The Shape of Things to Come: Will Changes in Prevalence of Obesity and Smoking Influence the Quantity and Quality of Life of White, Black and Hispanics in the U.S.?

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# The Shape of Things to Come: Will Changes in Prevalence of Obesity and Smoking 

## Influence the Quantity and Quality of Life of White, Black and Hispanics in the U.S.?


#### Abstract

Obesity and smoking are the two leading causes of preventable deaths and disability in the U.S., though trends in both health problems have changed significantly across birth cohorts. The current study estimates the extent to which changes in early adult obesity and lifetime smoking across birth cohorts born in the 1940s, 1950s, 1960s, 1970s, 1980s and 1990s are associated with life expectancy and healthy life expectancy at age 35 using data from the National Health and Nutrition Examination Studies and Linked Mortality files from 1999-2009. Simulations suggest that cohort changes in obesity are associated with a reduction in life expectancy and an increase in years spent disabled, while cohort changes in smoking are associated with increased life expectancy and spending fewer years of life as disabled. On balance, trends in obesity and smoking largely offset each other. Study findings are consistent across men and women and among U.S.-born black and white adults. Consistent patterns are also observed for U.S.-born and foreign-born Hispanic adults, although results from simulations estimating changes in healthy life expectancy due to cohort changes in obesity and smoking are not statistically significant. Study results have important implications for considering whether approaches to population health that focus on upstream drivers of life expectancy and disability would be more effective than focusing on downstream drivers that simply may replace each other over time with new, negative health problems.


## Introduction

Obesity and smoking are the two leading causes of preventable deaths and disability in the U.S., and trends in both health problems have changed significantly over the last 50 years. In 1965, nearly half of all adults in the U.S. smoked. The percentage of U.S. adult smokers has fallen steadily over time to only $15.5 \%$ of Americans in 2016 (Centers for Disease Control and Prevention 2016b, 2018). Conversely, during this same time period trends in obesity have gone in the opposite direction. From the mid-1960s until the mid-1970s the obesity prevalence of both men and women was relatively flat and low compared to today's estimates. Since the mid-70s when $10 \%$ of men and $15 \%$ of women were obese (Fryar, Carroll and Ogden 2016), the obesity prevalence has skyrocketed, with the most recent estimates suggesting that $39.6 \%$ of U.S. adults are currently obese (Hales, Fryar and Carroll 2018). These changing obesity estimates are also observed when same-aged adults are compared across birth cohorts; the earlier birth cohorts have lower mean BMIs (McTigue, Garrett and Popkin 2002), and this same pattern has been observed when cohorts of adolescents and young adults ages 12 - 26 were compared. Relative to sameaged peers assessed at one of 4 times between 1959 and 1980, adolescents in the 1990s and 2000s and young adults in the 2000s weighed significantly more, a pattern that was more evident among girls than boys and among U.S. African Americans (Lee et al. 2011).

In the current study, we ask whether and how offsetting trends of reduced smoking prevalence and increased obesity prevalence are associated with cohort changes in the life expectancy and healthy life expectancy of U.S. men and women ages 35 and older. We simulate how life expectancy and healthy life expectancy are likely to change as a consequence of changes in smoking and obesity across six cohorts born in the 1940s through the 1990s. We use
the 1999-2009 National Health and Nutrition Examination Studies and Linked Mortality Files to construct life tables by obesity and smoking status, and then assess how cohort changes in obesity and smoking are related to changes in total and healthy life expectancy. We produce results separately for U.S.-born white, U.S.-born black and U.S-born and foreign-born Hispanic men and women.

We present separate estimates by race/ethnicity, nativity and sex due to documented current and historical sex, racial/ethnic and nativity differences in mortality and disability. Presenting estimates by race/ethnicity and nativity is also important when considering historical racial/ethnic and nativity patterns of smoking and obesity. In general, smoking has been declining for all groups in our study over time since the 1960s (Centers for Disease Control and Prevention 2016a). Findings are not as uniform when obesity is considered. For example, at the turn of the century, similar proportions of white, black and Mexican-American men were obese (around 28\%), but obesity prevalence increased more for black and Mexican-American men than whites. By around 2010, 37.2\% and $35.9 \%$ of Black and Hispanic men were obese compared with $31.9 \%$ of white men (Flegal, Caroll and Ogden 2010). During this same time frame, obesity among white women was consistently lower than Black women. About one-third of white women versus half of black women were obese. However, obesity among Mexican-American women gradually increased from $29.7 \%$ to $45.1 \%$ (Flegal et al. 2010).

