Would the elimination of obesity and smoking reduce U.S. racial/ethnic/nativity disparities in total and healthy life expectancy?☆

Michelle L. Friscoa,*, Jennifer Van Hooka, Robert A. Hummerb

a Penn State University, United States
b University of North Carolina, Chapel Hill, United States

ARTICLE INFO

Keywords:
- Epidemiological paradox
- Obesity
- Smoking
- Racial disparities
- Mortality
- Healthy life expectancy

ABSTRACT

Obesity and smoking are the two leading causes of preventable death and disability in the United States. Both of these health risks are socially patterned in ways that likely produce racial/ethnic/nativity disparities in total and healthy life expectancy. The current study simulates the extent to which the hypothetical elimination of smoking and obesity would change disparities in longevity and disability by analyzing data from 19,574 U.S.-born white, black, Hispanic and foreign-born Hispanic men and women in the 1999–2000 through 2009–2010 National Health and Nutrition Examination Survey and linked mortality files. Results suggest that the elimination of both obesity and smoking would significantly narrow disparities in total and healthy life expectancy between black and white adults and remaining differences are statistically non-significant. The longstanding life expectancy advantage of Hispanic immigrants over whites is reduced, but remains large. The life expectancy advantage of U.S.-born Hispanics is reduced as well, though to a smaller extent than what is observed for Hispanic immigrants. There were no significant observed healthy life expectancy differences between white and U.S.-born Hispanic adults. Overall study results suggest that the elimination of obesity and smoking would change the shape of racial/ethnic/nativity disparities in ways that would result in greater health equity.

1. Introduction

When the total and healthy life expectancies of white, black and Hispanic U.S. adults are compared, a portrait of racial/ethnic health inequity emerges. Compared to white peers, Black men and women enjoy fewer years of life expectancy (Hayward, Hummer, & Chiu, 2014). Hispanics, conversely, live longer than U.S.-born whites despite their ethnic minority and lower socioeconomic status (Lariscy, Hummer, & Hayward, 2015), a well-documented “epidemiological paradox” that is more pronounced among immigrants (Markides & Eschbach 2011). Unfortunately, Hispanic men and women ages 50 and older also have fewer years of healthy life expectancy than U.S.-born whites (Hayward et al., 2014).

We estimate the extent to which these disparities in total and healthy life expectancy would change under three counterfactual conditions—that study respondents were never obese, never smoked, and were never obese and never smoked. These three scenarios are unlikely to be realized anytime soon, but our simulations are important estimates of the contributions that obesity and smoking make to racial, ethnic and nativity disparities in longevity and disability.

We focus on obesity and smoking because they are the two leading causes of U.S. preventable deaths and chronic health conditions. Hispanic immigrants are less likely than whites to smoke (Fenelon 2013; Lariscy et al., 2015) and do not differ markedly from whites in their obesity risk (Bates, Acevedo-Garcia, Alegría, & Krieger, 2008), but structural conditions lead U.S.-born black and Hispanic adults to be at greater risk of becoming obese (Ogden, Carroll, Kit, & Flegal, 2014) and long-term smokers (Trinidad, Pérez-Stable, White, Emery, & Messer, 2011) than white peers. Thus, although racism, socioeconomic disparities, and other health problems certainly contribute to disparities in the longevity and disability of white, black and Hispanic adults, we expect to find that plausible reductions in these health inequities could be yielded if obesity and smoking were eliminated.

We analyze nationally representative data from U.S.-born white, black and Hispanic adults and foreign-born Hispanic adults who participated in the 1999–2000 through 2009–2010 National Health and Nutrition Examination Survey (NHANES). An important study contribution is that we methodologically build on and improve the work of...
others who have assessed how changing patterns and trends in smoking and obesity are associated with current and future U.S. life expectancy (e.g., Fenelon 2013; Lariscy et al. 2015; Olshansky et al. 2005; Preston, Stokes, Mehta, & Cao, 2014; Stewart, Cutler, & Rosen, 2009). Like other researchers, we use survey data linked to follow-up mortality information to estimate age-specific mortality rates across various groups with different obesity and smoking experiences. However, these estimates suffer from measurement and sampling error because, when combined, they produce life expectancy estimates that are modestly higher than National Center for Health Statistics (NCHS) estimates (Arias, Heron, & Xu, 2017). To correct this, we calibrate the survey-based mortality estimates so that overall age-specific mortality rates are consistent with NCHS estimates by sex and race/ethnicity, following the example of others (Handcock, Huovilainen, & Rendall, 2000; Handcock, Rendall, & Cheadle, 2005).

2. Background

2.1. Obesity, smoking and total and healthy life expectancy

There is strong evidence that both current obesity and the length of time spent obese contract total life expectancy (e.g., Masters, Powers, & Link, 2013; Peto, Whitlock, & Cao, 2014; Preston, Vierboom, & Stokes, 2018) and contribute to later life disability (Ferraro and Kelley-Moore 2003). Preston and Stokes (2014) also offer compelling evidence that studies finding an “obesity paradox” (i.e., lower mortality among obese persons in some disease states) largely do so because of statistical biases. Given that permanent weight loss is uncommon among all adults regardless of race/ethnicity or nativity, disparities in current and previous accounts of obesity should be consequential for disparities in total and healthy life expectancy.

The negative relationship between cigarette smoking and life expectancy is unequivocal. More frequent smoking and more years spent as a smoker both reduce life expectancy and healthy life expectancy (Lariscy, Hummer, & Rogers, 2018; U.S. Department of Health and Human Services, 2014). Smokers who quit greatly reduce the impact that smoking has on early death and disease, and there are currently more former smokers in the U.S. than current smokers (U.S. Department of Health and Human Services, 2014) although there are racial, ethnic and nativity disparities in doctors’ advisement to quit, use of smoking-cessation aids (Cokkinides, Halpern, Barbeau, Ward, and Thun (2008)), and successful smoking cessation (Trinidad et al. 2011).

Given the strong evidence about the detrimental consequences of obesity and smoking, we can deduce that racial/ethnic and nativity groups with higher prevalence of smoking and obesity should benefit most from their elimination. If these gains are large enough both generally and in relation to gains made by groups with lower smoking and obesity prevalence, and disadvantaged groups also have fewer years of total and healthy life expectancy, then racial/ethnic and nativity disparities will shrink. However, if disadvantaged groups’ gains are quite small generally or relative to more advantaged groups, disparities in current and total healthy life expectancy may remain relatively static in our three counterfactual scenarios. Finally, if a group has a high total or healthy life expectancy despite being disadvantaged by a higher prevalence of obesity or smoking, eliminating these health behaviors could actually increase a group’s advantage.

