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Projecting the Effect of Changes in Smoking and Obesity on Future Life Expectancy in the United States

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Abstract We project the effects of declining smoking and increasing obesity on mortality in the United States over the period 2010-2040. Data on cohort behavioral histories are integrated into these projections. Future distributions of body mass indices are projected using transition matrices applied to the initial distribution in 2010. In addition to projections of current obesity, we project distributions of obesity when cohorts were age 25. To these distributions we apply death rates by current and age-25 obesity status observed in the National Health and Nutrition Examination Survey, 1988-2006. Projections of the effects of smoking are based on observed relations between cohort smoking patterns and cohort death rates from lung cancer. We find that both changes in smoking and in obesity are expected to have large effects on mortality. During the period 2010-2020 the effects largely offset one another because women's mortality continues to be heavily affected by their smoking histories. After 2020, the advantages of declines in smoking substantially outweigh the disadvantages of rising obesity. By 2040 we project that the combined effects of changes in obesity and smoking will raise male life expectancy at age 40 by 0.91 years and female life expectancy by 0.54 years.

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A wide variety of personal behaviors affect an individual's health. In the aggregate, these behaviors affect the health of populations. The two behaviors that have been singled out as especially damaging to the health of the US population are smoking and the combinations of diet and physical activity that produce obesity. Estimates by the Centers for Disease Control suggest that 18% of deaths in the US in 2000 were attributable to smoking and 15-17% to obesity (Mokdad et al., 2004, Mokdad et al., 2005). Uncertainty about the future of impact of these behaviors is central to uncertainty surrounding projections of future mortality (Technical Panel, 2011). According to simulations by the Office of the Actuary, the 75-year actuarial balance of the Social Security program is more sensitive to variation in future mortality rates than to any other demographic or economic parameter except real wages (Trustees 2012: Appendix D).

Like most risk factors for chronic disease, smoking and obesity have effects on health that are realized over an extended period of time rather than experienced instantaneously. Lags in the relationships between behaviors and health outcomes provide valuable information about the future of mortality, since some of the determinants of future levels may have already revealed themselves.

In this paper, we take advantage of recent observations on trends in smoking and obesity to project US mortality at ages 40+ to 2040. An earlier effort to model these effects used a two-part method of extrapolating changes in behavior and applying mortality risks associated with various behaviors (Stewart et al. 2009). Our approach adds an additional dimension of behavioral history to the projection, allowing mortality rates to be a function not only of current behaviors but also of past behaviors.

A. Background

It is well known that the prevalence of obesity has been rising in the United States while cigarette smoking has declined. Figure 1 present time trends in the prevalence of obesity and the mean number of daily cigarettes smoked per adult.

Just as the behaviors themselves have been changing, so have the mortality risks associated with them. The National Health and Nutrition Examination Surveys and the National Health Interview Surveys have been tracing surveyed individuals into the National Death Index since 1971 and 1986, respectively. Relative to earlier observations, individuals surveyed in more recent years have exhibited lower mortality risks associated with obesity (Mehta and Chang 2011) and higher risks associated with smoking (Mehta and Preston 2011). The rise in smoking risks continues a trend dating back to the 1960's that was documented in very large prospective studies conducted by the American Cancer Society (Thun and Heath 1997). These changes in relative risks have occasioned upward revisions in the estimated number of US deaths attributed to smoking (Rostron 2011) and downward revisions in the number of deaths attributed to obesity (Flegal, et al. 2007; Mehta and Chang 2009).

While obvious sources of heterogeneity in mortality risks such as age, sex, race, and educational attainment have been controlled in the studies of mortality risks, it is possible that

unobserved sources of heterogeneity have also been important contributors to the trends in relative risks. One hypothesis is that smokers are an increasingly "hard core" group that has become more adversely selected on unmeasured variables related to healthy behaviors. At the same time, the rapid inflow of people into the category of obese may have diluted the impact of especially unhealthy behaviors among those who had been obese for many years.

One concrete variable that presumably captures some of the selection effects is the length of time someone has spent in an unhealthy state. Thun et al. (1997: Appendix 17) find that, between 1959 and 1982, the mean duration of smoking among male current smokers rose modestly but among female current smokers aged 60-79 it rose by 9.5 to 11.3 years. Mehta and Preston (2011) find that these trends have continued but at a slower pace; they find a 1.0 year increase in mean duration of smoking among female smokers aged 50-74 between 1987-92 and 1997-2003, and a very small increase during this period occurring among men.

Meanwhile, declines in the duration of obesity have occurred among the obese. We compare data from NHANES 3 (1988-1994) and NHANES Continuous (2003-2004) surveys that contain three lifetime observations of body mass index (BMI), beginning at age 25 and extending to a final observation in the age interval 50-74. To estimate the number of years someone has spent obese, we use linear interpolation between the three observations and choose the conventional threshold of a BMI of 30.0 to define obesity. Among individuals aged 50-74 who were obese, the mean duration of obesity declined from 17.6 years in 1990 to 14.3 years in 2004. This reduction in the mean duration of obesity among the obese is likely to be a product of the rapid recent inflow of people into this category.

So smoking and obesity present mirror images. The prevalence of obesity has increased, the mean duration of obesity among the obese has decreased, and so has the mortality risk associated with obesity. On the other hand, the prevalence of smoking has declined, the mean duration of smoking among smokers has increased, and so has the mortality risk associated with smoking. At a minimum, these results suggest that "history matters" in translating behaviors into mortality risks. This conclusion is supported by direct evidence in prospective cohort studies that the history of obesity, in addition to baseline obesity, is an important risk factor in mortality (Preston, Mehta, and Stokes 2012; Abdullah et al. 2011). Duration of smoking is also strongly related to mortality risks among current smokers (Mehta and Preston, 2012).

B. Analytic Strategy

Our goal is to incorporate evidence on the history of smoking and obesity into mortality projections for the United States. These histories are important determinants of risk and they have, for the most part, already been observed. In the case of obesity, we project obesity distributions that contain past as well as current levels of obesity for cohorts in the future. We then apply multidimensional mortality risks that recognize the impact of past as well as current levels of obesity on mortality. These risks are derived from relatively recent experience observed in the National Health and Nutrition Examination Survey (NHANES).

In the case of smoking, we take advantage of the fact that there is a clear marker of the impact of smoking histories on mortality: death rates from lung cancer. Smoking is the overwhelming factor accounting for variation in lung cancer mortality. Among US men aged 30 and older in 2005, it is estimated that 90% of lung cancer deaths are attributable to smoking; for females, the figure is 84-85% (Oza, et al. 2011). Consistent with a major role for behavioral histories, death rates from lung cancer are organized on a cohort basis in the United States. This feature permits the identification of "cohort effects" that can be projected into the future as cohorts age. The final step in our analysis is translating projected death rates from lung cancer into all-cause mortality rates, using statistical relations that have been developed between smoking's impact on lung cancer and its impact on all-cause mortality.

In the case of both smoking and obesity, our principal goal is to project the proportionate effect of changes in these behaviors on age-specific death rates. Our comparison schedule is simply the death rates at baseline, which reflect the behavioral histories that have been accumulated at that point. We are not attempting to project all determinants of mortality into the future; instead, we attempt to isolate the impact of behavioral changes. If there are other sources of future change in mortality, we are assuming that the effects of behavioral change will be independent of them.

We convert the projected sets of proportionate changes in age-specific death rates into estimates of their effect on a summary measure, life expectancy at age 40, e(40). Appendix D describes the procedures used to translate our estimates of age-specific death rate changes into their effects on life expectancy.

C. Projecting the Effects of Changes in Obesity

We project the effects of past and future changes in obesity in three stages. First, we project obesity distributions from 2010 to 2040 using an observed BMI-transition matrix for 1998-2008. We present evidence that BMI transition matrices have been nearly constant for the past 18 years and we maintain the assumption of constancy into the future. Even though the transition matrix is assumed to be constant, large increases in obesity are projected. Second, we project the distribution of age-25 BMI from 2010 to 2040. That is, for each age group through 2040, we estimate the distribution of BMI when the cohort occupying that age group was aged 25. Finally, we apply death rates drawn from National Health and Nutrition Examination Surveys to the distribution of current BMI and BMI at age 25.

C.1) Forecasting the future distribution of obesity.

Previous forecasts of obesity can be classified into three categories. The most common procedure has involved extrapolation of past trends in the prevalence of obesity, most often by using a linear model (Wang et al. 2008; Ruhm 2007; Stewart et al. 2009). Linear models do not recognize that the proportion of the population at risk of becoming obese declines as the

proportion obese rises. A recent analysis using linear extrapolation reached the implausible conclusion that all Americans would become obese by the year 2048 (Wang et al. 2008).

A second approach involves forecasting future BMI levels on the basis of a predictive regression model and extrapolations of covariate series (Finkelstein et al. 2012). A wide array of factors associated with obesity have been identified as important influences on caloric imbalance: employment, physical activity at work, food prices, the prevalence of restaurants, cigarette smoking, cigarette prices and taxes, food stamp receipt, and urbanization (Baum and Chou 2011 provide a good review). The only study that has used such an approach to projection built a predictive model using state-level covariates believed to be associated with or causative of obesity. Covariates included alcohol and gas prices, the price of healthy relative to unhealthy foods, the unemployment rate and state indicator variables. The main limitations of this approach are that selection of covariates can be arbitrary and projection of covariate series often involves as much uncertainty as projecting obesity directly. Additionally, the R^2 value of the predictive regression model was 0.06 (Finkelstein et al. 2012), which raises concerns regarding the extent to which the model captured the relevant dynamics.

We adopt a third approach, Markov modeling (Basu 2010). Markov models simulate flows of individuals through mutually exclusive states. Individuals are arrayed by BMI at baseline (time 1) and are subjected to a set of probabilities of being found in various BMI states at some future date (time 2), dependent on what state they were in at time 1. A set of transition probabilities can also be applied to project the distribution from time 2 to time 3. Relative to extrapolation, this approach does not require specification of a functional form. A second advantage is that such models are able to recognize what is empirically observable, that an individual's BMI level at time 2 depends upon his or her BMI level at time 1. If those functions were changing wildly from period to period, they would provide an unstable basis for projection. However, we will show that the 10-year pattern of weight transitions has become relatively constant, and we project that constancy into the future.

To develop transition probabilities, we use data from the National Health and Nutrition Examination Survey III (1988-1994) and continuous waves 1999-2002 and 2007-2008. NHANES is a series of nationally representative surveys of the non-institutionalized U.S. population conducted by the National Center for Health Statistics. The survey includes an examination component in which extensive medical data, including data on height and weight, are collected by trained nurses in mobile clinics or in home-visits. An additional feature of certain of the NHANES surveys is that respondents are asked to recall their weight 10-years prior to the survey.[1] We combined data on recall weight with current height to estimate 'recall BMI' in each period. To reduce bias that may result from recall of past weight, we applied an individual-level correction factor based on the proportionate error between measured and self-reported BMI at baseline (Flegal 1995).[2] We used data on measured height and weight at the

[1] These data are available for ages 35 and above, thus we are limited to modeling transitions beginning at age 25.
[2] This is, in effect, a correction for errors of self-report. Recall weight data may be subject to both errors of self-reporting and recall bias. In a sensitivity analysis, we tested a simultaneous correction for both sources of error developed from NHANES 1 and NHEFS 1982-1983 data. However, in the validation step, this correction did not

time of the survey to calculate 'current BMI'. The combination of corrected recall weight (hereafter referred to as recall weight) and current BMI served as the data inputs for estimating 10-year transition probabilities. We use four BMI categories: Normal (BMI < 25.0), Overweight (25.0 to 29.9), Obese I (30.0 to 34.9) and Obese II-III (35.0+).

We used ordered logistic regressions models to estimate transitions across categories of BMI by age and sex in each of three decadal periods between 1980 and 2010. Independent variables, which pertain to 10 years prior to the survey, included age, the square of age, sex, BMI indicator variables (overweight, obese class 1, obese class 2) and interaction terms between each of the BMI indicator variables, age, the square of age and sex. We subsequently used the model parameters to generate a series of predictions for each decadal period. For purposes of comparing transition probabilities across time periods, we fixed demographic covariates at the average values observed in the most recent period. We used bootstrapping to estimate uncertainty intervals for the transition probabilities and differences in transition probabilities between periods.

