in 2010 indicate the 2012 values. Another continuous variable, for respondents who reported not belonging to the labor force in the preceding year, was weeks out of the labor force, ranging from 0 to 52 weeks. Weeks unemployed, also continuous, captured the number of weeks (0–52) in which respondents who noted being unemployed at some point in the preceding year had reportedly been unemployed. An additional continuous variable was family assets, measured in $100,000 increments. It stated the total family assets self-reported by each respondent, minus the total self-reported amount of family debt (NLSY researchers top-coded 2% of all values for family assets). Finally, we measured marital status dichotomously, 1 indicating married, 0 not married.

We measured as time-invariant the following three explanatory factors: age in 1979, gender, immigrant status. Respondents at NLSY’s initial interviews in 1979 ranged in age from 14 to 22; age was a continuous variable. We measured gender dichotomously, 1 indicating male, 0 female. We measured immigrant status dichotomously as well, 1 indicating respondent born outside U.S., 0 as reference. Our three dichotomous variables of
race/ethnicity reflected self-reports of respondents’ Hispanic, non-Hispanic White, or non-Hispanic Black status. Finally, we created and employed 17 dichotomous time variables, so that we could take the time factor into account while capturing 18 waves occurring over 32 years; 0 indicated 1981, the reference category, while 1 indicated any of the remaining 17 years (which fell between 1982 and 2012, inclusive).

**Data analysis**

To take into account the repeated measurements belonging to our longitudinal study, we used generalized estimating equations (GEE). The approach enabled us to use both time-varying and time-invariant variables to explain obesity’s likelihood, while estimating autocorrelations and autoregressive correlations in STATA (Hilbe, Hardin, and Hardin 2003). We conducted a multivariate analysis for each of the three racial/ethnic groups (Hispanic, non-Hispanic White, non-Hispanic Black), exploring the potential associations, by race/ethnicity, between these explanatory variables and obesity.

Procedures we followed to examine race/ethnicity’s possible moderating role in associations between obesity and the explanatory variables were the following. First, we created two groups of interaction terms. One group included interactions between non-Hispanic Black and each of the explanatory variables, including the 17 time factors; the other included interactions between Hispanic and these same variables and time factors. Second, we ran a series of multivariate analyses that employed the entire sample and included all explanatory variables, all time factors, the dummy variables Black and Hispanic, as well as the interaction terms between each of the two minority groups and each explanatory or time variable. Third, we tested for statistical significance, in order to determine whether the relationship between a given explanatory or time variable and obesity’s likelihood differed for White respondents versus minority respondents from (separately) one minority group or the other, with all other explanatory and time variables controlled. Wherever we observed a statistically significant moderating effect wielded by an explanatory or time variable, we noted the strength of obesity’s association with the minority subsample relative to the White subsample, ascertaining the magnitude of the variable’s significant moderating effect.

**Results**

Included in our multivariate data analyses were 10,754 respondents (6078 non-Hispanic White, 2816 non-Hispanic Black, and 1860 Hispanic). About half of respondents were male (49% of White respondents were male, as were 51% of Black and 51% of Hispanic respondents). About one-quarter of Hispanic respondents (25.6%) had been born outside the U.S., compared to 2.6% of Black and 2.1% of White respondents. For the sample as a whole, respondent age in 1979 averaged roughly 18 years.

Table 1 shows descriptive statistics for the time-varying outcome and explanatory variables, by racial/ethnic group and for the sample as a whole. Using ANOVA and Chi-square testing, we identified significant differences among the racial/ethnic groups for each variable except the 2002 time factor. Across three decades, about 17% of White respondents, 29% of Black respondents, and 25% of Hispanic respondents qualified as obese (that is, had a BMI of 30 or more). A significantly higher percentage of Blacks
exhibited poverty (28%) than Hispanics (19%) or Whites (10%). On average across the decades, Whites had completed 13.0 years of education, Blacks 12.7 years, and Hispanics 12.0 years. Whites averaged the fewest weeks – nine – reportedly out of the labor force and the fewest weeks – two – reportedly unemployed. Hispanics averaged the next lowest, 12 weeks for the labor force measure, 2 weeks for the unemployed measure. Blacks averaged 13 weeks for the labor force measure, 4 weeks for the unemployed measure. On average, Whites reported net family assets worth $138,000, versus Blacks’ average reported assets of $40,000 and Hispanics’ average reported assets of $78,000. On average for the 18 years, above half of White (59%) and Hispanic (53%) respondents reported they were married; roughly a third (32%) of Blacks reported they were married.