Previous projections of the increasing prevalence of obesity and declining prevalence of smoking suggest that these trends work in counteracting ways to change life expectancy. These estimates also suggest that men would benefit more than women, with men projected to gain .83 years of life and women projected to gain .09 years of life (Preston et al. 2014). This is one reason why we discuss research expectations for women and men separately and treat them as separate groups in the analysis. In both our discussion and analysis of disparities, we use U.S.-born whites as the reference group because they are the largest group in the analysis.

2.2. Research expectations for women given known disparities in obesity and smoking

Estimates suggest that 39% of white women, 48% of Hispanic women, and 56% of black women are obese (Hales, Fryar, & Carroll, 2018). Among Hispanic women, immigrants are leaner than U.S.-born peers (Antecol & Bedard 2006). Similar disparities have been evident in early and middle adulthood since the turn of the century (Flegel, Carroll, Ogden, & Curtin, 2010), suggesting that these disparities have been persistent.

Given that white women have higher total and healthy life expectancies than black women, these advantages should erode when we simulate the elimination of obesity if the simulation produces substantial enough gains for black women relative to the gains made by white women. Conversely, because U.S.-born and foreign-born Hispanic women have higher life expectancies and lower healthy life expectancies than whites, their life expectancy advantage may grow and their healthy life expectancy disadvantage may shrink if obesity were eliminated. Once again, though, this expectation is predicated on the size of the gains white and Hispanic women make under simulated conditions of no obesity.

Racial, ethnic and nativity disparities in smoking are different than those observed for obesity. Smoking is most prevalent among white women (15.5%), followed closely by black women (13.5%). Only 7% of Hispanic women smoke (Jamal et al. 2018), and Hispanic immigrants have an even lower smoking prevalence (Bostean, Ro, & Fleischer, 2017). It is also important to note, though, that U.S.-born black and Hispanic women are less likely to be former smokers than current smokers whereas white women are more likely to be former smokers versus current smokers (Trinidad et. al. 2011; Babb, Malaracher, Schauer, Asman, & Jamal, 2017).

Smoking makes a substantial contribution to Hispanic women’s life expectancy advantage over white women, with recent estimates suggesting that 50–60% of the Hispanic immigrant advantage in life expectancy vis-à-vis non-Hispanic whites is due to smoking (Fenelon 2013; Lariscy et al. 2015; McDonald & Paulozzi 2018). This means that the simulated elimination of smoking should lead this advantage to shrink if the gains made by white women outpace the gains made by Hispanic women. This may be especially evident when comparing white and Hispanic immigrant women (Fenelon 2013; Lariscy et al. 2015), given the low prevalence of both current and former smoking among the latter group. Hispanic women ages 50 and older spend more years disabled than white women (Hayward et al. 2014). Thus, if smoking were eliminated, Hispanic women’s disadvantage may grow.

The similar smoking prevalence of black and white women suggests that disparities between these groups may be relatively sticky when we simulate the elimination of smoking. Nonetheless, it is also possible that the simulation could narrow white women’s total and healthy life expectancy advantages because black women are less likely to quit smoking (Babb et al. 2017) and more likely to die from smoking-related illnesses (Ho & Elo 2013).

2.3. Research expectations for men given known disparities in obesity and smoking

Racial/ethnic disparities in obesity among men are not as stark as those observed for women. Currently, 37% of white and black men and 41% of Hispanic men are obese (Hales et al. 2018), and immigrant Hispanic men are leaner than U.S.-born Hispanic men (Albrecht, Rouz, Aiello, Schulz, & Abarro-Lanza, 2013). Thus, we do not expect the simulated elimination of obesity to substantially change observed racial, ethnic and nativity disparities in total and healthy life expectancy.

Racial, ethnic and nativity disparities in smoking among men mostly mirror those observed for women (Bostean et al. 2017; Jamal et al. 2018). Thus, when we simulate the elimination of smoking, our research expectations for men are mostly similar to the expectations.
above for women. There is one exception. Among men, the prevalence of smoking among foreign-born Hispanics and whites is similar (Fenelon 2013), a finding that has become even more evident as the heaviest smoking cohorts of white males have begun to die (Preston & Wang 2006). Thus, we expect that the simulated elimination of smoking may result in only modest changes in the total and healthy life expectancy disparities between white and foreign-born Hispanic men.

3. Materials and methods

3.1. Data and measures for life expectancy and healthy life expectancy

We produce estimates of total and healthy life expectancy at age 35 for women and men using the 1999–2000 through 2009–2010 NHANES and linked mortality files. We use 35 as the age floor because this is when smoking attributable mortality begins (U.S. Department of Health and Human Services, 2014). We pool data across multiple survey years and multiply impute all missing data using the STATA 14.0 “MI” procedure. This procedure iteratively replaces missing values on all variables with predictions based on random draws from the posterior distributions of parameters observed in the sample, creating multiple complete datasets. We averaged empirical results across five imputation samples and accounted for random variation across samples to calculate standard errors using Rubin’s (1987) rules. Our final sample includes 19,573 adults (3488 foreign-born Hispanics, 1959 U.S.-born Hispanics, 3828 blacks, and 10,298 non-Hispanic whites).

We also use NCHS 2012 life tables for Hispanic, white, and black adults by sex (Arias 2016) to calibrate estimates of the age-specific conditional probability of dying in our life tables (the $q_{x}$s). This is explained further below.

Life expectancy estimates are based in part on Cox proportional hazard models predicting the hazard of death as a function of body weight, smoking, education (less than high school, high school, some post-secondary education, and college graduate), age at interview (top-coded at 85), and survey year. The hazard function is stratified by race/ethnicity/nativity (U.S.-born, non-Hispanic white, black or Hispanic or foreign-born Hispanic) and the models are estimated separately by sex (available upon request). The dependent variable is whether (and if so, when) the person died during the follow-up period after the NHANES interview but before January 1, 2012, and age at death among decedents. The linked death certificates data provide vital status and age at death, measured to the nearest quarter-year. We omitted the first 2 years of mortality follow-up to reduce the effects of reverse-causality whereby sick people lose weight shortly before dying.