The central contribution of our study is showing whether younger cohorts of Americans may experience declines in life-expectancy and disability-free life expectancy as a result of the U.S. obesity epidemic or whether declines in smoking produce offsetting gains that lead to little cohort variation in life-expectancy and disability-free life expectancy. Study results have
important implications for understanding how obesity and smoking will contribute to trends in two important indicators of population health.

## Methods

Total life expectancy measures how many years after age 35 people live on average. In our study, healthy life expectancy refers to the average number of years of life after age 35 and before death that people experience no limitations in their ability to perform activities of daily living (ADLs). We first produced estimates of total and healthy life expectancy at age 35 for U.S.-born white, Black and Hispanics and foreign-born Hispanics by sex. Then, we produced hypothetical estimates of total and health life expectancy that change the levels of obesity and smoking prevalence similar to the observed changes in prevalence across birth cohorts from the 1940s to the 1990s.

We used the National Health and Nutrition Examination Studies (NHANES), a large national health survey with mortality follow-up linkages, to estimate inputs for the life expectancies. We describe the data and measure for these inputs in section IIa. We then used these inputs, in combination with the conditional probabilities of dying in the published National Center for Health Statistics life tables, to calculate how cohort changes in obesity and smoking prevalence are related to changes in total and healthy life expectancy, as described in section IIb. IIa. Data and Measures for Life Expectancy and Healthy Life Expectancy Estimates

Data were drawn from the 1999-2009 NHANES and linked mortality files. We used the NHANES because it includes both current, measured assessments of height and weight and retrospective self-reports of height and weight at age 25 . We restrict the sample to adults ages 35 or older. We pool data across multiple survey years and impute all missing data. Our final
sample includes 19,574 adults age 35+ (3,488 foreign born Hispanics, 1,959 U.S.-born Hispanics, 3,828 blacks, and 10,299 non-Hispanic whites).

We also used the National Center for Health Statistics (NCHS) 2012 life tables for Hispanic, white, and black adults by sex. We used these published life tables to calibrate our estimates of the age-specific conditional probability of dying in our life tables (the $\mathrm{q}_{\mathrm{x}}$ 's). This is explained further below. Suffice it to say, calibration ensures that our life expectancy estimates for each race/ethnic and sex group match the estimates produced by NCHS. It also ensures that the weighted average of the conditional probabilities of dying for subgroups (e.g., by smoking or by weight status) equals the conditional probability for the overall group as estimated by NCHS.

Our estimates of life expectancy were based on models predicting the hazard of death as a function of obesity status at age 25 , whether the respondent ever smoked, the interaction of obesity and smoking status, education, age, and survey year, estimated separately by sex. A key dependent variable was whether the person died during the follow-up period after the NHANES interview but before January 1, 2010, and age at death among decedents. Vital status and age at death, measured to the nearest quarter-year, was obtained from the linked death certificates data. Our estimates of healthy life expectancy relied on logistic regression models predicting no disability as a function age, obesity status, smoking, an interaction term between obesity and smoking status, education, and race/ethnicity/nativity, estimated separately by sex. Disability status was defined as the inability of NHANES respondents to perform one or more activities of daily living (ADLs), including dressing, eating, and getting in or out of bed or chairs.

A key independent variable was an indicator of whether individuals were obese at age 25, based on self-reports of weight at age 25 . We relied on weight in young adulthood rather than current weight to better tap cohort changes in weight status while holding the effects of age
constant. Between now and PAA, we plan to conduct a series of robustness checks to assess the validity of our retrospective measure of obesity, and if necessary, we will develop a method for adjusting it for recall/measurement error. Our second key independent variable is smoking status. This variable indicates whether respondents ever smoked more than 100 cigarettes in their life.