Table 2 sets out our GEE model, which explains obesity’s likelihood for each racial/ethnic group. For White respondents, such likelihood was found to be significantly higher for married respondents, less educated respondents, respondents less consistently in the labor force (in the past year), and respondents relatively young in 1979. For Black respondents, obesity’s likelihood was found to be higher for married respondents, less educated respondents, respondents relatively young in 1979, and female respondents. For Hispanic respondents, obesity’s likelihood was higher for married respondents, less educated respondents, respondents more consistently employed, respondents relatively young in 1979, and respondents born in the U.S. For all three racial/ethnic groups studied, all of the time factors except 1982 proved significantly and positively related to obesity. This means that all respondents were more likely to qualify as obese between 1985 and 2012 than in 1981.
<table>
<thead>
<tr>
<th></th>
<th>White</th>
<th>Black</th>
<th>Hispanic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR 95%CI</td>
<td>OR 95%CI</td>
<td>OR 95%CI</td>
</tr>
<tr>
<td>Poverty</td>
<td>0.967 0.915</td>
<td>0.961 0.915</td>
<td>1.034 0.968</td>
</tr>
<tr>
<td>Highest Grade Completed</td>
<td><strong>0.890</strong> 0.875</td>
<td><strong>0.931</strong> 0.910</td>
<td><strong>0.936</strong> 0.913</td>
</tr>
<tr>
<td>Wks Out of Labor Force</td>
<td>1.001* 1.000</td>
<td>1.001 0.999</td>
<td>1.000 0.998</td>
</tr>
<tr>
<td>Wks of Unemployment</td>
<td>1.001 0.999</td>
<td>1.001 0.999</td>
<td>0.997* 0.995</td>
</tr>
<tr>
<td>Family Asset (in $100,000)</td>
<td>0.994* 0.989</td>
<td>0.996 0.984</td>
<td>0.994 0.982</td>
</tr>
<tr>
<td>Married</td>
<td>1.214** 1.160</td>
<td>1.224** 1.157</td>
<td>1.190** 1.112</td>
</tr>
<tr>
<td>Age in 1979</td>
<td><strong>0.912</strong> 0.901</td>
<td><strong>0.920</strong> 0.904</td>
<td><strong>0.898</strong> 0.881</td>
</tr>
<tr>
<td>Male</td>
<td><strong>1.019</strong> 0.916</td>
<td><strong>0.485</strong> 0.425</td>
<td><strong>1.053</strong> 0.892</td>
</tr>
<tr>
<td>Immigrant Status</td>
<td>0.737 0.504</td>
<td>0.725 0.441</td>
<td>0.798* 0.654</td>
</tr>
<tr>
<td>1982</td>
<td>1.014 0.925</td>
<td>0.970 0.846</td>
<td>0.951 0.768</td>
</tr>
<tr>
<td>1985</td>
<td><strong>1.539</strong> 1.367</td>
<td><strong>1.578</strong> 1.345</td>
<td>1.388** 1.117</td>
</tr>
<tr>
<td>1986</td>
<td><strong>1.778</strong> 1.581</td>
<td><strong>1.980</strong> 1.668</td>
<td>1.734** 1.404</td>
</tr>
<tr>
<td>1988</td>
<td>2.199** 1.938</td>
<td>2.696** 2.272</td>
<td>2.364** 1.878</td>
</tr>
<tr>
<td>1990</td>
<td>2.778** 2.445</td>
<td>3.387** 2.828</td>
<td>3.028** 2.419</td>
</tr>
<tr>
<td>1993</td>
<td>3.577** 3.127</td>
<td>5.021** 4.210</td>
<td>4.028** 3.201</td>
</tr>
<tr>
<td>2000</td>
<td><strong>6.378</strong> 5.558</td>
<td><strong>9.097</strong> 7.574</td>
<td>6.806** 5.399</td>
</tr>
<tr>
<td>2002</td>
<td>7.024** 6.120</td>
<td>9.030** 7.491</td>
<td>6.942** 5.486</td>
</tr>
<tr>
<td>2004</td>
<td>7.279** 6.327</td>
<td>9.777** 8.139</td>
<td>7.192** 5.691</td>
</tr>
<tr>
<td>Wald Chi-Square</td>
<td>3811**</td>
<td>1795**</td>
<td>1165**</td>
</tr>
<tr>
<td># of Person-Waves</td>
<td>65,702</td>
<td>31,618</td>
<td>21,429</td>
</tr>
</tbody>
</table>