We tested several alternative model specifications and results were consistent. For example, we tested interactions between weight and age, and between weight and smoking status and race/ethnicity, but they were not significant, leading us to estimate simpler models that assume that the relative hazard of dying for each weight and smoking category is constant across all age groups and is the same across race/ethnic/nativity groups. This is somewhat surprising given that Hispanic immigrants who smoke tend to smoke significantly fewer cigarettes than their white counterparts, and we acknowledge that the lack of significant interactions could be due to a lack of statistical power.

Healthy life expectancy is based in part on logistic regression models that predict disability as a function of body weight, smoking, race/ethnicity/nativity, education, age at interview, and survey year. The models are estimated separately by sex (available upon request). Disability is defined as the inability to perform one or more activities of daily living (ADLs), including dressing, eating, or drinking, and getting in or out of bed.

One key independent variable is body weight. It distinguishes between those who were never obese (not obese at interview and not obese at age 25), formerly obese (obese at age 25 but not at interview), currently obese (obese at interview but not at age 25), and consistently obese (obese at interview and at age 25). These categories are based on body mass index constructed from measured height and weight ($kg/m^2$) during the NHANES examination and self-reported weight at age 25. It would be ideal to have (at least) two measured assessments of weight rather than only one. However, using both indicators better taps the cumulative consequences of obesity and avoids positive associations between current obesity and mortality that emerge using cross-sectional data (Preston & Stokes 2014). This is a primary reason we analyze NHANES data rather than National Health Interview Survey (NHIS) data.

Our second key independent variable is smoking. It indicates whether respondents never smoked, are former smokers, or are current smokers.

3.2. How total and healthy life expectancy are related to weight and smoking

We now show how life expectancy is mathematically related to a group’s weight status and smoking distribution. We build on these insights to develop a method for estimating how much life expectancy would change if a group’s weight status or smoking distribution was different.

Life expectancy is an estimate of average length of life for a synthetic cohort that experiences the complete set of conditional probabilities of dying ($q_x$) across the lifespan. If life expectancy is related to obesity and smoking, it is because the probabilities of dying ($q_x$) are higher among people who are or were obese relative to people who were never obese, and higher among those who smoke or used to smoke than those who never smoked. Taking obesity as an example, each of the $q_x$ is expressed as the sum of weight-specific $q_x$ for those who were never obese ($w_1$), currently obese ($w_2$), formerly obese ($w_3$), or have been consistently obese ($w_4$), weighted by the proportions in each of the four weight categories ($p_{x,w_1}, p_{x,w_2}, p_{x,w_3}, p_{x,w_4}$) and $p_{x,w}$:

$$q_x = q_{x,w_1}p_{x,w_1} + q_{x,w_2}p_{x,w_2} + q_{x,w_3}p_{x,w_3} + q_{x,w_4}p_{x,w_4}$$

(1)

We specify a similar equation for smoking. These equations are central to how we estimate observed and counterfactual life expectancies. To estimate the $p_{x,w}$ terms, we use the NHANES data to calculate the age-specific proportions in each weight (or smoking) category for each group by sex. To estimate the $q_{x,w}$ terms, we again use NHANES data. Although the NCHS publishes estimates of $q_x$ by race/ethnicity and sex, they do not provide these estimates separately by weight or smoking status. Therefore, we first estimate proportional hazard Cox models predicting the hazard of dying as described in the previous section. We use the estimated hazard ratios ($H$) for the weight and smoking categories (expressed as ratios relative to the sample average) to obtain estimates of the weight- and smoking-specific $q_x$ terms. Specifically, we multiply the hazard ratios by the NCHS published $q_x$ ($q_{x,NCHS}$) separately by race/ethnic/nativity and sex. For example, the $p_{x,w}$ term for Hispanic women in Eq. (1) is calculated as:

$$p_{x,w_1,Hispanic\ female} = q_{x,NCHS,Hispanic\ female}p_{w_1,Hispanic\ female}$$

(2)

We repeat this for all race/ethnic/nativity groups and by gender, and we estimate the $q_x$ for current smokers, former smokers, and people who never smoked in the same manner. Multiplying the hazard ratios by the NCHS estimates ensures that weight- and smoking-specific estimates, when averaged across categories, match the NCHS estimates for all weight- and smoking categories combined.

After estimating values for Eqs. (1) and (2), we use standard demographic methods to estimate life expectancy from estimated $q_x$ (see, for example, Rowland 2003). We did this for each race/ethnic/nativity group by sex. As expected, the results match the 2012 life expectancies reported by NCHS.

To estimate counterfactual $q_x$’s and life expectancies, we changed the prevalence proportions, $p_x$, to reflect a scenario in which all persons in every group had never been obese and had never smoked. For example, we set the values of $p_{x,w_1}, p_{x,w_2}, p_{x,w_3}$, and $p_{x,w_4}$ to 1, 0, 0, and 0;
this produces counterfactual \( q_x \) and life expectancy values if no one had ever been obese.

We use a similar approach to estimate observed and counterfactual healthy life expectancies at age 35 for each race/ethnic/nativity group by sex. To simplify calculations, we relied on the Sullivan (1971) method. Healthy life expectancy is expressed as a function of the probability of dying \( (q_x) \) and the proportion disabled at each age \( (A_x) \). Briefly, the method involves (1) estimating a single-decrement life Table, (2) multiplying the person-years lived \((L_x)\) in each age interval by the proportion with at least one ADL \((A_x)\) to obtain person-years lived between age \( x \) and \( x+n \) in the ADLs \((L_x)\) and non-ADL states \((L_{x,nA})\), and (3) summing \(L_{x,nA}\) from age \( x \) to the oldest age and dividing by the number of survivors to age \( x \) \((L_x)\) to obtain the total healthy life expectancy at each age \( x \) (Jagger, Cox, & Le Roy, 2006).