A key question is how well these transition matrices predict changes in obesity that have actually occurred. Appendix A is addressed to this issue. We show there that the retrospective reports on weight 10 years earlier, when combined with current weight to produce a transition matrix, are highly effective in projecting the cross-sectional BMI distributions from time t to time t+10. We repeat this demonstration for the periods 1980-90, 1990-2000, and 2000-2010 (the latter using the transition matrix from 1997/8 to 2007/8, the latest available).

The actual transition matrices, denoted by M, are shown in Table 1 A for these three periods. Table 1 B shows the changes in transition probabilities in each cell between the two successive matrices. The changes from $M_{80\text{-}90}$to $M_{90\text{-}00}$ are large and systematic. Relative to the earlier period, the probability of moving up in weight class was significantly higher during 1990-2000 than in 1980-90. The probability of moving to a higher weight class increased significantly for all weight classes except the highest, where no such movement was possible. But the highest weight class also contributed to the upsurge in obesity by virtue of a significant increase in the probability of remaining there if one started there.

In contrast, the changes between $M_{90\text{-}00}$ and $M_{98\text{-}08}$ were small. Only 1 of the 16 cells in the transition matrix showed a significant change in transition probabilities. On the basis of the relative stabilization of the BMI transition matrix over the past two decades, we have elected to assume that the matrix is constant at the rates represented by $M_{98\text{-}08}$ over the three decades starting in 2010. The implication of this assumption is that the multitude of processes that produce weight change would operate with the same intensity in the future as they did during 1998-2008.

We begin with initial population counts in the 2010 continuous NHANES and cross-classified into 96 categories according to sex, five-year age group (25-29 to 80-84) and measured BMI category (normal, overweight, obese class I and obese class II). Sample weights were

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perform as well as the one used above. In particular it didn't perform well in the most recent period, which may be because the pattern of error has changed.

incorporated so that counts are representative of the US population in that period. In each round of the simulation, the first step was to survive members of the population forward 10 years using age, sex, and BMI-specific life tables drawn from pooled NHANES III and NHANES Continuous 1999-2004 cohorts linked to deaths in the National Death Index through 2006. A discrete hazards model was employed to generate the underlying risks.

In the second step, sex, age and BMI-specific transition probabilities are applied to surviving members of the population. Each iteration of the projection produced new population counts which serve as the initial counts for the next iteration of the projection. A new cohort of 25-34 year olds is assumed to enter the population each decade. The distribution of BMI in these cohorts is predicted through linear extrapolation of the historical trend.[3] Uncertainty intervals around the forecasts of future BMI levels were obtained using a bootstrapping procedure designed to capture uncertainty in both the estimated transition probabilities and the life table parameters.

Figure 2 presents the results of these projections for men and women. By 2040, 49% of men and 57% of women are projected to be obese. Alarmingly, the morbidly obese (BMI>35.0) increase as a proportion of the obese, to the point where they constitute a majority of obese women by 2020 and thereafter.

C.2) Forecasting the future distribution of Age-25 BMI

We show below that death rates are functions not only of current levels of BMI but also of BMI at age 25. Data on *measured* BMI are available in NHANES from 1960 to 2010. We take advantage of that information to project age-25 BMI levels for all cohorts who will be aged 25-84 at any time between 2010 and 2040. Our age-25 series is interpolated/extrapolated in five-year intervals between 1952.5 and 2037.5 using the parameters from a linear spline regression with a knot at 1980 to reflect the rapid rise in obesity after that year. We then survive the age-25 distribution forward to attained ages in 2010-40 by a life table based on a discrete hazard model applied to pooled NHANES III (1988-1994) and NHANES continuous 1999-2004 cohorts linked to deaths in the National Death Index through 2006. The only variables in the hazard model other than age-25 BMI (normal, overweight, and obese) are attained age (measured in single years) and sex. An individual-level correction of self-reported age-25 weight is applied when estimating the relation between mortality and age-25 BMI. Details may be found in Appendix B.

C.3) Estimating death rates by category of BMI

Data for the mortality analysis are derived by pooling the National Health and Nutrition Examination Survey (NHANES) 3 (1988-1994) and NHANES 1999-2004 continuous waves.

[3] We estimate a historical series for ages 25-34 using measured data on height and weight from NHANES 2, NHANES 3 (phase I and II) and NHANES continuous waves 1999-2010. We regress the proportion in each BMI category on time and an indicator for sex and use the parameters of the model to predict the proportions in each category of BMI for each sex in 2020, 2030 and 2040.

NHANES 3 and NHANES 1999-2004 are independent cross-sectional surveys. Information on measured body weight is available at baseline and on self-reported body weight at age 25. BMI at baseline is calculated by combining weight and height measured at baseline. BMI at age 25 is calculated by combining self-reported weight at age 25 with measured height at baseline for individuals less than 50 years of age and by combining self-reported weight at age 25 with self-reported height at age 25 for individuals 50 years of age and above. We applied an individual-specific correction factor to reported age 25 BMI to account for potential errors in reporting. The correction factor was calculated as the proportionate error in BMI based on self-reported weight/height at baseline for each individual: $(BMI^{M}-BMI^{SR})/BMI^{M}$, where *M* indicates measured and *SR* indicates self-reported. A discrete hazard model is used on a person-month file. Respondents enter the risk set two years after they are surveyed, a delay designed to reduce potential confounding from illness-related weight loss. Information on deaths is available through December 31, 2006. There were 2976 deaths of 21,554 respondents.

Our BMI categories at age 25 are overweight (BMI 25.0 to <30.0), obese (≥30.0), and other (<25.0). At baseline, the categories are obese I (30.0 to <35.0), obese II/III (≥35.0), and other (<30.0). The main model includes corrected age-25 and baseline BMI categories, age attained over follow-up (years), sex, race/ethnicity (white, black, Hispanic, other), educational attainment (< high school, high school degree/GED, some college, college graduate), and smoking status at baseline (current, former, never). We include interaction terms between linear attained age (measured from age 35) and the two baseline obese categories because of evidence that the relative risk of death among obese individuals declines with age (Prospective Studies Collaboration 2009). Models are estimated on a sample of attained ages 40-84. Various sensitivity analyses are conducted with models that alternatively exclude covariates except for age and sex, use uncorrected age 25 BMI, and exclude age 25 BMI. NCHS-supplied survey weights and design elements (strata and primary sampling units) are used.

Coefficients from the main model and sensitivity analysis models are shown in Appendix Tables C1-C4. Weight at age 25 as well as baseline weight categories are related to the risk of death in the expected direction. Three of the four weight variables are significant at 5%. Age interactions with baseline obesity are negative, as expected, and are retained in predictions.

C.4) Results

The combined effect of the projected increases in obesity, including age-25 obesity, and the estimated mortality risks for the obese are shown in Table 2 and Figure 3. For both males and females, the effect of changes in BMI is expected to increase over time. By 2040, the changes are expected to raise death rates in the age range of 40-84 by 1%-17% for males and by 1%-25% for females. Projected effects on the two sexes are relatively similar. When converted into estimates of the effects on life expectancy at age 40, Table 5 shows that the decline in life expectancy (mean, males and females) is 0.21 years by 2020, 0.43 years by 2030, and 0.71 years by 2030.

These estimated effects are smaller than those estimated by Stewart et al. (2009), who project a loss of 1.02 years in life expectancy between 2005 and 2020 as a result of increases in obesity. There are probably several reasons for this disparity. First, their linear extrapolation of BMI proportions produced a somewhat faster increase in obesity than our use of BMI transition matrices. Second, Stewart et al. used NHANES mortality rates by obesity status for the period beginning in 1971, whereas our mortality rates are derived from a period beginning in 1988. As noted earlier, the mortality risks associated with obesity have declined in NHANES. Third, Stewart et al. did not include an age/BMI interaction term in their mortality analysis. The mortality risks of obesity decline with age in such a way as to reduce the risks at the ages where deaths are most heavily concentrated (Prospective Studies Collaboration 2009). On the other hand, unlike Stewart et al. we have introduced historical data on BMI at age 25. We show below that our estimated effects of obesity increases on life expectancy would have been lower had we not incorporated this information.

D. Projecting the Effects of Changes in Smoking

The risk of death from smoking is clearly a function of a multitude of smoking-related behaviors, including the number of cigarettes smoked per day, the degree of inhalation, the filtration and tar content of the cigarette, and how each of these (and other) components of a smoking profile have developed over a lifetime. Historical information is critically important because of a long lag between smoking behavior and its effects on mortality. Partly because of changes in smoking histories among current smokers, the relative risk of death among smokers has risen sharply. A single cross-sectional indicator of smoking prevalence cannot effectively capture these many dimensions. Prevalence-based estimates of smoking risks are also affected by imprecise classification of smoking status among participants. For example, the largest prospective study of smoking, the Cancer Prevention Study II (CPS II) included among "lifetime non-smokers" persons who had smoked but who had not reported themselves as smoking daily for at least a year (Lestikow, 2008).

Fortunately, there is another indicator of the health effects of smoking that comprehends the many dimensions of smoking: the death rate from lung cancer. As noted above, smoking is the overwhelming risk factor in death from lung cancer, with 90% of male and 84-85% of female lung cancer deaths in the US attributable to smoking (Oza et al. 2011). Figure 4 shows how the rise and fall of smoking in the US has been followed after a lag of 20-30 years by a rise and fall in lung cancer mortality. Because of the cumulative and delayed impact of smoking on lung cancer mortality, lung cancer exhibits prominent "cohort effects"; rates of death from lung cancer are systematically arrayed by birth cohort rather than by period. Figure 5 shows male death rates from lung cancer in the United States in various birth cohorts. Clearly, there is a near parallelism among these rates on a log scale, implying that the sequence of death rates for one cohort is nearly a constant multiple of the death rates for another cohort. Figure 6 shows that this parallelism is missing when data are arrayed by period rather than by cohort. Similar cohort

effects attributable to cohort smoking patterns have also been observed in Europe and Japan (Janssen and Kunst 2005; Yamaguchi et al. 2000).

Although lung cancer mortality is an excellent indicator of the health effects of smoking, lung cancer does not account for a majority of deaths attributable to smoking. Cardiovascular diseases and chronic obstructive pulmonary diseases (COPD, which includes bronchitis and emphysema) also make large contributions. Two methods have been developed to connect smoking-related mortality from lung cancer to smoking-related mortality from other causes of death. Peto et al. (1992) translate observed lung cancer death rates into an estimate of smoking "prevalence" by referring to the difference between lung cancer death rates for smokers and non-smokers in CPS-II. This estimate of smoking prevalence is then used to estimate the risk attributable to smoking for other smoking-related causes of death by employing the cause-specific relative risks for smokers versus non-smokers from CPS-II.

The second method also uses lung cancer mortality as the basic indicator of the damage caused by smoking (Preston, Glei, and Wilmoth 2011). However, rather than relying on the relative risks from CPS-II or any other study, it estimates the macro-level statistical association between lung cancer mortality and mortality from all other causes of death in a dataset of 21 countries covering the period 1950 to 2006. In addition to lung cancer mortality, the statistical model includes age, sex, period, and country effects as well as interactions among them. This approach is motivated by the expectation that lung cancer mortality is a reliable indicator of the damage from smoking and that such damage has left a sufficiently vivid imprint on other causes of death that it is identifiable in country-level data. The strong statistical relations that emerge are consistent with that expectation.

The two methods of translating lung cancer mortality into all-cause mortality give very similar results. The proportion of deaths attributable to smoking that are estimated by the two methods is correlated at 0.96 for males and 0.94 for females across 20 countries in 2000 (Preston, Glei, and Wilmoth 2011) . Both methods implicitly assume that the pattern of lags between smoking and lung cancer death is similar to that between smoking and other causes of death. Is that assumption reasonable? Preston, Glei, and Wilmoth (2011) experimented with various lags between lung cancer mortality and mortality from other causes of death and found that a model in which the two death rates were contemporaneous (i.e., exhibited no lags) worked best (unpublished result). Oza et al. (2011) examined time-patterns of relative mortality risks of smokers from various causes of death. Relative to the lag between smoking behavior and death for lung cancer, they found the lag structure to be longer for COPD and shorter for cardiovascular diseases. Using the Peto et al. (1992) approach, the estimated number of deaths attributable to smoking differed by only 1.7% when lag structures were incorporated compared to when they weren't. Thus, it appears that the pattern of lung cancer lags is sufficiently similar to that for the aggregate of other causes of death that serious distortions do not arise from assuming that they are the same.

Our projections of the mortality effects of smoking are based on the identification of cohort effects in lung cancer mortality. Mortality levels that are unique to cohorts are obviously a

convenient vehicle for projecting mortality because cohorts age with completely predictable regularity.