Additional measures in the NHANES data include age at interview (categorized into 5year groupings, with $85+$ as the highest category), sex, and educational attainment (less than high school, high school, some post-secondary education, and college graduate). We also account for race, Hispanic ethnicity, and for Hispanic respondents, foreign place of birth. Individuals were classified as U.S.-born, non-Hispanic white, Black or Hispanic or as foreign-born Hispanic. IIb. How Total, Healthy, and Unhealthy Life Expectancy are Related to Weight and Smoking

In this section, we 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 had a different weight status or smoking distribution (i.e., if obesity and smoking changed as much as it did between the 1940s to the 1990s birth cohorts).

Life expectancy (LE) is an estimate of average length of life for a synthetic cohort that experiences the complete set of conditional probabilities of dying $\left(\mathrm{q}_{\mathrm{x}}\right)$ across the lifespan. LE is related to obesity and smoking because the probabilities of dying (the $\mathrm{q}_{\mathrm{x}}$ 's) are higher among people who are obese relative to people who are not obese, and higher among those who ever smoked than those who never smoked. Taking obesity as an example, each of the $\mathrm{q}_{\mathrm{x}}$ 's can be expressed as the sum of weight-specific $\mathrm{q}_{\mathrm{x}}$ 's for those who were not obese ( $\mathrm{w}_{1}$ ) and obese at age $25\left(\mathrm{w}_{2}\right)$, weighted by the proportions in each of the two weight categories $\left(\mathrm{p}_{\mathrm{x}, \mathrm{w} 1}\right.$ and $\left.\mathrm{p}_{\mathrm{x}, \mathrm{w} 2}\right)$.

$$
\begin{equation*}
\mathrm{q}_{\mathrm{x}}=\mathrm{q}_{\mathrm{x}, \mathrm{w} 1} \mathrm{p}_{\mathrm{x}, \mathrm{w} 1}+\mathrm{q}_{\mathrm{x}, \mathrm{w} 2} \mathrm{p}_{\mathrm{x}, \mathrm{w} 2} \tag{eq1}
\end{equation*}
$$

If the risk of death is greater for the higher-weight categories, equation 1 shows how a group's overall $\mathrm{q}_{\mathrm{x}}$ value could increase if the share of people in the higher-weight categories were to increase, which would then lower the group's life expectancy. Similarly, the $\mathrm{q}_{\mathrm{x}}$ 's can be expressed as the sum of the conditional probabilities of dying among people who never smoked $\left(s_{1}\right)$ and those who ever smoked $\left(s_{2}\right)$, weighted by the proportions in each of these two groups.

$$
\begin{equation*}
\mathrm{q}_{\mathrm{x}}=\mathrm{q}_{\mathrm{x}, \mathrm{~s} 1} \mathrm{p}_{\mathrm{x}, \mathrm{~s} 1}+\mathrm{q}_{\mathrm{x}, \mathrm{~s} 2} \mathrm{p}_{\mathrm{x}, \mathrm{~s} 2} \tag{eq2}
\end{equation*}
$$

Equations 1 and 2 are central to how we estimate observed and counterfactual life expectancies. To obtain $\mathrm{q}_{\mathrm{x}}$ values (and the corresponding life expectancies), we estimated and inserted values for each of the terms in equation 1 for each group. To estimate the $p_{x, w}$ and $p_{x, s}$ terms, we used the NHANES data to calculate the age-specific proportions in each weight and smoking category for each group by sex. To estimate the $\mathrm{q}_{\mathrm{x}, \mathrm{w}}$ and $\mathrm{q}_{\mathrm{x}, \mathrm{s}}$ terms in equations 1 and 2 , we again used 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, using the NHANES data, we first estimated proportional hazard Cox models predicting the hazard of dying as a function of weight status and smoking status while controlling for age at interview, race/ethnicity/nativity, and education. The model coefficients tell us how weight and smoking are related to the conditional probability of dying, net of the other factors in the model. The models were estimated separately by sex and were weighted and adjusted to account for the stratified, clustered NHANES sample design. We tested several alternative model specifications ${ }^{1}$, but the results were consistent across specifications.