The weight status and smoking distributions of a group are mathematically related to healthy life expectancy in two ways. They could affect the probabilities of dying, as just discussed. They could also affect the proportion of each group with an ADL \((A_x)\). For example, \( A_x \) can be expressed as the weighted average of ADL rates across the four weight status categories:

\[
A_x = A_{x,w1}p_{w1} + A_{x,w2}p_{w2} + A_{x,w3}p_{w3} + A_{x,w4}p_{w4} \tag{3}
\]

To estimate healthy life expectancies for each group, we use Eqs. (1) and (2) to calculate the \( q_x \) terms, as already described, and Eq. (3) to calculate the \( A_x \) terms. We combine the results, using the Sullivan method, to estimate healthy life expectancy. To obtain values for the \( A_{x,w} \) terms in Eq. (3), we first estimate logistic regression models predicting the probability of having an ADL \((A_x)\) as described in the previous section. Similar to how we estimate the weight- and smoking-specific \( q_x \) estimates, we use odds ratios from the models to estimate \( A_{x,w} \) values while ensuring that their weighted average across the weight and smoking status categories equals the observed age-specific ADL rate within each gender and race/ethnic/nativity group. To calculate counterfactual healthy life expectancy estimates, we repeat the calculations just described assuming that everyone had never been obese and never smoked.

Finally, we bootstrap estimates of standard errors of observed and counterfactual estimates of life expectancy and healthy life expectancy across 1000 iterations.

4. Results

4.1. Obesity and smoking

Table 1 shows racial/ethnic and nativity differences in obesity and smoking among women and men. A greater share of white women (64.0%) were never obese compared to black (43.0%) and Hispanic women (53.1% of U.S.-born and 58.5% of foreign-born). Former obesity is quite rare, representing the experience of only 1–2% of women in each group. Additionally, most currently obese women became obese over time. This includes 29.7% of white women, 46.1% of black women, 36.2% of U.S.-born Hispanic women, and 34.6% of foreign-born Hispanic women.

Like women, being formerly obese among men is quite rare. Unlike women, more foreign-born Hispanic men have never been obese, followed by whites, U.S.-born Hispanics, and blacks. The racial/ethnic/nativity gaps in the share who were never obese and who become obese are smaller among men than women.

Turning to smoking, U.S.-born white, black, and Hispanic women are similar. Slightly more than half never smoked, and 19.8% of white, 24.1% of black, and 22% of U.S.-born Hispanic women currently smoke. In contrast, nearly three-quarters of foreign-born Hispanic women never smoked and only 12.4% currently smoke. White women are more likely than black and Hispanic women to be former smokers.

Among men, the percentage who never smoked is similar across groups, thus illustrating that racial/ethnic and nativity patterns in smoking among men are more similar than among women. Further, compared to women, a smaller share of men in each group never smoked. Among white and foreign-born Hispanic men, roughly 22% report being current smokers while 37.2% and 28.1% of black and U.S.-born Hispanic men, respectively, are current smokers.

4.2. Women’s total and healthy life expectancy

Panel 1 of Table 2 shows women’s observed values of total and healthy life expectancy at age 35 and simulated gains under three counterfactual conditions: that no one was ever obese, no one smoked, and no one was ever obese or smoked. Panel 2 shows observed disparities in total and healthy life expectancy and simulated disparities under the three counterfactual conditions.

Looking first at actual total life expectancy at age 35, black women live an average of 45.1 years, 2.2 years less than white women. Conversely, Hispanic women on average live longer than white women, and foreign-born Hispanics have the largest advantage. Estimates of healthy life expectancy are shorter and suggest that on average all women will live over 15 years with some disability. Group differences in healthy life expectancy parallel results for life expectancy.

Estimated gains in women’s total and healthy life expectancy under simulated conditions where women were never obese and never smoked are shown next. Black women would gain 1.7 years of life if obesity were eliminated, but no other group would make statistically significant gains. Gains in healthy life expectancy would be even larger for all groups of women if obesity were eliminated, with white and
black women experiencing statistically significant gains of 2.3 and 3.7 years of health life, respectively. All groups of women would make large, significant gains in total and healthy life expectancy if smoking were eliminated, with one exception. The 1.6 year estimated gain in healthy life expectancy among foreign-born Hispanic women is not statistically significant, which is unsurprising given their relatively low level of smoking. Finally, if both obesity and smoking were eliminated, this would lead to large gains in women’s total and healthy life

| Panel 1: Observed values, observed disparities, and simulated changes in both if obesity and smoking were eliminated. |
|---------------------------------|---------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|                                | TLE                             | HLE              |                  |                  |                  |                  |
|                                | White                           | Black            | Hispanic USB     | Hispanic FB      | White           | Black           | Hispanic USB     | Hispanic FB      |
| Observed                        | 47.3 (0.0)                       | 45.1 (0.0)       | 49.1 (0.0)       | 51.1 (0.0)       | 32.3 (0.3)      | 28.9 (0.6)      | 33.6 (1.0)       | 35.2 (1.0)       |
| No one was ever obese           | 0.7 (0.4)                        | 1.7 (0.8)        | 0.6 (0.4)        | 0.9 (0.5)        | 2.3 (0.5)       | 3.7 (1.0)       | 2.5 (1.3)        | 2.4 (1.5)        |
| No one smoked                   | 2.6 (0.5)                        | 3.9 (0.7)        | 2.0 (0.4)        | 1.6 (0.4)        | 3.1 (0.6)       | 3.8 (1.0)       | 2.9 (1.4)        | 1.6 (1.5)        |
| No smoking or obesity           | 2.9 (0.6)                        | 4.7 (1.0)        | 2.4 (0.5)        | 2.0 (0.6)        | 5.1 (0.7)       | 7.1 (1.1)       | 5.1 (1.4)        | 3.8 (1.6)        |
| Simulated gains if              |                                |                  |                  |                  |                  |                  |
| No one was ever obese           | -2.2 (0.0)                       | 1.8 (0.0)        | 3.8 (0.0)        | n/a              | -3.4 (0.6)      | 1.2 (1.0)       | 2.8 (1.1)        |                  |
| No one smoked                   | -1.3 (0.9)                       | 1.7 (0.5)        | 4.0 (0.6)        | n/a              | 2.0 (0.9)       | 1.5 (1.0)       | 3.0 (1.2)        |                  |
| No smoking or obesity           | -0.9 (0.9)                       | 1.2 (0.6)        | 2.7 (0.6)        | n/a              | 2.7 (1.0)       | 1.0 (1.2)       | 1.3 (1.2)        |                  |
| Panel 2: Observed disparity     | n/a                             | 1.6 (0.8)        | 3.0 (0.9)        | n/a              | -1.3 (1.2)      | 1.3 (1.2)       | 1.6 (1.4)        |                  |
| Simulated LE/HLE disparity if   |                                |                  |                  |                  |                  |                  |
| No one was ever obese           | -2.9 (0.6)                       | 1.5 (0.5)        | 2.6 (0.5)        | n/a              | -4.1 (0.7)      | 0.2 (1.1)       | 1.7 (1.4)        |                  |
| No one smoked                   | -1.3 (1.2)                       | 2.3 (1.1)        | 2.8 (1.1)        | n/a              | -2.7 (1.1)      | 0.2 (1.4)       | 2.1 (1.4)        |                  |
| No smoking or obesity           | -1.0 (1.5)                       | 2.1 (1.4)        | 2.5 (1.3)        | n/a              | -2.3 (1.4)      | 0.2 (1.6)       | 1.6 (1.3)        |                  |