D.1) Data for analysis of smoking effects

Data on lung cancer deaths by age, sex, and period are drawn from the National Center for Health Statistics and the World Health Organization/International Agency for Research on Cancer. Estimates of population size and counts of deaths from all causes combined are taken from the Human Mortality Database. Estimates pertaining to birth cohorts are created by organizing a data matrix in 5-year age groups and 5-year time blocs, each starting with 0 or 5, and then assembling data along the resulting diagonals. Our earliest observations are for the period 1935-39, reflecting the fact that the US death registration area was not completed until 1933. The mortality data series extends to 2005-09.

Data on smoking by cohort are based on a detailed reconstruction of smoking histories by Burns et al. (1998). They employed a total of 15 National Health Interview Surveys (NHIS) conducted between 1965 and 1991 to estimate cohort smoking histories. David Burns supplied us with unpublished estimates using the same methodology that incorporated data from three additional National Health Interview Surveys through 2001. We updated the series through NHIS data for 2009. We converted these data into an estimate of the average number of years spent as a current smoker before age 40. This value is derived by summing across ages between 0 and 39 the proportion of cohort members who were estimated to be current cigarette smokers.

For cohorts that have not reached age 40 in 2010, we estimate the future cumulative years of smoking by age 40 based on observed cumulative years smoked at earlier ages. We use regressions predicting the mean cumulative years of smoking by age 40 with independent variables representing cumulative smoking indexes by age 35, by age 30, by age 25 and by age 20. We add a sex indicator and a trend variable to the regressions. Regressions are estimated on data for the 16 cohorts for which we have complete data up to age 40. The regressions in all cases explain at least 97% of the variance in cumulative years of smoking before age 40. For the two cohorts born after 1990, we fix the variable at its level estimated for the 1985-90 cohort. The resulting series are presented in Figure 7.

D.2) Methods for projecting the mortality effects of smoking

Our goal is to identify how lung cancer mortality varied from cohort to cohort so that we can project these cohort effects into the future. We try two principal ways to estimate cohort effects. One is to use a cohort smoking variable that had proven useful in prior research to relate smoking to all-cause mortality (Preston and Wang 2006, Wang and Preston 2009). As noted, that variable is the mean cumulative number of years that a member of a cohort had smoked prior to age 40, designated $S(a,t)$ for the cohort born a years earlier at time t. For each sex, we estimate an equation of the form

$$ln\, M(a,c) = A + B_a X_a + B_s\, ln\, S(a,c), \qquad (1)$$

where $M(a,c)$ is the death rate at age a in cohort c, X_a is an indicator of age category a, B_a is the coefficient of age category X_a and B_s is the coefficient of $ln\, S(a,c)$. We estimate this model using negative binomial regression on death counts on all observations at ages 40-44 to 80-84 for cohorts born between 1915-19 and 1975-79. This combination was chosen because it produced the closest fit to actual death rates in 2010 among the data restrictions that we imposed. The coefficients of ln $S(a,t)$ are very similar at 1.332 for males and 1.318 for females. Age coefficients are monotonically and smoothly rising at a diminishing rate for both sexes.

The second approach is to estimate "cohort effects" as coefficients of dummy variables pertaining to various cohorts, without any reference to smoking histories.[4] We estimate the parameters of a straightforward age/cohort model,

$$\ln M(a,c) = A + \Sigma\, B_a X_a + \Sigma\, B_c X_c, \qquad (2)$$

where $M(a,c)$ is the death rate in cohort c at age a, B_a and B_c are the coefficients of age category a and cohort c and X_a and X_c are indicators of age and cohort membership.

We estimated this model using negative binomial regression on death counts. Figures 8.A and 8.B plot estimated cohort effects from equation (2) and the mean number of years of smoking before age 40 for each cohort. The two series for women obviously track one another closely for both sexes, including an upward bump for female cohorts born 1955-64. For men, both series are an inverted-U shape, although the peak of the smoking series occurs earlier than the peak cohort coefficient.

Our projections are based on equation (1), which uses the smoking series. One advantage of this approach is that we are able to observe smoking behavior for cohorts as young as age 15-19, thus providing substantially more information for the projection. The cohort coefficients are not robustly estimated until age 40-44, when substantial numbers begin to die from lung cancer. Furthermore, the smoking-based analysis produces predicted death rates in 2010 that are much closer to the actual death rates in that year than the analysis using cohort coefficients, which underestimate mortality badly for older cohorts.

To translate projected lung cancer death rates into death rates from all causes, we use the set of translation factors by age and sex drawn from Preston, Glei, and Wilmoth (2011). These are based on the relation between lung cancer mortality and mortality from other causes of death

[4] Such an estimate could be made using an age/period/cohort model, but it is widely recognized that introducing age, cohort, and period variables into the same model creates an identification problem because of the perfect linear association between any two of these variables and the third (Feinberg and Mason 1978). Our efforts to introduce period measures into an age/cohort model were unsuccessful in the sense that they resulted in implausible cohort and period effects, presumably because of these colinearity issues. In addition, we had no strong hypothesis about period effects on lung cancer mortality, since we considered such mortality to be primarily a function of cohort smoking histories.

observed in a dataset from 21 countries between 1950 and 2006, representing some 10 billion person-years of exposure and 600 million deaths.[5] Below, we explore the sensitivity of results to this choice.

D.3) Results

Table 3 and Figure 9 present the results of this analysis. Looking across the rows in Table 3 shows that male age-specific death rates are expected to decline at every age throughout the projection period. The heaviest smoking male cohorts are already age 80+ in 2010 and the impact of changes in cohort smoking patterns is to produce a steady decline in relative death rates as age and time advance. However, female rates are expected to rise in a number of age intervals during the early years as heavy-smoking cohorts replace lighter-smoking ones (cf. Figure 7). Not until 2035 is projected mortality for women lower at all ages than it was in 2010. By 2040, female declines in age-specific mortality range from 5 to 21% while male declines are in the range of 2-18%.

Table 5 converts the age-specific projections into estimates of the effect on life expectancy at age 40. Males show a relatively steady improvement in life expectancy over time and a total gain of 1.62 years by 2040. In contrast, female life expectancy is expected to fall from changing smoking patterns between 2010 and 2015 as the heaviest smoking cohorts move into the prime ages of dying. There is projected to be virtually no gain in female gains in life expectancy as a result of smoking reductions between 2010 and 2020. However, female gains accelerate sharply after 2025 as the heaviest smoking cohorts disappear. By 2040, women are projected to have gained 1.14 years in e(40) from smoking reductions.

There are two other projections of anticipated changes in mortality as a result of changing smoking patterns. Wang and Preston (2009) add a cohort smoking term to the conventional Lee-Carter model of mortality change from all causes of death combined. They summarize their results in the form not of life expectancy but rather of the probability of surviving from age 50 to age 85. For the projection period 2009-2034, they estimate that reductions in smoking will increase the probability of male survival by 15.8% and of female survival by 7.2%. Changes in this probability between 2010 and 2035 in the present set of projections are 14.2% for males and 6.4% for females. Because mortality from lung cancer is a more sensitive indicator of the damage from smoking than is all-cause mortality, we believe the present estimates are more reliable. Fortunately, they estimates are in close agreement.

Stewart et al. (2009) also project the effects of changes in smoking on future life expectancy by extrapolating trends in smoking distributions and applying death rates by smoking status from NHANES. They do not differentiate between the sexes. They estimate that, in a 15-

[5] Preston, Glei, and Wilmoth do not estimate coefficients for ages below 50. We assume that the coefficients for ages 50-54 apply to ages 40-49. Since coefficients decline with age, this choice probably produces an underestimate of smoking-attributable deaths, but there are very few deaths in the age interval 40-49, so results are scarcely affected by this assumption.

year projection period beginning in 2005, declines in smoking will produce a 0.31 year gain in life expectancy at age 18. In our 15-year projection beginning in 2010, we estimate that declines in smoking will raise life expectancy at age 40 by 0.86 years for males and 0.24 years for females, with an average gain of 0.55 years.[6] While our results appear to show a faster improvement than theirs, the rate of improvement accelerates through the period. In our 10-year projection ending in 2020, the same year that the Stewart et al. projections end, our gain in life expectancy (mean, males and females) is 0.33 years compared to their 0.31 years over the preceding 15-year period. Thus, the disparity between our combined-sex smoking projections and theirs is relatively small over their shorter projection period.

E. Combining obesity and smoking

Our research design does not allow us to investigate interactions between smoking and obesity. Are the effects of changes in smoking and obesity likely to be additive and independent, as we have assumed, or might there be important interactions between them? Two types of interactions are relevant. One refers to correlations in behavior between smoking and obesity. To take the most obvious example, if smoking reduces the likelihood of being obese, then declines in smoking should be reflected in increases in the prevalence of obesity. Flegal (1995) has estimated that 20% of the increase in adult obesity between 1980 and 1990 is a result of smoking cessation during that period. Using data on two cohorts from the National Longitudinal Study of Youth, Baum and Chou (2011) estimated that only 2% of the increase in obesity among young adults over a recent 20-year period was attributable to declines in smoking. These are not large effects.

The second type of interaction is interaction between the risks themselves. If the mortality risk from obesity is lower among smokers, as is sometimes claimed (Allison et al. 2001), then the projected reduction in smoking should raise the risk of death associated with obesity. However, the largest meta analysis of cohort studies of obesity concluded: "Throughout the range 25–50 kg/m², the effects of BMI and smoking seemed to be roughly additive, rather than multiplicative, both for vascular mortality and for all-cause mortality" (Prospective Studies Collaboration 2009: 1090).

Assuming independence between the mortality risks of obesity and smoking, we multiply the effects of changes in obesity and smoking presented in Tables 2 and 3. Results are shown in Table 4 and Figure 10. The preponderant downward slope of both sets of results when smoking and obesity are considered independently is accentuated when the effects are multiplied together.

Table 5 summarizes these changes in the form of life expectancy at age 40. The combined effect of changes in smoking and obesity is expected to be relatively small between 2010 and 2020 because women actually lose life expectancy from the combined changes. Thus, the pattern of reductions in the female life expectancy advantage that has been evident in national

[6] Changes in life expectancy at ages 18 and 40 are highly comparable because so few years of life are lost between these ages.

data since 1980 is expected to continue for another decade, at least from these sources. Beyond 2020, the advantages of smoking reductions are expected to outweigh the disadvantages of increases in obesity for both sexes. By 2040, life expectancy is anticipated to be 0.91 years higher for males and 0.54 years higher for females because of these combined behavioral changes.

F. Sensitivity Analyses

We performed four analyses of the effect of changes in procedures on outcomes. In each case, we estimated the effect of an alternative procedure on age-specific death rates and converted those rates into estimated effects on life expectancy at age 40. Results with respect to life expectancy at age 40 are shown in Table 6. The values in that Table are the difference between the life expectancy value produced by the alternative procedure and that produced by our main procedures. A positive value means that the alternative procedure resulted in a gain in projected life expectancy relative to the main procedure. When the alternative procedure relates to obesity, the comparison is made with the main obesity results; smoking results are compared to smoking results.

Three of the four sensitivity analyses are made with respect to obesity. To show the impact of controlling educational attainment, race/ethnicity, and smoking on our results, we have repeated the mortality analysis for obesity without these controls, using the regression equation presented in Table C2. Table 6 shows that results are insensitive to this change in procedure: the effect on e(40) never reaches 0.04 years.

Our main procedure uses *measured* BMI at age 25, or estimates thereof, in combination with a mortality regression that attempts to correct at the individual level for errors in self-reported BMI at age 25. This procedure includes surviving the age-25 BMI distribution forward to attained age in 2010-40 using mortality rates based on corrected age-25 BMI. The alternative procedure is simpler because it involves no corrections: it estimates age-25 BMI distributions for individuals aged 25-84 in 2005 based on age-25 recall data available in NHANES 2003-2006. A mortality model was estimated based on uncorrected self-reported BMI at age 25 (see Appendix B for more information about the model's implementation and for parameters of the model). Sex and BMI-specific life tables developed from the model were applied to the baseline distribution of self-reported BMI at age 25. (In this alternative approach, it is not necessary to survive individuals from age 25 to baseline since all had survived). Table 6 shows that results were not sensitive to whether estimates were based on measured or self-reported data on age-25 BMI. No difference between the procedures in any projection period was as large as 0.05 years.