[^0]We then used the estimated hazard ratios for the weight and smoking categories (expressed as ratios relative to the sample average) to obtain estimates of the weight- and smoking-specific $\mathrm{q}_{\mathrm{x}}$ 's ${ }^{2}$. Specifically, we then multiplied the hazard ratios (H) by the NCHS published $\mathrm{q}_{\mathrm{x}}$ 's ( $\mathrm{q}_{\mathrm{x}, \mathrm{NCHS}}$ ), separately by race/ethnic and sex. For example, the $\mathrm{q}_{\mathrm{x}, \mathrm{w}}$ terms for Hispanic women in equation 1 were calculated as:

$$
\begin{aligned}
& q_{x, w 1, \text { Hispanic female }}=q_{x, N C H S} \text {, Hispanic female } H_{w 1, \text { female }} \\
& q_{x, w 2, \text { Hispanic female }}=q_{x, N C H S} \text {, Hispanic female } H_{w 2, \text { female }}
\end{aligned}
$$

We repeated this for all race/ethnic/nativity groups and by gender, and we estimated the $\mathrm{q}_{\mathrm{x}}$ 's for people who ever smoked and never smoked in the same manner. Multiplying the hazard ratios by the NCHS estimates ensures that the weight- and smoking-specific estimates, when averaged across categories, match the NCHS estimates for all weight- and smoking categories combined.

After estimating values for equation 1, we used standard demographic methods to estimate life expectancy from the estimated $\mathrm{q}_{\mathrm{x}}$ 's (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 $\mathrm{q}_{\mathrm{x}}$ 's and life expectancies, we changed the $\mathrm{p}_{\mathrm{x}}$ values to reflect changes in obesity and smoking across different birth cohorts. That is, we set the values of $\mathrm{p}_{\mathrm{x}, \mathrm{w} 1}$, $p_{\mathrm{x}, \mathrm{w} 2}, \mathrm{p}_{\mathrm{x}, \mathrm{s} 1}$, and $\mathrm{p}_{\mathrm{x}, \mathrm{s} 2}$, to observed values for those born in the 1940s, 1950s, 1960s, 1970s, 1980s, and 1990s. This information was generated from all adults ages 20+ in the NHANES from 1999

[^1]through 2015 (i.e., more age groups and years than we used to estimate the mortality and disability models). For those born in the 1980s and later, we used observed proportion obese for those ages 20-29 in place of the retrospective measure of obesity at age 25 . Changing the $p_{x}$ values in equations 1 and 2 produced counterfactual $\mathrm{q}_{\mathrm{x}}$ values, which we used to produce estimates of how life expectancy would change due to cohort changes in obesity and smoking.

We use a similar approach to estimate observed and counterfactual healthy life expectancies (HLE) at age 35 for each race/ethnic/nativity group by sex. To simplify the calculations, we relied on the Sullivan method for estimating HLE ${ }^{3}$. With the Sullivan method, HLE can be clearly expressed as a function of the probability of dying $\left(q_{x}\right)$ 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 $\left({ }_{n} \mathrm{~L}_{\mathrm{x}}\right)$ in each age interval by the proportion of with at least one $\operatorname{ADL}\left(\mathrm{A}_{\mathrm{x}}\right)$ to obtain person-years lived between age x and $\mathrm{x}+\mathrm{n}$ in the $\operatorname{ADLS}\left({ }_{\mathrm{n}} \mathrm{L}_{\mathrm{x}}{ }_{\mathrm{x}}\right)$ and non-ADL states $\left({ }_{n} L^{n a}{ }_{x}={ }_{n} L_{x}-{ }_{n} L^{a}\right.$ ) , and (3) summing ${ }_{n} L^{n a}{ }_{x}$ from age $x$ to the oldest age and dividing by the number of survivors to age $\mathrm{x}\left(\mathrm{l}_{\mathrm{x}}\right)$ to obtain the total HLE at each age x (Jagger et al. 2006). Unhealthy life expectancy is total life expectancy minus HLE.

The weight status and smoking distributions of a group is mathematically related to HLE in two ways. First, they could affect the probabilities of dying, as just discussed. Second, they could affect the proportion of each group with an $\operatorname{ADL}\left(\mathrm{A}_{\mathrm{x}}\right)$. For example, $\mathrm{A}_{\mathrm{x}}$ can be expressed as the weighted average of ADL rates across the two weight status categories:

$$
\begin{equation*}
A_{x}=A_{x, w 1} p_{x, w 1}+A_{x, w 2} p_{x, w 2} \tag{eq3}
\end{equation*}
$$

[^2]If the risk of having an ADL is greater for the higher-weight categories, equation 3 shows how a group's overall $\mathrm{A}_{\mathrm{x}}$ value would increase if the share of people in the higher-weight categories were to increase, which in turn would lead to declines in HLE. A similar logic applies to the share of people who never smoked and ever smoked.