Note: Bold-faced, underlined figures signify significant interaction effects involving race/ethnicity and the independent variable, Whites serve as the reference.

*p < .05; **p < .01.
The procedures outlined at ‘Data Analysis’ above allowed for examining race/ethnicity’s moderating role in associations between obesity and all explanatory and time factors. Compared with the reference group of White respondents, we found Hispanics to benefit less from additional education, in terms of reduced risk of obesity. The interaction involving Hispanic race/ethnicity and education (OR = .97, \( p < .05 \)) indicated that each completed year of schooling reduced obesity’s likelihood by 11% for Whites, by just 6% for Hispanics. Respondent age in 1979 showed a negative relationship, in this study, to obesity risk; the negative link was strongest for Hispanics (OR = 1.18, \( p < .01 \)) and weakest for Blacks (OR = 1.23, \( p < .01 \)). Each one-year increase in the age measure was associated with, for Whites, a 9% decrease in obesity likelihood; for Blacks, an 8% decrease in obesity likelihood; and for Hispanics, a 10% decrease in obesity likelihood.

We found gender to have a statistical link to obesity among Blacks, in that female Blacks were 51% more likely than male Blacks to be obese. We observed no statistical link between gender and obesity for Whites or Hispanics. Additionally, we found no significant White–Hispanic differences in obesity’s associations with time factors but did observe five significant White–Black differences, as follows: In 1985 (OR = .90, \( p < .05 \)), 1992 (OR = 1.10, \( p < .05 \)), 2000 (OR = 1.09, \( p < .01 \)), 2008 (OR = .92, \( p < .05 \)), and 2012 (OR = .92, \( p < .05 \)) – but not in 1981 – Blacks’ likelihood of obesity increased significantly more than Whites’ did. Specifically, for interview waves dated 1985, 1992, 2000, 2008, and 2012, White respondents’ obesity likelihood saw average increases (over level measured at 1981 wave) of, on average, 54%, 230%, 538%, 822%, and 973%, respectively. Black respondents’ obesity likelihood also rose, by, on average, 58%, 385%, 810%, 935%, and 1084%, respectively.

To sum up, we used 18 waves of NLSY data (dating 1981–2012) to evaluate associations between obesity likelihood and certain socioeconomic and other status factors. Separate evaluations were made for our subsamples of Whites, Blacks, and Hispanics. As well, we also examined race/ethnicity’s potential moderating role in the said associations. The results confirmed socioeconomic factors’ power to foster obesity among respondents in these three racial/ethnic groups. From group to group, however, our socioeconomic factors were associated differentially with likelihood of obesity. By linking each wave of NLSY data to a time factor, we were able to estimate White–Black and White–Hispanic differences in obesity’s likelihood across 32 years. Over that period, the White–Hispanic differences stabilized, while the White–Black differences grew: For five survey years (interview waves), we observed an increase in obesity’s likelihood (versus 1981 measures) that was significantly larger for Blacks than Whites.