The third sensitivity analysis involving obesity examines the effect of omitting information about age-25 BMI. In this case, the regression equation predicting mortality based on BMI does not include terms representing BMI at age 25 (see Appendix C). Table 6 shows that including age-25 information has an important effect on results. By 2040, losses in life expectancy are 0.29 years greater for men and 0.20 years greater for women when age-25 BMI is

included than when it is not. These represent increases in impact of 67% for men and 41% for women relative to the estimated impacts when age-25 BMI is omitted. We believe that these results justify the effort to include life history information in the analysis.

The sensitivity analysis involving smoking uses an alternative set of relations between lung cancer mortality and all-cause mortality. The main results presented above are based on relations estimated across 21 countries from 1950 to 2006. Fenelon and Preston (2012) instead estimate coefficients relating lung cancer to all-cause mortality that are based on variations across 50 states of the US between 1996 and 2004. Coefficients that predict mortality from other causes of death on the basis of lung cancer mortality are very similar for men but lower for women at younger ages.[7]

Results in Table 6 show that the sensitivity of results is minor for the first 10 years of projection, modest for the second 10 years, and sizeable by 2040. Of the projected 1.62 years of gain in life expectancy from reductions in smoking by 2040 for males in Table 5, 0.33 years is eliminated if the alternative relations are used. Of the 1.14 year gain for women, 0.47 years is eliminated if the alternative relations are used. The alternative results have the virtue of being based on contemporary relations in the US, but the main results are based on many more data points. We believe that the comparison of the two approaches provides a realistic picture of the degree of uncertainty in the smoking results; they are clearly less robust than the obesity results. However, using either the main approach or the alternative, declines in smoking are expected to produce substantial gains in life expectancy over the next three decades.

G. Conclusion

The combined effects of past and future changes in obesity and smoking are likely to result in an improvement in US life expectancy over the next 30 years. This improvement occurs because the advantages of reductions in smoking outweigh the penalty imposed by increases in obesity. Over the next decade, however, the combined effects are likely to produce only a very small improvement in mortality for the combined sexes because the heaviest smoking cohorts of American women are still in or approaching the ages of greatest vulnerability to death.

Our results differ from those of Stewart et al. (2009), who projected that the negative survival effects of obesity would exceed the advantages of reduced smoking over the period 2005-2020. Some of the apparent difference in results is a product of the different periods of analysis. As noted, we find relatively small net effects of the two forces between 2010 and 2020; more precisely, sizable gains in life expectancy are projected for men and with small losses for women. It is only in the years beyond 2020 when the advantages of reduced smoking among women start to be fully realized that smoking gains strongly outpace obesity losses. On the other hand, some of the differences between our results and those of Stewart et al. reflect a much

[7] Neither approach estimated a coefficient for ages 85+. Preston, Glei, and Wilmoth (2011), the source of the main analysis, used the mean coefficient at ages 70-74, 75-79, and 80-84 to apply to ages 85+. We make this same assumption for the alternative method based on Fenelon and Preston (2012).

smaller role for obesity in the present estimates. The reduced role probably results primarily from our projection of a slower increase in obesity and our use of lower mortality risks associated with the condition. Our smoking results appear to be in reasonable accord with those of Stewart et al. over their shorter projection period, and with those of Wang and Preston (2009) over a longer period.

Are the changes that we have projected large or small? One useful metric is provided by projections made by the Social Security Administration (Bell and Miller 2005). They anticipate that life expectancy at age 40 will grow between 2010 and 2040 by 2.55 years for men and 2.17 years for women. Against this backdrop, the gains that we estimate from reduced smoking alone (1.62 years for men and 1.14 years for women in Table 5) would themselves account for over half of the gains in life expectancy. These gains will be offset by the consequences of increases in obesity. As a percentage of the life expectancy increases projected by the Social Security Administration, growing obesity imposes a penalty of 29% for men and 31% for women. These two behaviors clearly exert a major influence on American mortality and warrant continued monitoring.

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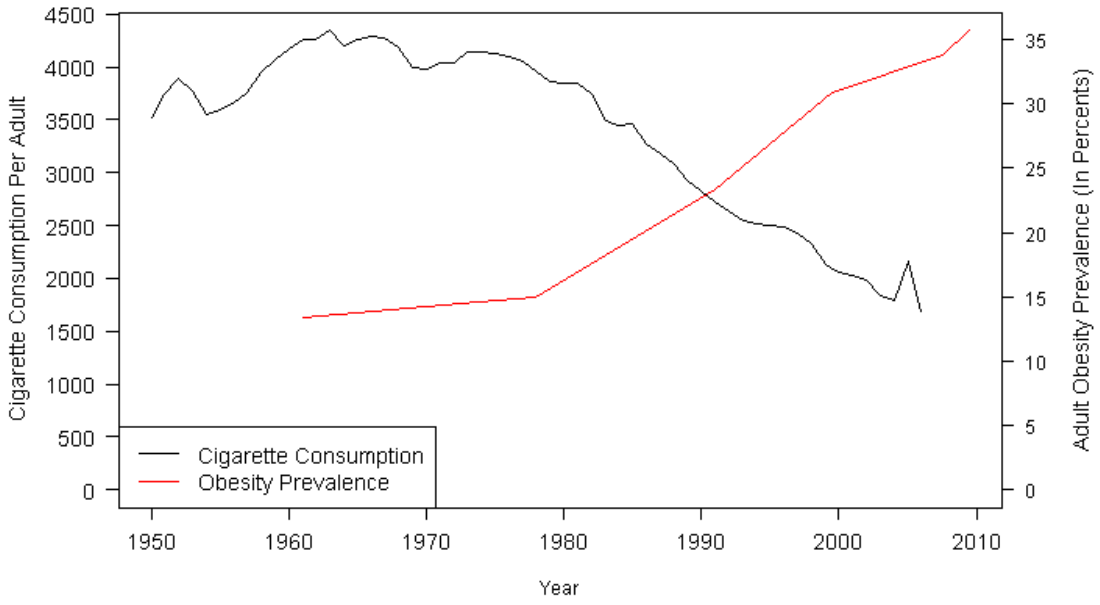
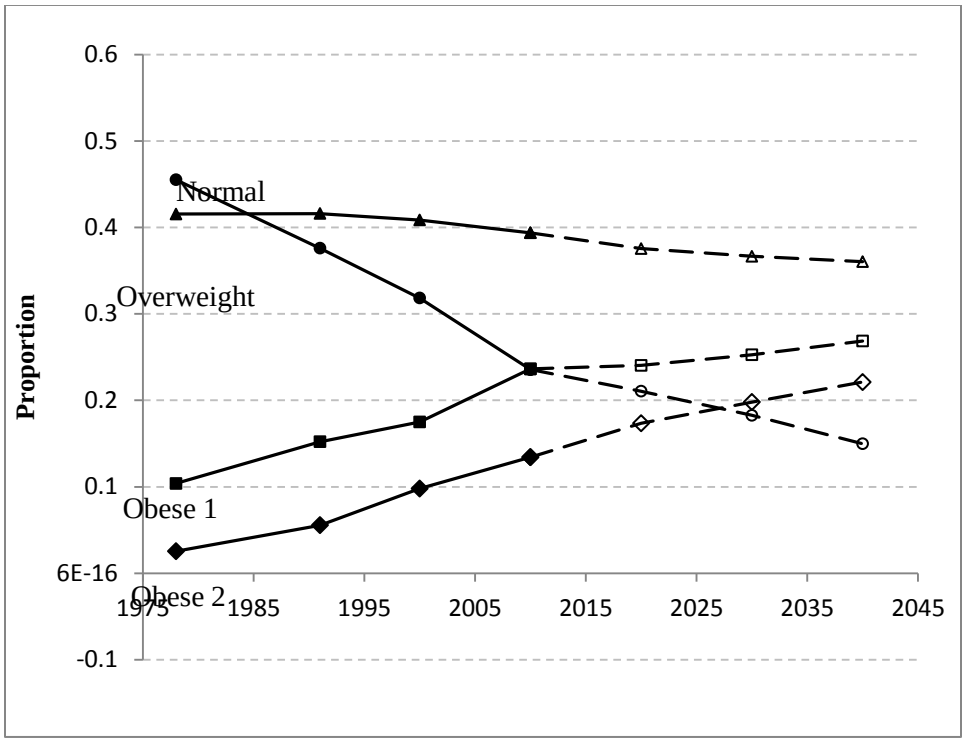
Figure 1. Trends in Smoking and Obesity in the United States



Sources: Cigarette consumption data per adult per year are extracted from Tobacco Situation and Outlook Report Yearbook. U.S. Department of Agriculture, October 2007. Obesity data based on measured body mass index in NHANES from 1960 to 2010.

Figure 2. Actual and Projected Trends in Body Mass Index

A. Males



B. Females

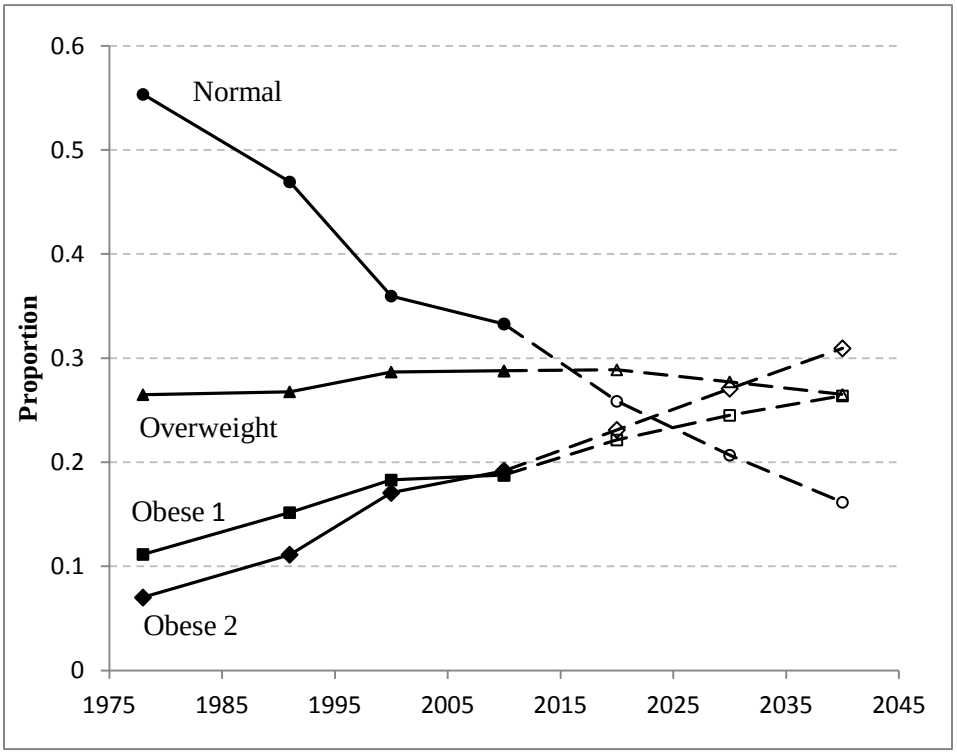
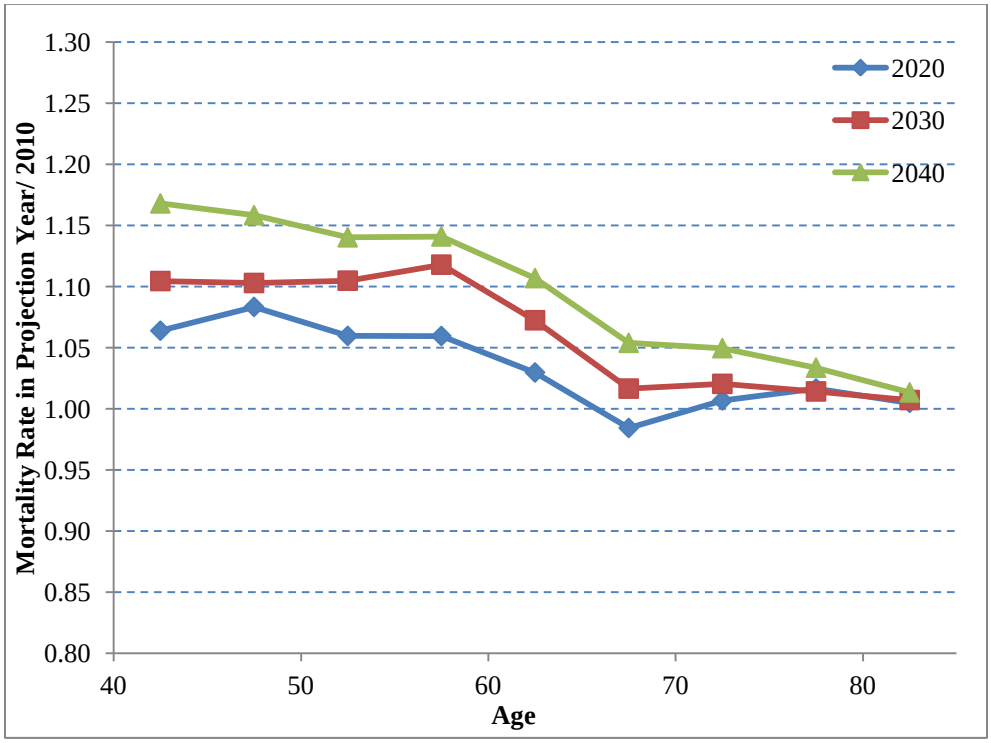