To estimate HLEs for each group, we used equations 1 and 2 to calculate the $\mathrm{q}_{\mathrm{x}}$ terms, as already described, and equation 2 to calculate the $\mathrm{A}_{\mathrm{x}}$ terms. We combined the results, using the Sullivan method, to estimate HLE. To obtain values for the $\mathrm{A}_{\mathrm{x}, \mathrm{w}}$ terms in equation 2, we first estimated logistic regression models predicting the probability of having an ADL as a function of weight status (or smoking status) while controlling for age at interview and race/ethnicity/nativity. The models were estimated separately by sex and were weighted and adjusted to account for the stratified, clustered NHIS sample design. Similar to how we estimated the weight- and smoking-specific $\mathrm{q}_{\mathrm{x}}$ estimates, we used odds ratios from the models to estimate the $\mathrm{A}_{\mathrm{x}, \mathrm{w}}$ values while ensuring that their weighted average across the two weight and smoking status categories equals the observed age-specific ADL rate within each gender and race/ethnic/nativity group`.

Finally, to calculate counterfactual HLE estimates, we repeated the calculations just described only we assumed different levels of obesity and smoking that corresponded with the levels observed for each birth cohort.

## Results

## Cohort Differences in Early Adult Weight and Smoking Behavior, Life Expectancy, \& Healthy

## Life Expectancy

Table 1 shows estimates of the percentage of respondents reporting that they were obese at age 25 (or obese 10 years ago in the case of respondents born in the 1990s) and whether they
were current or former smokers. The top panel reports results for women. In general, these results are in line with what is known about the increasing obesity prevalence that began in the mid-1970s. Less than $10 \%$ of women born in the 1940s reported that they were obese at age 25 , but among the 1980s cohort, $27.2 \%$ of white women, $29.3 \%$ of foreign-born Hispanic women, 34.4.\% of U.S.-born Hispanic women, and $49.8 \%$ of Black women were obese at age 25. Trends in smoking are also consistent with national estimates. Women born in the 1940s and 1950s are more likely to smoke than those women born in the 1980s and 1990s. We note, though that there is racial/ethnic and nativity variation in when substantial cohort drops in smoking occurred. Among white women, a substantial consistent decline is not observed until the 1980s, whereas substantial declines for Hispanic women regardless of nativity and black women are observed in the 1970s and 1960s respectively.

Estimates for men follow similar trends as women. In general, the obesity prevalence among birth cohorts increases over time. Less than $7 \%$ of men born in the 1940s reported being obese at age 25, but by the 1980s roughly one-quarter of white and foreign-born Hispanic men were obese at age 25 and 29.8 and $39.4 \%$ of U.S.-born Black and Hispanic men were obese at age 25. The smoking prevalence also declines across birth cohorts. This decline is linear and consistently downward for U.S.-born and foreign-born Hispanics. Among Black men, though, there is little difference in percentage who smoke among the 1970s, 1980s and 1990s birth cohort. Conversely, among white men the percentage of smokers in the 1950s birth cohort is much smaller relative to the 1940s birth cohort, but then the percentage of smokers across cohorts hovers around $50 \%$ until the 1990s birth cohort begins to report smoking behavior. Estimates of Cohort Changes in Life Expectancy and Healthy Life Expectancy among Women

Table 2 presents estimates from models indicating whether cohort changes in obesity and smoking between 1940 and 1990 are associated with changes in life expectancy (top panel) and healthy life expectancy (bottom panel) between women in the youngest and oldest birth cohorts in the sample. The first row of results shows actual average life expectancy at age 35 for all women in the study by race/ethnicity and nativity. On average, white women's life expectancy is 47.3 years and black women's life expectancy is only 45.1 years. Hispanic women, regardless of nativity have the longest life expectancy, although foreign-born Hispanics are expected to live two more years than U.S.-born Hispanics.