Notes: Source: NHANES, 1999–2000 through 2009–2010, N = 9923
Mean/(SD),
In a few cases, differences between observed TLE/HLE, counterfactual gains, and disparities do not perfectly align due to rounding error of decimal places.
*p < .05

Table 3
Men’s total and healthy life expectancy (TLE/HLE) at age 35: observed values, observed disparities, and simulated changes in both if obesity and smoking were eliminated.

| Panel 1: Observed values, observed disparities, and simulated changes in both if obesity and smoking were eliminated. |
|---------------------------------|---------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|                                | TLE                             | HLE              |                  |                  |                  |                  |
|                                | White                           | Black            | Hispanic USB     | Hispanic FB      | White           | Black           | Hispanic USB     | Hispanic FB      |
| Observed                        | 43.3 (0.0)                       | 40.1 (0.0)       | 45.0 (0.0)       | 46.3 (0.0)       | 31.2 (0.3)      | 26.8 (0.5)      | 30.6 (1.0)       | 33.5 (0.7)       |
| Simulated gains in LE/HLE if    |                                |                  |                  |                  |                  |                  |
| No one was ever obese           | 1.1 (0.4)                        | 1.4 (0.5)        | 0.9 (0.3)        | 0.7 (0.3)        | 1.7 (0.5)       | 1.9 (0.8)       | 1.6 (1.4)        | 1.1 (1.1)        |
| No one smoked                   | 3.8 (0.7)                        | 5.7 (1.0)        | 4.5 (0.9)        | 3.7 (0.7)        | 3.9 (1.0)       | 5.6 (1.1)       | 4.7 (1.6)        | 3.7 (1.2)        |
| No smoking or obesity           | 4.6 (1.0)                        | 6.8 (1.2)        | 4.2 (1.0)        | 5.0 (1.0)        | 5.8 (1.2)       | 7.8 (1.2)       | 6.5 (1.7)        | 5.1 (1.7)        |
| Panel 2: Observed disparity     | n/a                             | 1.6 (0.8)        | 3.0 (0.9)        | n/a              | 4.4 (0.5)       | -0.6 (1.0)      | 2.3 (1.3)        |                  |
| Simulated LE/HLE disparity if   |                                |                  |                  |                  |                  |                  |
| No one was ever obese           | -2.9 (0.6)                       | 1.5 (0.5)        | 2.6 (0.5)        | n/a              | -4.1 (0.7)      | 0.2 (1.1)       | 1.7 (1.4)        |                  |
| No one smoked                   | -1.3 (1.2)                       | 2.3 (1.1)        | 2.8 (1.1)        | n/a              | -2.7 (1.1)      | 0.2 (1.4)       | 2.1 (1.4)        |                  |
| No smoking or obesity           | -1.0 (1.5)                       | 2.1 (1.4)        | 2.5 (1.3)        | n/a              | -2.3 (1.4)      | 0.2 (1.6)       | 1.6 (1.3)        |                  |

Notes: Source: NHANES, 1999–2000 through 2009–2010, N = 9650
Mean/(SD),
In a few cases, differences between observed TLE/HLE, counterfactual gains, and disparities do not perfectly align due to rounding error of decimal places.
*p < .05
expectancy. Black women would make the largest gains (4.7 years of life and 7.1 years of healthy life expectancy). U.S.-born white and Hispanic women’s gains of 5.1 years of healthy life expectancy are also notable.

Turning to Panel 2, the first row repeats observed disparities in total and healthy life expectancy. The next three lines show how the elimination of smoking and obesity would change observed racial/ethnic/nativity disparities. The black-white life expectancy disparity is reduced to statistical non-significance under all three simulated conditions and the no smoking or obesity simulation reduces black women’s disadvantage from 2.2 years to .4 years (3 months). Each simulation also reduces black-white disparities in healthy life expectancy, but only the simulated elimination of both obesity and smoking reduces it to statistical non-significance.

U.S.-born Hispanic women’s significant life expectancy advantage over white women would decline by only .1 years if obesity were eliminated, and by .6 years if smoking were eliminated, but in both simulations the disparity remains statistically significant. However, if both smoking and obesity were eliminated, this would reduce the disparity by .5 years and to statistical non-significance. U.S.-born Hispanic women’s healthy life expectancy advantage was not statistically significant, and simulations do little to change this estimate.

Simulations change the magnitude but not the significance of foreign-born Hispanic women’s life expectancy advantage over white women. A large, statistically significant, nearly 3-year life expectancy advantage remains when we simulate the elimination of both obesity and smoking. However, when we simulate the elimination of smoking only, and the elimination of both smoking and obesity, foreign-born women’s healthy life expectancy advantage over white women would be reduced to non-significance.

4.3. Men’s total and healthy life expectancy

Table 3 provides results for men. Observed total and healthy life expectancy values are lower for men than women, but disparities across groups of men are similar to women. Black men have significant, large disadvantages in total (3.2 years) and healthy life expectancy (4.4 years) relative to whites. Foreign-born Hispanic men have significant, large advantages over white men in both outcomes. U.S.-born Hispanic men have a significant life expectancy advantage (1.6 years), but no significant healthy life expectancy advantage, over white men.

All groups of men would make significant predicted gains in life expectancy if obesity, smoking, or obesity and smoking were eliminated. These gains are larger than women’s gains under the same simulated conditions. All groups of men would also make significant gains in healthy life expectancy in the three simulations, with two exceptions. U.S.-born and foreign-born Hispanic men would not make significant healthy life expectancy gains if obesity were eliminated.