Figure 3. Effect of Projected Trends in Body Mass Index on Age-Specific Death Rates

A. Males



B. Females

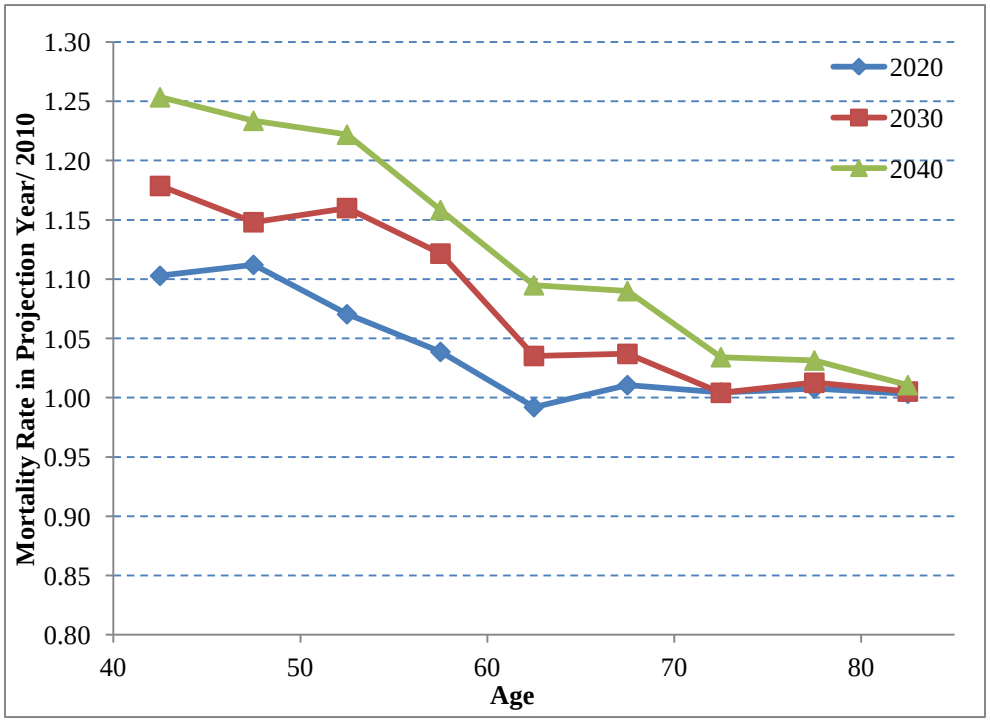
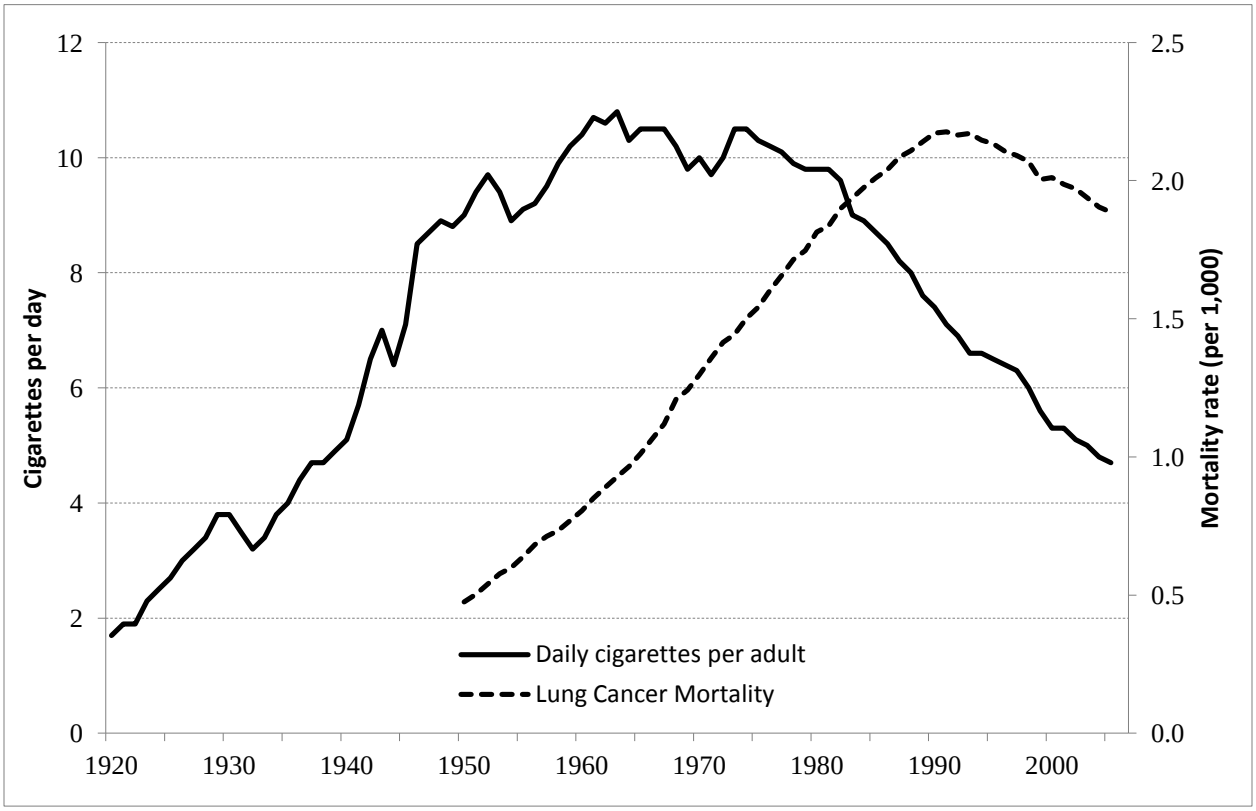


Figure 4. Trends in Smoking and Lung Cancer Mortality in the United States



Sources: Cigarette consumption data: International Smoking Statistics (2011); Lung cancer mortality data: National Center for Health Statistics.

Figure 5. U.S. Lung Cancer Mortality by Cohorts: Males

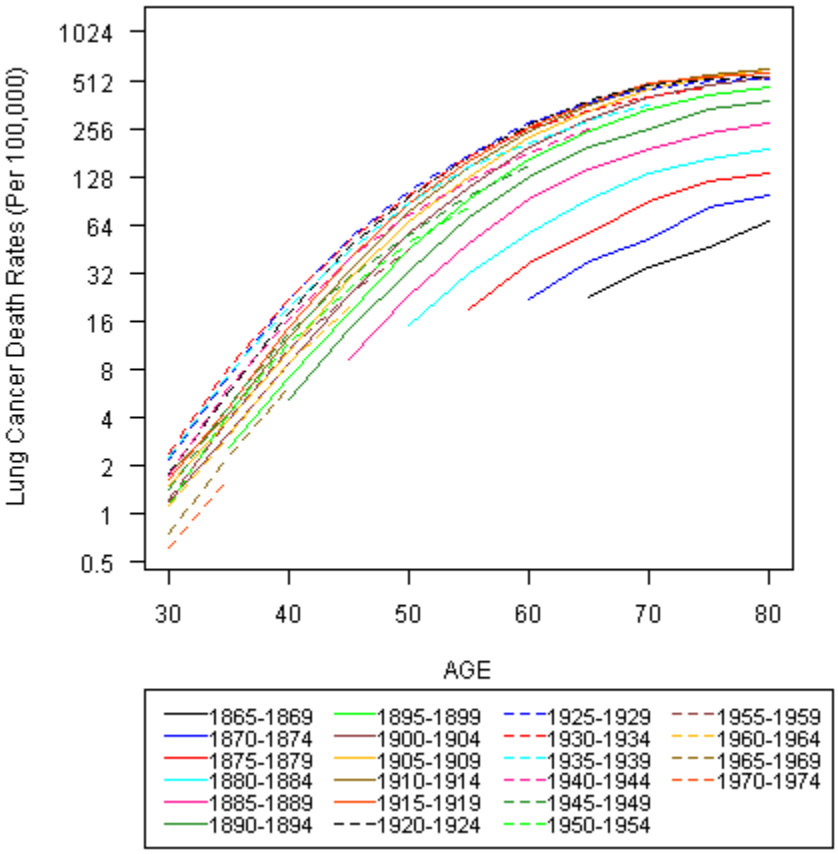


Figure 6. U.S. Lung Cancer Mortality by Period: Males

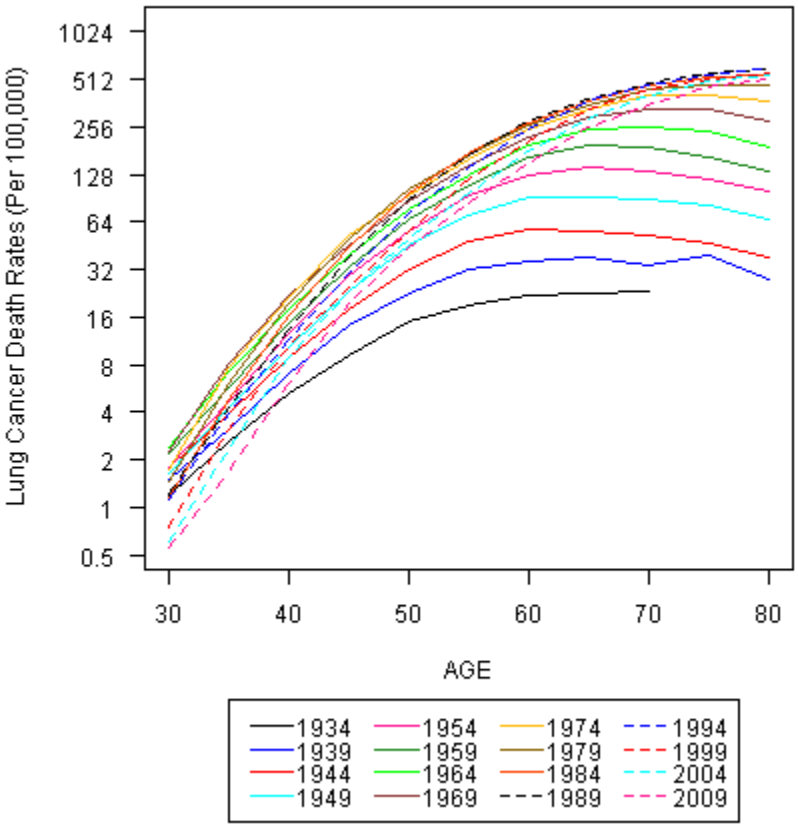


Figure 7. Mean Number of Years Spent as a Cigarette Smoker before Age 40 by Cohort