These life expectancy estimates are period estimates, meaning that they pertain to mortality and disability patterns for a mixture of birth cohorts during the first decade of the 2000s. The next row of results shows these estimates would change if the population experienced changes in obesity at age 25 on par with the cohort changes in obesity observed between the 1940s and 1990s birth cohorts. Results suggest that on average all women across groups would experience a significant decline in life expectancy, though this decline is largest for U.S-born Hispanics. We should note, though, that the simulated differences are not large enough to change the long-standing immigrant advantage of Hispanic women or the longstanding disadvantage of Back women.

The next row of results shows how cohort declines in smoking would change life expectancy. All women would experience a significant gain in life expectancy given changes in the prevalence of smoking from the 1940s to 1990s birth cohort. These gains range from 1.3 years for white and Hispanic U.S.-born women to 2.0 years for black women.

The final row of results from life expectancy simulations for women show how expected longevity would change if they experienced changes in both obesity and smoking that are on par
with the changes seen between the 1990s birth cohort and the 1940s birth cohort. These results suggest that among white and Black women, obesity and smoking changes largely cancel each other out. Among Hispanic women, though, changes in obesity outweigh changes in smoking and still result in slightly more than a 1 year loss in life expectancy. This simulated change is not estimated as being statistically significant, but we suspect this is due to a lack of statistical power and plan to consider this issue further prior to the 2019 PAA annual meeting.

We next turn to actual and simulated values of healthy life expectancy when the 1940s and 1990s birth cohorts are compared. On average, women in the sample can expect to experience around 30 years of healthy life expectancy, with black women estimated as having the fewest years of healthy years (28.9 years) and foreign born Hispanic women having the most healthy years ( $35.3 \%$ ). Note, that these estimates suggest that the average healthy life expectancy is 15 years fewer than actual life expectancy.

Increases in obesity and decreases in smoking across cohorts are not estimated as changing Hispanic women's healthy life expectancy, though we should note that we again plan to test whether the large non-significant estimated differences shown in Table 2 are due to issues of statistical power prior to the 2019 PAA annual meeting. Among black and white women, increases in obesity and decreases in smoking largely offset each other.

## Estimates of Cohort Changes in Life Expectancy and Healthy Life Expectancy among Men

Table 3 shows estimates of changes in life expectancy and healthy life expectancy that would occur if they experienced changes in obesity and smoking on par with the changes observed between the 1940s and 1990s birth cohorts. The top panel of results show life expectancy estimates and the pattern of results for men is quite similar to the pattern observed for women. The actual life expectancies of all men are lower than that of women, but race/ethnic and
nativity differences are the same. Black men's life expectancy is lower than white men's life expectancy and Hispanic men (and especially those who are foreign-born) have a life expectancy advantage over white and black men. In addition, once again, the change in obesity at age 25 from the 1940s birth cohort to the 1990s birth cohort is associated with a reduction in life expectancy for all men, while changes in smoking are associated with an increase in life expectancy for all groups except foreign-born Hispanic men. We should also note that when changes in both obesity and smoking are considered in tandem, foreign-born Hispanic men are still estimated to lose 1.8 years of life expectancy, though this estimated difference is not statistically significant. We plan to explore whether this is due to statistical power issues prior to the 2019 PAA annual meeting.

The bottom panel of results shows estimates of healthy life expectancy. The first row of results are actual values of healthy life expectancy at age 35 for all groups of men. Foreign-born Hispanic men have the longest healthy life expectancy and Black men have the lowest health life expectancy. As was the case among women, birth cohort changes in obesity at age 25 suggest reduced healthy life expectancies among white and black men, while birth cohort changes in smoking suggest healthy life expectancy gains among white and black men. Neither changes in obesity nor smoking are estimated to significantly change the healthy life expectancy of Hispanic men, though again, our ongoing work will assess whether this is due to issues of statistical power.

## Discussion and Next Steps

Our analyses to date suggest that birth cohort changes in early adult obesity and current and former smoking behavior have offsetting simulated effects on life expectancy and healthy life expectancy at age 35 . Furthermore, although our study is not explicitly interested in
racial/ethnic and nativity disparities in these outcomes, there is little evidence that changes in early adult obesity and smoking will substantially change disparities. It is also notable that results are quite consistent among U.S. women and men, though this may in part result from comparing only the youngest and oldest cohorts in work to date, where the widest differences in obesity and smoking are observed.