How would disparities in men’s total and healthy life expectancy change if obesity and/or smoking were eliminated? Results are similar to women’s results, with three exceptions. The “no obesity” scenario reduced the black-white disparity in life expectancy substantially and to statistical non-significance among women, but this simulation did little to reduce the significant black-white life expectancy disparity among men. Conversely, this simulation reduces foreign-born Hispanic men’s healthy life expectancy advantage over whites by .6 years and to statistical non-significance, but among women, the Hispanic foreign-born estimated advantage actually grew slightly and remained statistically significant.

The other small discrepant finding for men versus women is that the simulation in which both obesity and smoking are eliminated leads foreign-born Hispanic men’s life expectancy advantage to only be significant at the p < .10 level. It remained significant for women at the p < .05 level. Nonetheless, the estimated advantage is similar and large for both groups.

4.4. Supplementary analyses

To test the need to account for current and former obesity and smoking, we conducted alternative analyses that dropped former smoking and obesity. Results confirmed the importance of accounting for previous indicators. For example, results shown predict that Black women would make significant life expectancy gains of 1.7 years if obesity were eliminated and 4.7 years if obesity and smoking were both eliminated. When simulations draw from inputs that only account for current obesity and smoking, Black women’s life expectancy gain would be only .7 years if obesity were eliminated and 2.1 years if obesity and smoking were eliminated, and only the latter is statistically significant. Similar muted simulated gains in total and healthy life expectancy are evident for all groups of men and women, and this often mutates simulated changes in disparities.

We also checked study findings for robustness. We first treated current and former smoking and obesity as interactive rather than additive to produce inputs for simulations. Results from simulations produced using these inputs were substantively and statistically similar to results we show. We also replicated simulations using data from the NHIS, which includes a much larger sample of U.S. adults and in turn, more Hispanic respondents, to ensure that results did not reflect a lack of statistical power. Estimates from simulations using NHIS data were substantively and statistically similar to estimates from simulations using NHANES data that did not account for previous obesity, which the NHIS does not assess.

5. Discussion and conclusion

This study’s goal was to simulate how the elimination of smoking and obesity would reshape U.S. racial/ethnic and nativity disparities in total and healthy life expectancy at age 35. We were curious whether the U.S. could come closer to achieving health equity if the two leading causes of preventable deaths, chronic diseases, and disability were no longer health risks. We expected some changes in disparities given the known consequences of obesity and smoking and disparities in both health risks, but were unsure about the magnitude of expected changes and if they would be enough to significantly close racial/ethnic and nativity gaps in total and healthy life expectancy.

Results generally supported research expectations about black-white disparities. The simulated elimination of obesity reduced black-white gaps in total and healthy life expectancy, and among women, to the point that the gap was not statistically significant. We were unsure whether the elimination of smoking would reduce black-white gaps in study outcomes because black-white differences in smoking tend to be small, yet blacks are less likely to quit smoking than whites (Babb et al. 2017) and die more from smoking-related causes (Ho & Elo 2013). Under the “no smoking” scenario, we found large simulated reductions in the black-white disparity in life expectancy that led it to become statistically non-significant. The healthy life expectancy disparity was also reduced, but remained significant. Moreover, under the scenario in which both obesity and smoking are eliminated, there were no statistically significant black-white disparities in total or healthy life expectancy and these disparities were reduced by over two years for men and women on both study outcomes. These reduced disparities reflect the very large gains in total and healthy life expectancy that black women and men are estimated to make if obesity and smoking were eliminated. Across men and women, both study outcomes, and all simulated counterfactual conditions, the estimated gains of black adults outpace all other groups.

These results suggest that policy interventions that prevent obesity and weight gain, reduce smoking, and increase smoking cessation could make substantial progress in curbing black-white disparities in total and healthy life expectancy. That said, policies that blame the victim or point only to individualistic interventions are unlikely to succeed. The most successful interventions must attend to structural conditions that
lead U.S. black men and women to have elevated risks of long-term smoking and obesity. For example, tobacco companies disproportionately target minorities (National Cancer Institute, 2008) and minority neighborhoods (Center for Public Health Systems Science, 2014) as do fast food restaurants and corporations who sell soda (Grier & Kumanicky 2008). These and other forms of structural racism must be addressed. This is especially important in light of fundamental cause theory, which argues that in the absence of more proximate determinants of disease, other health problems stemming from more distal disadvantages will arise and take their place (Link & Phelan 1995). Originally, this theory explicitly and solely spoke to socioeconomic disadvantage, but it has been updated to address race and racism (Phelan & Link 2015).

When we compare Hispanic and white adults, results do not fall as neatly in line with research expectations. Hispanics’ life expectancy advantage over whites changed very little with the simulated elimination of obesity. It produced a .1-year reduction in the advantage of U.S.-born Hispanic men and women, a .2-year increase for immigrant women, and a .4-year reduction for immigrant men. We did not expect Hispanic men’s advantage to change much but expected Hispanic women’s advantage to grow. It only did so very modestly.

Findings were more consistent with research expectations when we simulated life expectancy if smoking was eliminated. As expected, eliminating smoking reduced the life expectancy advantage of Hispanic women regardless of nativity, and among immigrants, by over one year. This is consistent with earlier work on the topic (Fenelon 2013; Lariscy et al. 2015), although the magnitude of the explanatory power of smoking was somewhat smaller in our study compared to previous research. One possible reason is that our data, which are the most recent used in this area and are drawn from the 2000s, reflect recent declines in smoking that are particularly acute among white women. In contrast, the life expectancy advantage of U.S.-born Hispanic men actually grew by .7 and the advantage of immigrant men was only reduced by .2 years. The modest reduction in the life expectancy difference between immigrant Hispanic and white men with the elimination of smoking is much smaller than estimated in previous research. Moreover, the estimated increase in the life expectancy difference between US-born Hispanic men and non-Hispanic white men was not exhibited in earlier research (Fenelon 2013; Lariscy et al. 2015). These patterns may reflect the current modest differences in smoking behavior between Hispanic men (both foreign-born and U.S.-born) and white men, as shown in Table 1—differences that were much more pronounced among earlier cohorts of light-smoking Hispanic men and very heavy smoking white men (Fenelon 2013; Lariscy et al. 2015). Additionally, as expected, the simulated elimination of both smoking and obesity together led the life expectancy advantage of U.S.-born Hispanic men and women over whites to be reduced to statistical non-significance. Thus, smoking and obesity together explained the advantage of U.S.-born Hispanics relative to whites.