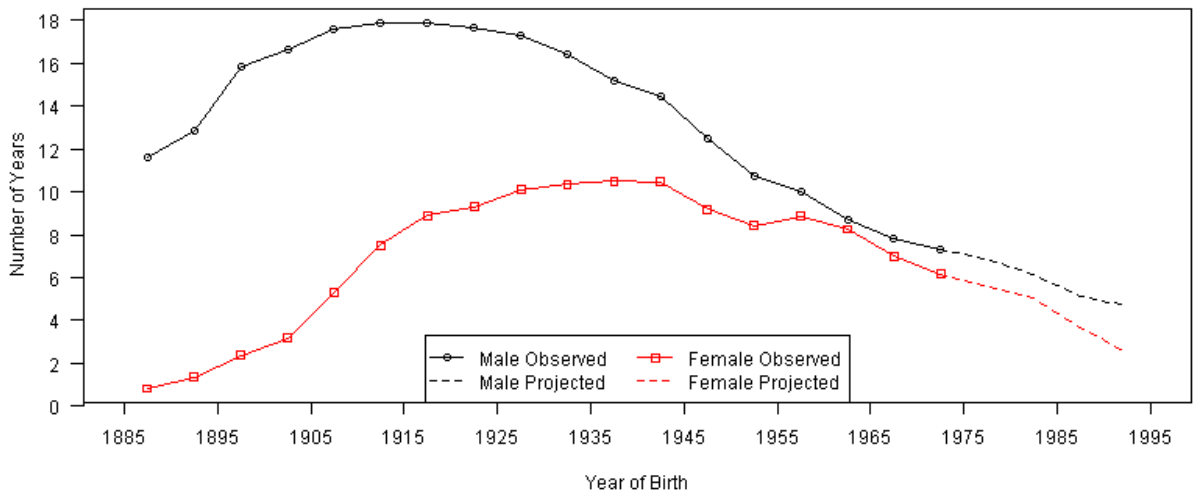
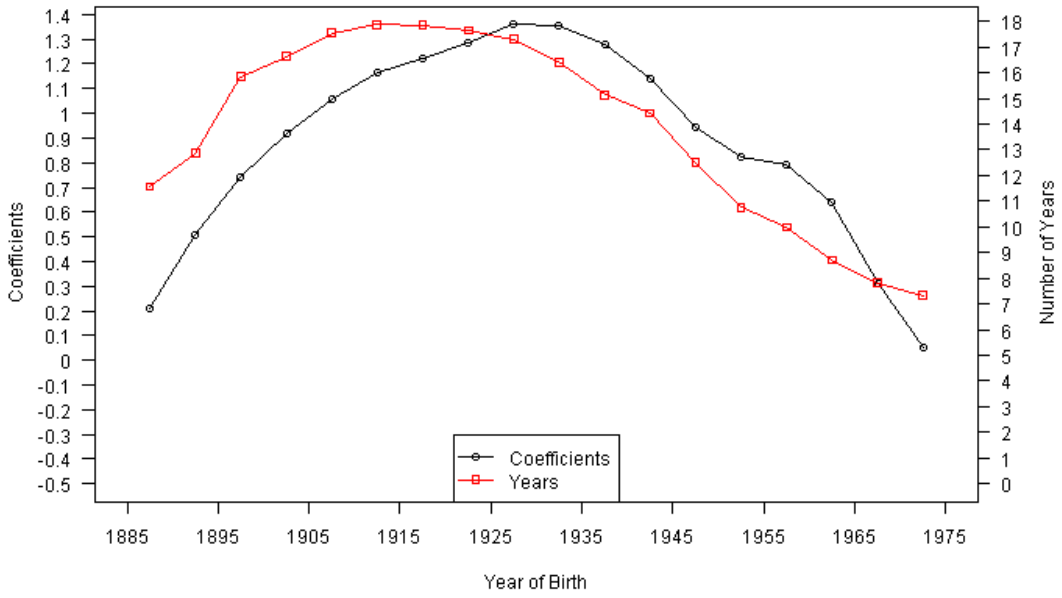


Figure 8. Cohort Coefficients Predicting Lung Cancer Mortality and Cumulative Cohort Smoking by Age 40

A. Males



B. Females

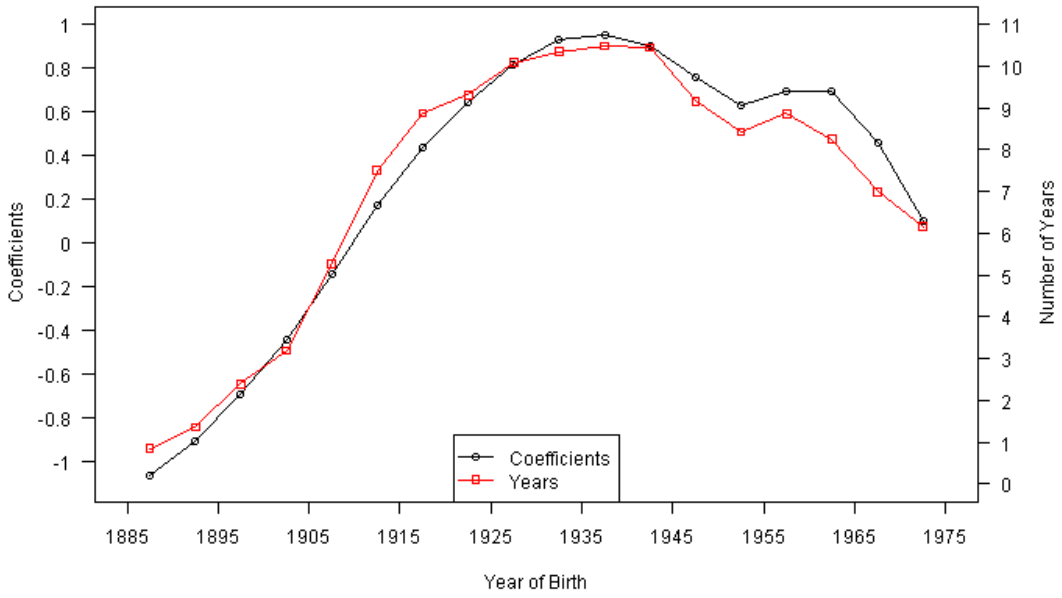
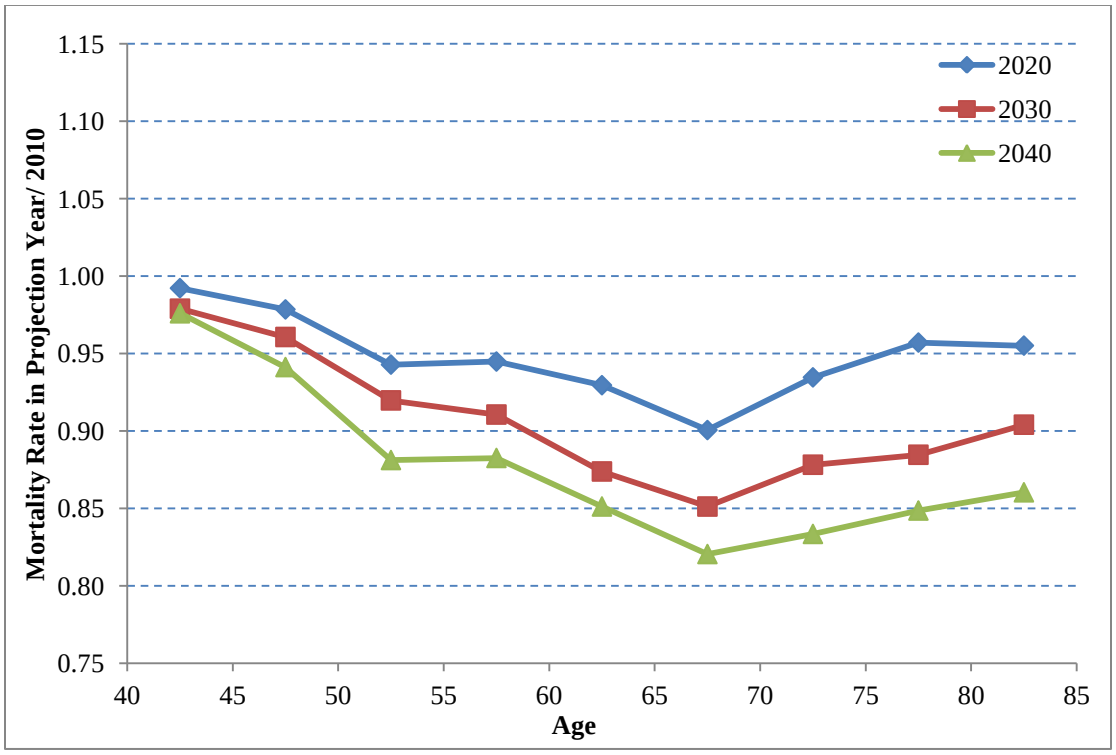


Figure 9: Effect of Projected Trends in Smoking on Age-Specific Death Rates

A. Males



B. Females

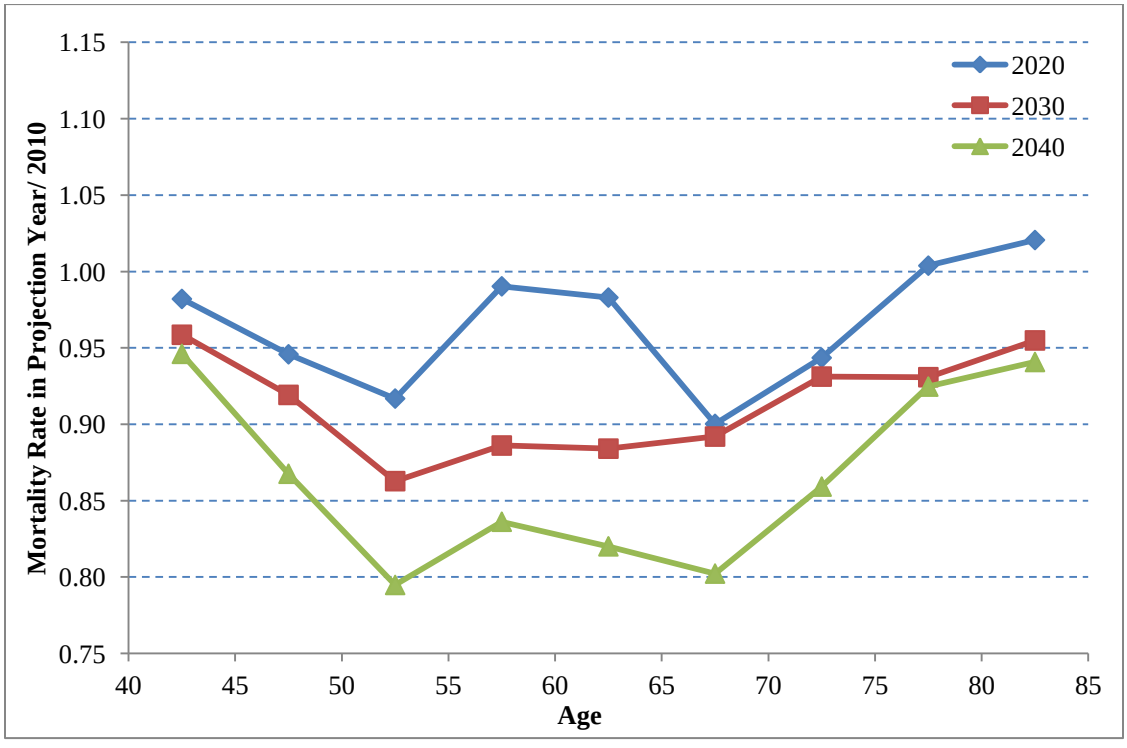
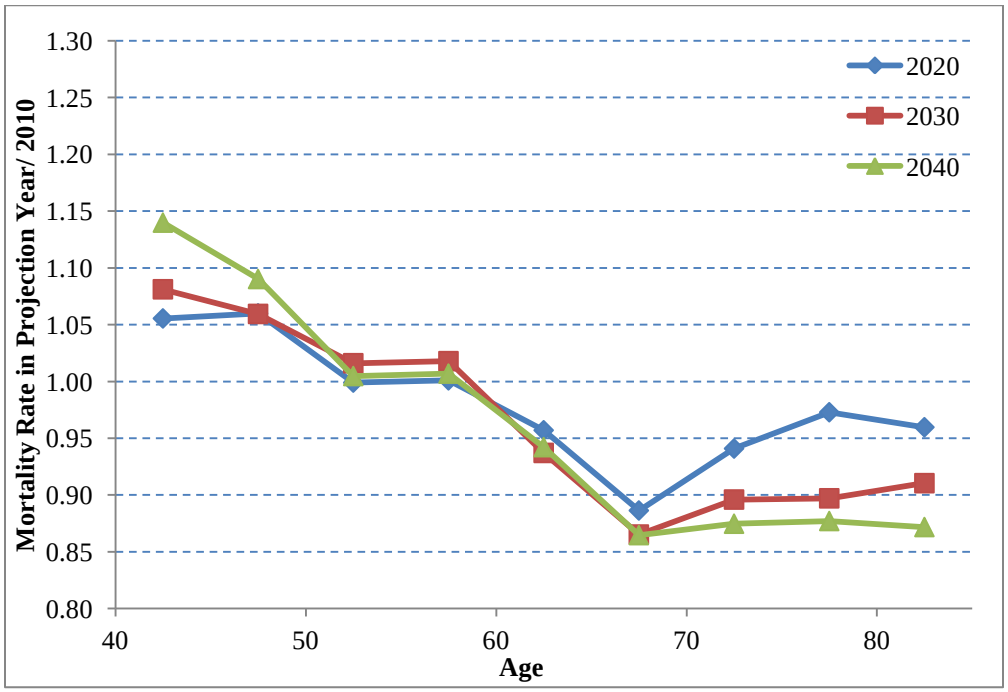