Nonetheless, we emphasize that our results are preliminary and we plan to address several limitations of our analysis to date. First, the negative consequences of obesity for disability and longevity accumulate over time (Ferraro and Kelley-Moore 2003; Reilly and Kelly 2011) as do the consequences of smoking (Taghizadeh, Vonk and Boezen 2016). Currently our simulations are based on self-reported estimates of obesity at the age of 25 that likely contain some error and do not fully tap the risks of early adult obesity for youngest cohorts, who are actually more likely than older cohorts to be obese well before the age of 25 . In our ongoing work, we plan to use other data to evaluate the validity of our estimates of obesity and cohort differences in life course exposure to obesity and possibly adjust them for measurement error. Similarly, our current conceptualization of smoking is quite blunt and does not disaggregate smoking cessation from current smoking or heavy smokers from light and moderate smokers. Our ongoing work will account for smoking variability given that how long one smokes and how much one smokes both influence lifetime mortality and disability.

Results to date, though, have important implications. While it is encouraging that reductions in smoking could have positive implications for life expectancy and healthy life expectancy, it is discouraging to see how obesity may erode life expectancy gains. These results suggests support for fundamental cause theory, which argues that "upstream" determinants of health such as socioeconomic status (Link and Phelan 1995; Phelan, Link and Tehranifar 2010)
and race/ethnicity (Daw 2015) are more likely to move the needle and improve health outcomes because new downstream health problems like obesity will arise in the amelioration of other health problems such as smoking that poor health outcomes such as reduced life expectancy and increased disability.

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Table 1. Changes in obesity at age 25 and smoking prevalence by birth cohort and race/ethnicity/nativity

|  | \% Obese at Age 25 |  |  |  | \% Ever Smoked |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | NH- <br> White | FB <br> Hispanic | USB <br> Hispanic | NH- <br> Black | NH- <br> White | FB <br> Hispanic | USB <br> Hispanic | NH- <br> Black |
| Women |  |  |  |  |  |  |  |  |
| 1940s | 3.6 | 5.0 | 8.5 | 5.6 | 50.1 | 23.5 | 40.9 | 48.2 |
| 1950s | 9.3 | 9.5 | 16.1 | 16.8 | 45.9 | 32.2 | 43.7 | 48.8 |
| 1960s | 16.7 | 14.9 | 24.7 | 22.5 | 49.9 | 22.4 | 43.4 | 36.7 |
| 1970s | 18.2 | 13.9 | 21.4 | 30.5 | 47.3 | 12.9 | 29.8 | 27.1 |
| 1980s | 27.2 | 29.3 | 41.0 | 49.8 | 40.7 | 12.2 | 27.6 | 27.1 |
| 1990s | 27.4 | 37.1 | 34.4 | 32.8 | 34.6 | 11.5 | 17.8 | 16.3 |
| Change | 23.7 | 32.1 | 26.0 | 27.1 | -15.6 | -11.9 | -23.1 | -31.9 |
| Men |  |  |  |  |  |  |  |  |
| 1940s | 5.8 | 3.0 | 6.4 | 6.6 | 65.0 | 62.7 | 70.0 | 70.9 |
| 1950s | 11.1 | 9.2 | 15.8 | 13.2 | 55.8 | 58.9 | 62.4 | 64.3 |
| 1960s | 20.4 | 11.9 | 21.7 | 22.1 | 47.5 | 49.6 | 52.7 | 48.2 |
| 1970s | 21.5 | 19.9 | 31.9 | 36.4 | 52.5 | 42.3 | 47.4 | 39.5 |
| 1980s | 23.7 | 25.7 | 39.4 | 29.8 | 49.5 | 40.8 | 42.7 | 35.8 |
| 1990s | 22.2 | 25.9 | 41.1 | 25.6 | 38.8 | 34.5 | 39.2 | 35.1 |
| Change | 16.4 | 22.9 | 34.7 | 18.9 | -26.2 | -28.1 | -30.8 | -35.8 |