However, this was not the case for Hispanic immigrants. Even under the scenario in which both obesity and smoking are eliminated, the large advantage of Hispanic immigrant women over white women remains significant, and the advantage of Hispanic immigrant men remains large and fell just outside of the boundary of statistical significance at the p < .05 level. Clearly, the life expectancy simulations suggest that other social, behavioral and demographic processes are needed to fully explain Hispanic immigrants’ advantage. For example, relative to U.S.-born peers, Hispanic immigrants are more likely to eat healthy diets that are less like the typical American diet (Akresh 2007; Batis, Hernandez-Barrera, Barquera, Rivera, & Popkin, 2011; Van Hook, Quiros, & Frisco, 2015), less likely to have substance use disorders (Salas-Wright, Vaughn, Clark, Terzis, & Cordova, 2014), and they enjoy high levels of social capital and ethnic cohesion (Eschbach, Ostrir, Patel, Markides, & Goodwin, 2004). Self-selection of healthier immigrants into immigrant streams may also contribute to a persistent immigrant life expectancy advantage (Riosmena, Kuhn, & Jochem, 2017).

Findings regarding healthy life expectancy disparities between Hispanic and white adults are even more different than expected. This is largely because we based research expectations on a previous study that used the Health and Retirement Study (HRS). This study found that Hispanic adults, regardless of nativity, had a healthy life expectancy disadvantage relative to whites (Hayward et al. 2014). We found a healthy life expectancy advantage for Hispanic immigrants and no significant differences between U.S.-born whites and Hispanics. This is not entirely surprising. There is known variability in the measurement of ADL’s across the NHANES and the HRS (Glei, Goldman, Ryff, & Weinstein, 2017) and the two data sources tap a different subset of Hispanics, especially given that we analyze data from adults ages 35 and older rather than 50 and older. Our analysis suggested that the healthy life expectancy advantage that we found for Hispanic immigrants is largely driven by smoking among women and to some degree is driven by obesity and to a lesser extent smoking among men.

Study results and our interpretation of them should be considered within study limitations. First, even though we found no significant group differences in the associations of obesity and smoking with disability and mortality, this could be due to a lack of statistical power given the relatively small sample sizes for some groups in the NHANES. Second, social desirability bias may influence results and estimates of disparities if there are racial/ethnic and nativity biases in retrospective reports of weight at age 25, smoking behavior, or reports of ADL’s. Despite our efforts to reduce reporting error for the retrospective measures of obesity and smoking, measurement error could lead to an underestimation of their associations with disability and mortality. Finally, even though we control for educational attainment, our analysis cannot fully account for selection of people who are unhealthy or who have unhealthy or risky health behaviors into obesity and smoking. Our cross-sectional data also do not allow us to control for early life conditions such as low socioeconomic status in childhood or poor parental mental or physical health. This means that the associations of obesity and smoking with disability and mortality may be overestimated. For that matter, our cross-sectional data do not permit us to observe the many possible mechanisms that link racial and socioeconomic disadvantages to obesity and smoking in early adulthood, and these health conditions to later life health and mortality outcomes. Richer, longitudinal data would help us more fully account for the complex web of factors that may produce disparities such as discrimination, poor working conditions or a lack of access to health care.

Nonetheless, study results paint a portrait of how the leading two causes of preventable deaths in the U.S. may be influencing racial/ethnic health equity in longevity and disability. Smoking and obesity appear to play a major role in black-white disparities in length and quality of life and play some role in the advantage of Hispanic immigrants, especially in years of healthy life expectancy.

Ethics approval statement

This study uses free, publicly available data from the National Center for Health Statistics. It is exempt under the Penn State University Office of Research Protection IRB.

References

Akresh, I. R. (2007). Dietary assimilation and health among Hispanic immigrants to the United States. Journal of Health and Social Behavior, 48(4), 404–417.
Albrecht, S., Rezza, A. D., Aiello, A. E., Schulz, A., & Abraido-Lanza, A. (2013). Secular trends in the association between nativity/length of U.S. residence with body mass index and waist circumference among Mexican-Americans, 1988–2008. International Journal of Public Health, 58(4), 573–581.
Antecol, H., & Bedard, K. (2006). Unhealthy assimilation: why do immigrants converge to American health status levels? Demography, 43(2), 337–360.
Arias, E. (2016). Changes in life expectancy by race and Hispanic origin in the United States, 2013–2014. Data Briefs, NCHS Statistics. Hyattsville, MD: National Center for Health Statistics.
Arias, E., Heron, M., & Xu, J. (2017). United States life tables, 2014. National Vital Statistics System, 66(9). Hyattsville, MD: National Center for Health Statistics.
M.L. Frisco, et al.

Statistics Reports, 66(4), 1-64.

Babb, S. D., Malaracher, A., Schauer, G., Asman, K., & Jamal, A. (2017). Quitting smoking among adults-United States, 2000-2015. MMWR Morbidity and Mortality Weekly Report, 65, 1457–1464.

Bates, L. M., Acevedo-Garcia, D., Alegria, M., & Krieger, N. (2008). Immigration and ethnic disparities in smoking-cessation interventions: Analysis of the 2005 National Health Interview Survey. American Journal of Preventive Medicine, 34(5), 404–412.

Eschbach, K., Ostir, G. V., Patel, K. V., Markides, K. S., & Goodwin, J. S. (2005). Neighborhood context and mortality among older Mexican Americans: Is There a barrio Advantage? American Journal of Public Health, 94(10), 1807-1812.

Fenelon, A. (2013). Revisiting the Hispanic mortality advantage in the United States: The role of smoking. Social Science and Medicine, 82, 1-9.

Ferraro, K. F., & Kelley-Moore, J. A. (2003). Cumulative disadvantage and health: Long-term consequences of obesity? American Sociological Review, 68(5), 707-729.

Flegal, K. M., Carroll, M. D., Ogden, C. L., & Curtin, L. R. (2010). Prevalence of childhood and adult obesity in the United States, 2007-2008. JAMA, 303(3), 235–241.