Figure 10. Effect of Projected Trends in Smoking and Body Mass Index on Age-Specific Death Rates

A. Males



B. Females

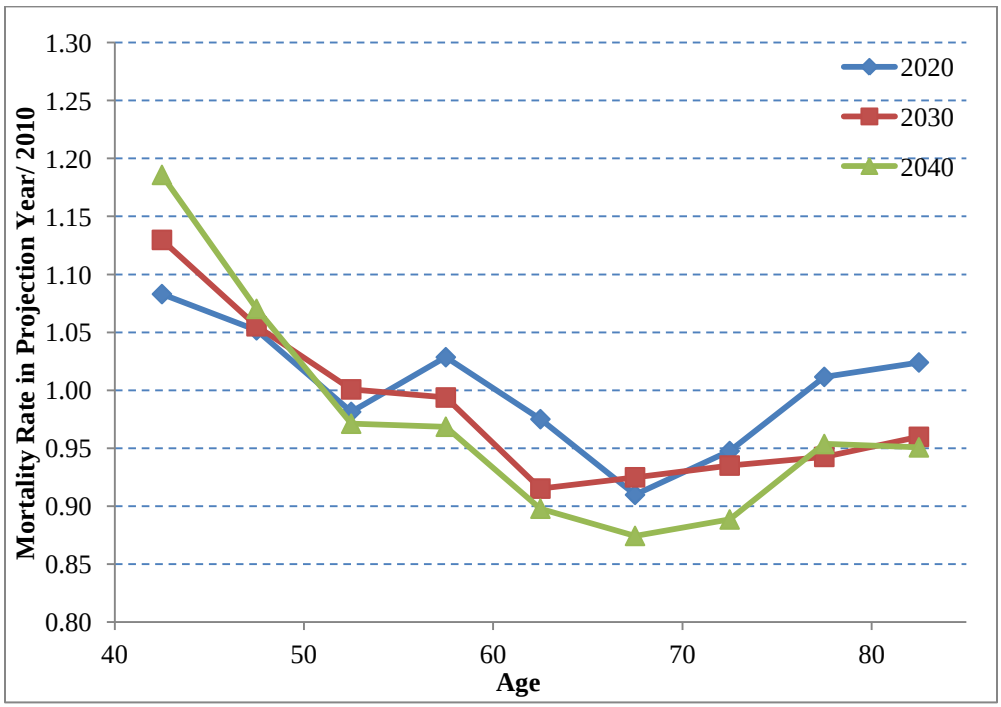


Table 1. Ten-Year BMI Transition Matrices in the United States

A. Transition probabilities across categories of BMI over three periods (adults ages 25-84)[a]

	1980-1990				1990-2000				1998-2008			
	Normal	Over	Obese 1	Obese 2	Normal	Over	Obese 1	Obese 2	Normal	Over	Obese 1	Obese 2
Normal	0.67	0.30	0.03	0.00	0.61	0.34	0.04	0.01	0.63	0.33	0.03	0.01
Overweight	0.12	0.52	0.29	0.08	0.10	0.48	0.32	0.10	0.09	0.51	0.31	0.09
Obese Class 1	0.02	0.22	0.44	0.31	0.02	0.16	0.41	0.41	0.02	0.21	0.42	0.35
Obese Class 2	0.01	0.07	0.28	0.64	0.00	0.04	0.20	0.76	0.00	0.06	0.24	0.70

B. Differences in transition probabilities (standard errors) across periods (adults ages 25-84)

	1980-1990 and 1990-2000				1990-2000 and 1998-2008			
	Normal	Over	Obese 1	Obese 2	Normal	Over	Obese 1	Obese 2
Normal	-0.057* (0.019)	0.044* (0.016)	0.011* (0.004)	0.002* (0.001)	0.022 (0.020)	-0.011 (0.017)	-0.009* (0.005)	-0.001 (0.001)
Overweight	-0.012 (0.011)	-0.040* (0.017)	0.030 (0.015)	0.022* (0.010)	-0.010 (0.013)	0.029 (0.022)	-0.014 (0.021)	-0.004 (0.014)
Obese Class 1	-0.006 (0.004)	-0.061* (0.023)	-0.033 (0.019)	0.100* (0.035)	0.003 (0.004)	0.046 (0.024)	0.010 (0.022)	-0.059 (0.031)
Obese Class 2	-0.002 (0.001)	-0.026* (0.013)	-0.089* (0.035)	0.117* (0.047)	0.001 (0.001)	0.015 (0.014)	0.041 (0.039)	-0.056 (0.053)

Sources: See Table A1.

[a]Transition probabilities across periods were predicted fixing demographic covariates at their average values in the most recent period.

* $p<0.05$

Table 2. Estimated Effects of Projected Trends in Body Mass Index on Mortality by Age and Sex: Mortality Rates in Projection Year Relative to 2010

	Males			Females		
Age	2020	2030	2040	2020	2030	2040
40-44	1.06	1.10	1.17	1.10	1.18	1.25
45-49	1.08	1.10	1.16	1.11	1.15	1.23
50-54	1.06	1.10	1.14	1.07	1.16	1.22
55-59	1.06	1.12	1.14	1.04	1.12	1.16
60-64	1.03	1.07	1.11	0.99	1.04	1.09
65-69	0.98	1.02	1.05	1.01	1.04	1.09
70-74	1.01	1.02	1.05	1.00	1.00	1.03
75-79	1.02	1.01	1.03	1.01	1.01	1.03
80-84	1.00	1.01	1.01	1.00	1.01	1.01

Notes: Mortality rates are obtained by applying projected BMI distributions at baseline (using categories normal, overweight, obese 1 and obese 2) and age 25 (using categories normal, overweight and obese) to coefficients from the discrete hazards model in Table A2.

Sources: Initial population data for the projection is derived from data from NHANES 2009-2010. Transition probabilities are developed from NHANES 2007-2008. Mortality rates are estimated using pooled NHANES 3 and NHANES 1999-2004 and follow-up in the National Death Index through Dec. 2006.

Table 3. Estimated Effect of Projected Trends in Smoking on Mortality by Age and Sex: Mortality Rate in Projection Year Relative to 2010

A. Males

Age	2015	2020	2025	2030	2035	2040
40-44	0.996	0.992	0.986	0.979	0.976	0.976
45-49	0.986	0.978	0.971	0.961	0.947	0.941
50-54	0.966	0.943	0.931	0.920	0.903	0.881
55-59	0.979	0.945	0.922	0.911	0.899	0.882
60-64	0.950	0.929	0.896	0.874	0.863	0.851
65-69	0.946	0.900	0.882	0.851	0.831	0.820
70-74	0.982	0.935	0.895	0.878	0.851	0.833
75-79	0.972	0.957	0.918	0.885	0.871	0.849
80-84	0.981	0.955	0.941	0.904	0.873	0.860

B. Females

Age	2015	2020	2025	2030	2035	2040
40-44	0.989	0.982	0.974	0.959	0.946	0.946
45-49	0.967	0.946	0.933	0.919	0.890	0.868
50-54	0.971	0.917	0.883	0.863	0.840	0.795
55-59	1.024	0.990	0.926	0.886	0.863	0.836
60-64	0.958	0.983	0.949	0.884	0.844	0.820
65-69	0.935	0.900	0.921	0.892	0.837	0.802
70-74	0.998	0.943	0.914	0.931	0.906	0.859
75-79	1.005	1.004	0.957	0.931	0.946	0.925
80-84	1.013	1.021	1.018	0.955	0.921	0.941

Table 4. Estimated Effects of Projected Trends in Smoking and Body Mass Index on Mortality by Age and Sex: Mortality Rates in Projection Year Relative to 2010

	Males			Females		
Age	2020	2030	2040	2020	2030	2040
40-44	1.06	1.08	1.14	1.08	1.13	1.19
45-49	1.06	1.06	1.09	1.05	1.06	1.07
50-54	1.00	1.02	1.01	0.98	1.00	0.97
55-59	1.00	1.02	1.01	1.03	0.99	0.97
60-64	0.96	0.94	0.94	0.98	0.92	0.90
65-69	0.89	0.87	0.87	0.91	0.93	0.87
70-74	0.94	0.90	0.88	0.95	0.94	0.89
75-79	0.97	0.90	0.88	1.01	0.94	0.95
80-84	0.96	0.91	0.87	1.02	0.96	0.95

Notes: Combined mortality ratios for smoking and body mass index are obtained by multiplying together the mortality ratios for each separately (Tables 2 and 3).

Sources: See Tables 2 and 3.

Table 5. Changes in Life Expectancy at Age 40 Resulting from Changes in Smoking and Obesity

	Changes in Smoking Alone		Changes in Obesity Alone		Changes in Smoking and Obesity	
Year	Males	Females	Males	Females	Males	Females
2015	0.281	-0.012				
2020	0.576	0.089	-0.241	-0.174	0.338	-0.076
2025	0.865	0.245				
2030	1.115	0.477	-0.467	-0.391	0.662	0.117
2035	1.387	0.858				
2040	1.617	1.142	-0.733	-0.677	0.914	0.539

Table 6. Sensitivity of Results to Changes in Procedures

Change in Procedure	Effect on Life Expectancy at age 40 Relative to Main Projection					
	2020		2030		2040	
	Male	Female	Male	Female	Male	Female
Use of mortality rates with no control except age & sex	0.016	0.009	0.023	0.018	0.029	0.031
Use of mortality rates without inclusion of BMI at age 25	0.029	0.012	0.131	0.082	0.293	0.198
Use of uncorrected data on obesity at age 25	-0.024	-0.026	-0.005	-0.036	0.044	-0.002
Use of alternative series translating lung cancer into all-cause mortality	-0.069	0.000	-0.178	-0.159	-0.330	-0.474

Appendix A. Validation of Retrospective Reports on BMI 10 Years Earlier

We carried out a validation procedure in order to investigate the consistency of 10-year recall data on BMI with actual cross-sectional measurements of BMI for the same cohort made 10 years earlier. In this comparison, it was necessary to recognize that cohort membership changed between observations through selective mortality by BMI status during the 10-year period; only the survivors of the 10-year period reported on their BMI 10 years earlier. Thus, our strategy is to begin with the cross-section of BMI's at t-10, survive these individuals forward by mortality rates observed in NHANES at various BMI levels, and then apply the transition matrix for the period t-10 to t to the survivors. We then compare the predicted distribution at time t to the actual distribution at time t. The cross-sectional distributions at t-10 and t are based on actual measurements, whereas the retrospective information about individual's BMI 10 years earlier is based on retrospective self-reports. To construct the transition matrix, we have corrected the recalled BMI at t-10 by applying an individual-level correction factor based on the proportionate error between measured and self-reported BMI at baseline (Flegal 1995).[8]

Concordance between predicted and actual distributions of BMI was assessed using root mean-squared error (RMSE) and also by examining the frequency with which predicted values of the proportion of individuals in various BMI categories fell within the 95% confidence intervals of actual values. Because we examine historical obesity dynamics as well as forecasting future BMI distributions and data quality may change over time, we conducted the validation separately for each of the three periods under examination: 1980-1990, 1990-2000 and 2000-2010. The transition matrix pertaining to 1997/98 to 2007/08 was used in the latter analysis since no later data were available.

Results are shown in Table A.1. It is clear that the predicted BMI distributions for 1990, 2000 and 2010 closely track the rightward shift in the BMI distribution that occurred over this period in all age-groups. For purposes of assessing the discrepancy between predicted and observed distributions of individuals across categories of age and BMI, the data were organized into three age-groups (35-54, 55-69 and 70-84) and four BMI intervals (normal, overweight, obese 1 and obese 2). RMSE was calculated separately for each of the three prediction periods (1990, 2000 and 2010) by comparing values across ages and BMI categories (for a total of 12 values per period). RMSE were 1.81, 2.09 and 1.74 for 1990, 2000 and 2010 respectively.