Table 2. Changes in Life Expectancy and Healthy Life Expectancy Due to Cohort Changes in Obesity and Smoking, Women

|  | NH-White | Black | FB Hispanic | USB-Hispanic |
| :--- | :---: | :---: | :---: | :---: |
| Life Expectancy | $\mathbf{4 7 . 3}$ | $\mathbf{4 5 . 1}$ | $\mathbf{5 1 . 1}$ | $\mathbf{4 9 . 1}$ |
| 1940-1990 cohort change due to <br> increases in obesity | $-1.4 *$ | $-1.9 *$ | $-2.0 *$ | $-2.6 *$ |
| decreases in smoking <br> changes in both obesity \& smoking | $1.3 *$ | $2.0 *$ | $1.0^{*}$ | $1.3 *$ |
| Healthy Life Expectancy | -0.3 | 0.0 | -1.1 | -1.2 |
| 1940-1990 cohort change due to <br> increases in obesity | $\mathbf{3 2 . 3}$ | $\mathbf{2 8 . 9}$ | $\mathbf{3 5 . 3}$ | $\mathbf{3 3 . 6}$ |
| decreases in smoking <br> changes in both obesity \& smoking | $-1.6 *$ | $-1.9 *$ | -2.1 | -3.4 |

Table 3. Changes in Life Expectancy and Healthy Life Expectancy Due to Cohort Changes in Obesity and Smoking, Men

|  | NH-White | Black | FB Hispanic | USB-Hispanic |
| :--- | :---: | :---: | :---: | :---: |
| Life Expectancy | $\mathbf{4 3 . 3}$ | $\mathbf{4 0 . 1}$ | $\mathbf{4 6 . 3}$ | $\mathbf{4 5 . 0}$ |
| 1940-1990 cohort change due to |  |  |  |  |
| increases in obesity | $-1.7 *$ | $-2.0 *$ | $-2.2 *$ | $-1.7 *$ |
| decreases in smoking | $1.0 *$ | $2.4 *$ | 0.9 | $1.5 *$ |
| changes in both obesity \& smoking | -0.8 | -0.2 | -1.8 | -0.5 |
|  |  |  |  |  |
| Healthy Life Expectancy | $\mathbf{3 1 . 2}$ | $\mathbf{2 6 . 8}$ | $\mathbf{3 3 . 5}$ | $\mathbf{3 0 . 6}$ |
| 1940-1990 cohort change due to <br> increases in obesity | $-1.7 *$ | $-2.0 *$ | -2.2 | -1.8 |
| decreases in smoking <br> changes in both obesity \& smoking | $1.0 *$ | $2.2 *$ | 0.8 | 1.5 |
|  | -0.9 | -0.1 | -1.7 | -0.4 |


[^0]:    ${ }^{1}$ We also tested interactions between race/ethnic/nativity group and smoking and obesity, but these were not significant. We further tested the proportional hazard assumption by testing interactions between weight status and age, but these were not significant. On the basis of these tests, we opted for simpler models that assume that the

[^1]:    relative hazard of dying for each weight and smoking category is constant across all age groups ages 50 and older and is the same across race/ethnic/nativity groups.
    ${ }^{2}$ The weighted average of the $\mathrm{q}_{\mathrm{x}, \mathrm{w}}$ 's was very close but not identical to $\mathrm{q}_{\mathrm{x}, \mathrm{NCHS}}$. Therefore, we divided the $\mathrm{q}_{\mathrm{x}, \mathrm{w}}$ 's by a constant ( $q_{\mathrm{x}, \mathrm{NCHS}} / \mathrm{q}_{\mathrm{x}}$ ) to ensure that the weighted average of the adjusted $\mathrm{q}_{\mathrm{x}, \mathrm{w}}$ 's equals $\mathrm{q}_{\mathrm{x}, \mathrm{NCHS}}$.

[^2]:    ${ }^{3}$ The Sullivan method has fewer data requirements than the multi-state method and yields healthy life expectancy estimates that are nearly identical to those produced by multi-state methods except in periods of rapid change Mathers, C.D.and J.-M. Robine. 1997. "How good is Sullivan's method for monitoring changes in population health expectancies?" Journal of Epidemiology and Community Health 51(1):80-86..