Griët, D., Goldman, N., Ryff, C. D., & Weinstein, M. (2017). Can we determine whether physical limitations are more prevalent in the US than in countries with comparable life expectancy? Social Science Medicine-Population Health, 3, 808–813.

Grier, S., & Kumanyika, S. K. (2008). The context for choice: Health implications of targeted food and beverage marketing to African Americans. American Journal of Public Health, 98(9), 1616–1629.

Hales, C. M., Fryar, C. D., & Carroll, M. D. (2018). Trends in obesity and severe obesity prevalence in U.S. Youth and adults by sex and age, 2007-2008 to 2015-2016. JAMA, 319(14), 1723-1725.

Handcock, M. S., Huovilainen, S. M., & Rendall, M. S. (2000). Combining registration-system and survey data to estimate birth probabilities. Demography, 37(2), 187–192.

Handcock, M. S., Rendall, M. S., & Cheadle, J. E. (2005). Improved regression estimation of a multivariate relationship with population data on the bivariate relationship. Sociological Methodology, 35(1), 291–334.

Hayward, M. D., Hummer, R. A., & Chiu, C.-T. (2014). Does the Hispanic Paradox in U.S. adult mortality extend to disability? Population Research and Policy Review, 33(1), 81–96.

Ho, J. Y., & Elo, I. T. (2013). The contribution of smoking to black-white differences in U.S. mortality. Demography, 50(2), 545–568.

Jaggers, C., Cox B., and Le Roy S. (2006). Health expectancy calculation by the Sullivan method: HEMU Technical Report 2006-3.

Jamal, A., Phills, E., Gentzke, A. S., Homa, D. M., Babb, S. D., King, B. A., & Neff, L. J. (2018). Current cigarette smoking among adults—United States, 2016. MMWR Morbidity and Mortality Weekly Report, 67, 53-59.

Lariczy, J. T., Hummer, R. A., & Hayward, M. D. (2015). Hispanic older adult mortality in the United States: New estimates and an assessment of factors shaping the Hispanic Paradox. Demography, 52(1), 1-14.

Lariczy, J. T., Hummer, R. A., & Rogers, R. G. (2018). Cigarette smoking and all-cause and cause-specific mortality in the United States. Demography, 55(5), 1855-1885.

Link, B. G., & Phelan, J. (1995). Social conditions as fundamental causes of disease. Journal of Health and Social Behavior, Extra Issue, 80-94.

Markides, K. S., & Eschbach, K. (2011). Hispanic Paradox in adult mortality in the United States. In R. G. Rogers, & E. Crimmins (Eds.), International Handbook of Adult Mortality (pp. 227–240). New York, NY: Springer.

Masters, R., Powers, D., & B. Link, G. (2013). Obesity and U.S. mortality risk over the adult life course. American Journal of Epidemiology, 177(5), 431–442.

McDonald, J. A., & Paulozzi, L. J. (2018). Parsing the Paradox: Hispanic mortality in the U.S. by detailed cause of death. Journal of Immigrant and Minority Health, 1–9 (online first).

National Cancer Institute (2008). The role of the media in promoting and reducing tobacco use. In: Smoking and Tobacco Control Monograph No. 19, NIH Pub. No. 07-6242. Bethesda, MD: National Institute of Health.

Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2014). Prevalence of childhood and adult obesity in the United States, 2013–2014. JAMA, 311(8), 806-814.

Olshansky, S. J., Passaro, D. J., Herzog, R. C., Layden, J., Carnes, B., Brody, J., Hayflick, L., Butler, R. N., Allison, D. B., & Ludwig, D. S. (2005). A potential decline in life expectancy in the United States in the 21st century. Obstetrical Gynecological Survey, 60, 450–452.

Peto, R., Whitley, G., & Jha, P. (2010). Effects of obesity and smoking on U.S. life expectancy. New England Journal of Medicine, 362(9), 855-857.

Phelan, J., & Link, B. G. (2015). Is racism a fundamental cause of inequalities in health? Annual Review of Sociology, 41, 311-330.

Preston, S., & Wang, H. (2006). Sex mortality differences in the United States: The role of cohort smoking patterns. Demography, 43, 631–646.

Preston, S. H., & Stokes, A. (2014). Obesity paradox. Epidemiology, 25(3), 454–461.

Preston, S. H., Stokes, A., Mehta, N. K., & Can, B. (2014). How will more obesity and less smoking affect life expectancy? Boston: Center for Retirement Research at Boston College.

Preston, S. H., Verboom, Y. C., & Stokes, A. (2018). The role of obesity in exceptionally slow U.S. mortality improvement. Proceedings of the National Academies of Sciences, 115(5), 957–961.

Riosnema, F., Kuhn, R., & Jochem, C. (2017). Explaining the immigrant health advantage: Self-selection and protection among five major national-origin immigrant groups in the United States. Demography, 54(1), 175–200.

Rowland, D. T. (2003). Demographic methods and concepts. New York: Oxford University Press.

Rubin, R. (1987). Multiple imputation for nonresponse in surveys. New York: Wiley.

Salas-Wright, C. P., Vaughn, M. G., Clark, T. T., Terriz, L. D., & Cordova, D. (2014). Substance use disorders among first- and second-generation immigrant adults in the United States: Evidence of an immigrant paradox? Journal of Studies on Alcohol and Drugs, 75(6), 958–967.

Stewart, S. T., Cutler, D. M., & Rosen, A. B. (2009). Forecasting the effects of obesity and smoking on U.S. life expectancy. New England Journal of Medicine, 361(23), 2252-2260.

Sullivan, D. F. (1971). A single index of mortality and morbidity. HSMHA Health Reports, 86(4), 347–354.

Trinidad, D. R., Pérez-Stable, E. J., White, M. M., Emery, S. L., & Messer, K. (2011). A nationwide analysis of US racial/ethnic disparities in smoking behaviors, smoking cessation, and cessation-related factors. American Journal of Public Health, 101(4), 699-706.

U.S. Department of Health and Human Services (2014). The health consequences of smoking-50 years of progress: A report by the surgeon general. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion.

Van Hook, J., Quiroz, S., & Frisco, M. L. (2015). The food similarity index: A new dimension of dietary acculturation based on dietary recall data. Journal of Immigrant Minority Health, 17(2), 441–449.