**Discussion**

Results of our study suggest three interpretations and implications that particularly deserve mention, as follows. The first is that resource and structural factors matter in the explanation of obesity – a notorious condition creating chronic disease and long a leading cause of death in this country. For example, the results for education’s role were consistent with the literature documenting links among education, lifestyle factors, and use of disease-related treatment (Cutler, Glaeser, and Shapiro 2003; Phelan, Link, and Tehranifar 2010). Through education, individuals access knowledge and resources, becoming situated to seek the health information necessary for better health (Link and
Our study clearly showed the less educated in all three of our racial/ethnic groups to have relatively higher likelihoods of obesity. Many American public-health experts view lifestyle and diet as key determinants and view obesity patients to be responsible for their dietary and lifestyle choices – and thus for our epidemic of obesity. Some even advocate using health insurance premiums to punish the obese. Mounting evidence shows, however, that ‘choosing’ to exercise or overeat may actually be the consequence of attitudes and beliefs born of the legacy of SES and material/social deprivation in certain communities (Henderson et al. 2014). Our results point to education, an important indicator of SES, as a resource for reducing obesity in each of the three racial/ethnic groups our three subsamples comprised. Since, however, we observed obesity’s association with education to be stronger among Whites than Hispanics, White-Hispanic disparities in obesity could actually be aggravated by improving education for all racial/ethnic groups. Future research should elaborate more fully how and why Hispanics, but not Whites, experience a diminishing return on educational investment where obesity likelihood is concerned.

One structural factor we employed, family assets, helped indicate social structure and among Whites was found to contribute significantly to reduced obesity likelihood; no such contribution was observed concerning Black and Hispanic respondents. As the multiple disadvantage model suggests, different social factors may serve to explain the various health-risk behaviors of different racial/ethnic groups (Lo, Howell, and Cheng 2013). We included family assets in our study, a factor rarely mentioned in obesity research. It added a further, unusual structural consideration useful in understanding health and disease (Williams and Collins 1995). Family assets are linked to obesity likelihood, and such assets are often linked to generational financial inheritance; thus obesity (and other health risks) are comparatively unlikely among the comparatively wealthy (LaVeist 2005). For many years now researchers have attributed Whites’ better health to their comparative wealth (Williams and Collins 1995). In our study too, Whites with more family wealth were less likely to be obese. But this particular wealth–health relationship was not observed for Blacks and Hispanics in our study, possibly due to the fairly homogeneous value of our ethnic-minority respondents’ family assets.

Employment status was associated with obesity among Whites and Hispanics in our study, but not Blacks. The results showed that only among Whites was obesity fostered by time out of the labor force; and only among Hispanics was obesity diminished by time unemployed. It is documented by previous research that employment has strong associations with decision-making power and prestige. This is because economic success surpasses in significance achievements won in other domains, such as family relationships (Rosenfield 1989; Messner and Rosenfeld 2001). Withdrawal from the labor force often is voluntary and lacks the dire implications pertaining to material losses. In contrast, not being employed can signal real deprivation and stress presenting subsequent health problems including obesity (Cutler, Glaeser, and Shapiro 2003). Many Hispanics are day laborers (Valenzuela Jr 2003), so our study’s results pertaining to working status may reflect, to some extent, the differential occupational prestige of jobs typically held by members of given racial/ethnic groups (Xu and Leffler 1992). Future research might add occupational status to employed measures of SES.
be scrutinized in order to cut obesity rates. Worse rates of obesity among ethnic-minority Americans raise their risk of chronic disease and early mortality. Consequently, Black Americans have significantly shorter life expectancy than White (National Center for Health Statistics 2012). While Hispanic Americans have both higher rates of obesity and longer life expectancy than Whites (National Center for Health Statistics 2012), it remains true that reducing Hispanics’ obesity rates might improve their health further. Racial disparities in obesity and health may be attributable to social resources and to harm done to minority groups by unequal distribution of returns on human-capital investment. The results from the tested interactions involving race/ethnicity and education, gender, and age illustrate additional obesity-related disadvantages associated with minority race/ethnicity. For instance, education brings Whites an advantage (a lower likelihood of obesity) that it fails to bring equal amount of advantage to Hispanics. As well, additional disadvantage plagues Black females (who have a significantly higher obesity risk than Black males have), yet doesn’t affect White or Hispanic females. This indicates a need to analyze gendered differences between racial/ethnic categories with an eye to their intersectionality (Hankivsky 2012; Etherington 2015). Intersectional effects are best understood as multiplicative, not additive. Still, our findings may suggest that intersectionality has a role in Black women’s substantively worse outcomes: These women are, after all, structurally disadvantaged by race as well as gender (compared, that is, to Black men or White women) (Etherington 2015). Race was the variable of primary interest to us, but our study findings do suggest that additional research is warranted on differential experiences of women of minority race/ethnicity. Furthermore, an advantage for older Blacks (lower obesity likelihood relative to younger Blacks) pales in comparison to the advantage we observed among older Whites and older Hispanics. Over-emphasizing lifestyle and diet has left public-health professionals and government officials with an unsolved problem, and their intervention measures have failed to undo the harm of our nation’s socioeconomic and social status legacies (Link and Phelan 1995; Phelan et al. 2004).

Third, future research should ask if (and why) White–Hispanic disparities in obesity have stabilized at the very time White–Black disparities have increased. Our good fortune in having longitudinal data spanning 32 years meant we had a lengthy trend of racial disparities to examine, showing any extant widening or narrowing of the obesity gap in that period. The handful of years in which this gap widened between Whites and Blacks might reflect national/international events and evolutions: the 2007–2011 recession, for instance, or failed awareness campaigns targeting obesity-related disease and mortality. Failure to close the Black–White gap is evidence of serious disadvantage associated with Blacks in this country, and of a need to attend to socioeconomic factors like education and family assets (Kahng 2010). Putting college admission within reach of ethnic-minority Americans is an example of an important policy supporting reduction of obesity in the U.S.; policies responsible for gains in this area should be continued and reinforced (Phelan, Link, and Tehranifar 2010).

Our study, although it produced significant results in the field of racial disparities in obesity, had at least two limitations. We intended to estimate and understand racial disparities in obesity influenced by SES and other social status factors, so we used obesity as our outcome variable. The literature, however, outlines obesity’s potential role in income reduction, a means through which obesity becomes the determinant of SES, rather than
the reverse (Mason 2012). Future research could examine SES and obesity in a temporal ordering model, untangling the income–obesity relationship.

Using secondary data limited how we could include variables generated from self-report data and precluded the inclusion of any medical-care variables in our model, despite their likely relevance to obesity. All of the NLSY data we employed was collected in interviews, meaning we had to construct all variables (height, weight, and family assets included) in our model from self-report data, which, of course, is vulnerable to memory or accuracy problems. The NLSY data being, moreover, short on measures of discrimination, we could not directly test whether daily, institutionalized discrimination might explain some part of racial disparities in obesity (Pearlin et al. 2005). Additionally, NLSY did not develop some important contextual information about respondents’ immediate surroundings, including the communities where they lived. Such information probably would have been useful, since segregated communities more often house Blacks than Whites, and residential segregation is a marker for resource deprivation of the type that makes it difficult to fight obesity and chronic disease (Williams and Jackson 2005). Further research might pin down contextual factors like these.

Despite the limitations on our study, it did clearly contribute to the literature by locating racial disparities in obesity in the context of time. It also confirmed that certain socio-economic and social status factors importantly affect racial disparities in obesity. Finally, our study confirmed recent reports that some racial disparities in obesity have shown signs of stabilizing.

Disclosure statement

No potential conflict of interest was reported by the authors.

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