Concordance was also assessed by examining whether predicted values were within the 95% confidence intervals of observed values. There was a good concordance between the actual and observed proportions using this criterion. For the first of the three outcome years, three of the 12 comparisons of predicted to actual proportions showed a predicted proportion that was outside the 95% confidence interval of the observed proportion. For the second and third

[8] This is, in effect, a correction for errors of self-report. Recall weight data may be subject to both errors of self-reporting and recall bias. In a sensitivity analysis, we tested a simultaneous correction for both sources of error developed from NHANES 1 and NHEFS 1982-1983 data. However, in the validation step, this correction did not perform as well as the one used above. In particular it didn't perform well in the most recent period, which may be because the pattern of error has changed..

outcome years, only two of the 12 predicted values were outside the 95% CI of the observed proportion. We conclude that the retrospective reports on BMI 10 years earlier are quite effective in tracing the dynamics of the huge increase in obesity that occurred over a period of three decades. We therefore feel justified in using these reports to project BMI distributions into the future.

Table A1. Comparison of Predicted and Actual Obesity Distributions (95% CI) by Age and Period

Age (y)	BMI Cat. (kg/m^2)	1990[b] Predicted Value	1990[b] Actual (95% CI)		2000[c] Predicted Value	2000[c] Actual (95% CI)		2010[d] Predicted Value	2010[d] Actual (95% CI)	
35-54	<25	39.01	40.43	(37.60-43.27)	30.64	32.83	(30.06-35.59)	25.22	28.39	(25.68-31.11)
	25 to 30	35.50	34.30	(32.59-36.02)	34.40	35.36	(33.15-37.56)	34.02	34.03	(31.50-36.57)
	30 to 35	16.19	15.55	(13.92-17.19)	18.77	17.65	(16.09-19.20)	22.24	20.38	(18.49-22.27)
	>35	9.32	9.71	(8.00-11.42)	16.14	14.17	(12.03-16.31)	18.53	17.19	(15.36-19.02)
55-69	<25	35.94	32.33	(29.88-34.79)	27.11	26.21	(23.64-28.78)	24.98	23.13	(19.91-26.36)
	25 to 30	36.57	38.12	(35.29-40.95)	33.63	35.64	(32.93-38.36)	33.39	35.15	(30.73-39.57)
	30 to 35	17.76	20.30	(18.37-22.24)	21.44	22.69	(20.64-24.74)	21.79	23.13	(18.92-27.35)
	>35	9.79	9.24	(7.79-10.70)	17.85	15.45	(12.62-18.29)	19.83	18.58	(16.24-20.92)
70-84	<25	43.78	41.11	(38.42-43.80)	32.66	32.13	(28.93-35.32)	25.51	22.93	(19.32-26.53)
	25 to 30	35.43	37.91	(35.86-39.96)	36.84	41.25	(37.49-45.00)	35.85	36.15	(30.76-41.55)
	30 to 35	14.56	14.62	(12.56-16.68)	19.51	18.69	(15.81-21.57)	21.53	23.64	(20.50-26.78)
	>35	6.28	6.37	(5.11-7.63)	10.93	7.94	(6.27-9.60)	17.10	17.28	(13.45-21.10)
RMSE[a]			1.81			2.09			1.74	

[a]RMSE=root mean-squared error. This metric was used to assess error in predictions and was calculated within each period by comparing predicted to actual BMI values across age categories.

[b]Sources of data for 1990 prediction: *Initial population counts*: National Health and Nutrition Examination Survey (NHANES) 2 (1976-1980); *life tables*: NHANES 2 (1976-1980) with mortality follow-up through 1992; *transition probabilities and validation data*: NHANES 3 (1988-1994). Ten-year recall weight was corrected prior to estimating transition probabilities.

[c]Sources of data for 2000 prediction: *Initial population counts*: NHANES 3 (1988-1994); *life tables*: NHANES 3 (1988-1994) with mortality follow-up through 2006; *transition probabilities and validation data*: NHANES Cts. (1999-2002). Ten-year recall weight was corrected prior to estimating transition probabilities.

[d]Sources of data for 2010 prediction: *Initial population counts*: NHANES Cts. (1999-2002); *life tables*: NHANES 3 (1988-1994) and NHANES Cts. (1999-2004) with mortality follow-up through 2006; *transition probabilities:* NHANES Cts. (2007-2008); *validation data*: NHANES Cts. (2009-2010). Ten-year recall weight was corrected prior to estimating transition probabilities.

Appendix B. Procedures for Forecasting the Future Distribution of Age-25 BMI and Estimating the Effects of Weight History on Mortality Levels - Baseline and Sensitivity Analysis

Our baseline procedure for forecasting future distributions of age-25 BMI and estimating the effects of weight history on mortality levels relies on historical measured data on height and weight for deriving age-25 BMI distributions and corrected age-25 recall data for estimating life tables and effects of obesity on future mortality levels.

Measured data on height and weight with which to reconstruct cohort obesity patterns are available periodically since 1960 from the National Health and Nutrition Examination Survey (NHANES). All the available NHANES data were used to reconstruct the distribution of BMI at age 25 across five-year US birth cohorts between 1935-1937 and 1984-1985 (age 25 in 1960-62 to 2009-2010). BMI distributions at central age 25 (ages 20-29) were calculated in each of the following surveys, taking into account complex survey design: NHES I (1960-1962), NHANES I, NHANES II, NHANES III (disaggregated into Phases I (1988-1991) and II (1991-1994) and NHANES continuous waves 1999-2000, 2001-2002, 2003-2004, 2005-2006, 2007-2008, 2009-2010. In calculating BMI distributions, we used categories normal (BMI < 25 kg/m2), overweight (BMI 25-30) and obese (BMI > 30). As the proportions morbidly obese at age 25 in the historical series were so low, we did not further differentiate the obese category into obese class I, obese class II, etc.

Separately for the proportions overweight and obese, we fit trends to the data using linear regressions with splines, adjusting only for sex. We specified a knot at 1980 to reflect the rapid rise in obesity after that year. For the proportion overweight series, the knot has little effect (e.g. the slope is similar before and after 1980 and not statistically significantly different). However, for the proportion obese series, the slope is essentially flat before 1980, rising rapidly thereafter (a test of the statistical significance of the difference in slopes before and after 1980 indicates that the difference is significant). Using the parameters from the model, we interpolated the series to obtain proportions overweight and obese for birth cohorts separated by five-year periods (age 25 in 1962.5, 1967.5,…,2007.5). We also extrapolated the series to obtain proportions overweight and obese for earlier cohorts (age 25 in 1952.5) and later cohorts (age 25 in 2037.5). We obtain estimates for 1952.5 because we require weight histories for 80-84 year olds in 2010. We extrapolate to 2037.5, as this gives us weight histories for 25-29 year olds in 2040.

The age-25 series developed above provides weight histories for all adults ages 25-84 during the projection interval 2010-2040. However, these are not representative of the BMI distributions at age 25 of *surviving* adults in the projection interval as a result of differential mortality. Therefore, the next step in our procedure is to adjust the age-25 distributions for differential mortality according to BMI-at-age-25 specific mortality rates. We develop sex- and BMI-at-age-25 specific life tables for the US adult population, in order to survive members of each age-25 cohort to their attained ages in 2010, 2020, 2030 and 2040. We generate the life tables using a discrete hazards model implemented on person-month data. The parameters of the model appear in Table B1. Baseline data for the modeling are drawn from pooled NHANES 3

and continuous waves 1999-2004 and mortality follow-up data through 2006 are obtained from the National Death Index. Adults aged 25-84 are included in the sample and mortality is modeled as a function of BMI at age 25 using categories of normal, overweight and obese. BMI at age 25 is elicited through recall at the time of the survey. Recall data rather than measured historical data were used for the modeling as we required information on BMI at the individual level for estimating mortality risks. Prior to implementing the model, we used an individual-level correction of the recall data, based on the proportionate error between measured and self-reported current BMI, which were also elicited in the survey. We graphically assessed the quality of the correction by plotting the corrected age-25 mean BMI recall series against the same series derived from measured data for the sexes combined and comparing their correspondence with respect to the uncorrected age-25 series. We found close correspondence between the corrected and measured series, indicating that the correction improved upon the original data. After estimating the discrete hazards model, we generated the life tables on the basis of predictions from the model parameters.

The above data and procedures provide the inputs necessary for surviving forward cohorts of 25 year olds by sex and BMI at age 25. As a result of these projections, we obtain age-25 BMI levels, adjusting for differential mortality, for all cohorts aged 25-84 between 2010 and 2040. To calculate the total effects of obesity (age-25 and baseline BMI levels) on mortality levels in the future, we apply the parameters of the "joint" mortality model to the projected baseline and age-25 BMI distributions. As before, the recall data are corrected prior to running the model.

In our sensitivity analysis, we reconstruct the age-25 BMI series using (uncorrected) recall rather than measured data and use uncorrected rather than corrected recall data for developing life tables and modeling mortality as a function of BMI at age 25. As a first step in the sensitivity analysis, we estimate age-25 BMI distributions for individuals aged 25-84 in 2005 based on age-25 recall data available in NHANES 2003-2006. Distributions for each age-25 cohort are estimated using the same categories of BMI as above. We begin in 2005 instead of 2010, as recall data were not available in the most recent NHANES continuous wave (2009-2010). Second, we estimate age-25 BMI distributions for future cohorts of 25-29 year olds, beginning with 25-29 year olds in 2010 (who were age 25 in 2007.5) and ending with 25-29 year olds in 2040 (who were age 25 in 2037.5). To do so, we first estimate age-25 BMI distributions for three-year birth cohorts between 1930-32 and 1978-1980 (i.e. age-25 cohorts 1955-57 to 2003-2005) on the basis of age-25 recall data in NHANES continuous waves 1999-2008. This involved organizing BMI data by cohort by subtracting age from survey year. We grouped birth cohorts into three-year intervals to increase sample sizes. Second, we separately regress the proportion overweight and obese on a dummy for male and linear time. Using the parameters of these regressions, we extrapolate the age-25 overweight and obese series forward in five-year increments between 2007.5 and 2037.5.

Next, we carry out the life table step outlined above for the baseline procedure; however, in the sensitivity analysis we estimate the equations on uncorrected rather than corrected weight

history data. The parameters of this model appear in Table B2. Using the sex and BMI-at-25 specific life tables, we survive the population forward by BMI category and sex. In contrast to the main procedure, it was not necessary to apply differential mortality beginning at age 25 for all cohorts since those reporting age-25 BMI had survived to 2005. Instead, differential survival rates were applied beginning from age at baseline in 2005. In the last step of the sensitivity analysis, we apply the parameters of the uncorrected "joint" mortality model to baseline and age-25 BMI projections to obtain the effects of obesity on future mortality rates.

Table B1. Baseline Model Parameters for Regression Equation Predicting Mortality as a Function of BMI at Age 25. Model with correction of obesity history data.

Covariates	Coefficients	SE	t	P>t
Female	-0.373	0.048	-7.84	0.000
Age	0.088	0.003	28.10	0.000
Age 25[a]				
Overweight (BMI 25-30)	0.184	0.054	3.44	0.001
Obese (BMI>30)	0.486	0.109	4.46	0.000
Constant	-9.294	0.121	-77.04	0.000

Notes: Parameters are estimated using a discrete hazards model implemented on person-month data. Age represents the number of attained years beyond age 35. Ages 25-84 are included in the analysis. The first two years of mortality follow-up are excluded. Estimates reflect sample weights and complex survey design. Sample size=21,734; person-years of follow-up=2,041,354; No. of deaths=3,006.

Sources: See Table C1.

[a]BMI at age 25 is corrected for misreporting errors.

Table B2. Sensitivity Analysis Model Parameters for Regression Equation Predicting Mortality as a Function of BMI at Age 25. Model implemented on uncorrected obesity history data.

Covariates	Coefficients	SE	t	P>t
Female	-0.363	0.048	-7.63	0.000
Age	0.089	0.003	28.33	0.000
Age 25				
Overweight (BMI 25-30)	0.209	0.062	3.36	0.001
Obese (BMI>30)	0.534	0.116	4.61	0.000
Constant	-9.306	0.119	-78.23	0.000

Notes: See Table B1.
Sources: See Table C1.

Appendix C: Model Parameters for Regression Equations Predicting Mortality as a Function of Current Obesity and Obesity at Age 25: Baseline and Sensitivity Analysis Models

Table C1. Baseline Model Parameters for Regression Equation Predicting Mortality as a Function of Current Obesity and Obesity at Age 25. Model with covariate adjustment and correction of obesity history data.

Covariates[a]	Coefficients	SE	t	P>t
Female	-0.309	0.050	-6.13	0.000
Age	0.099	0.004	25.58	0.000
Age 25[b]				
Overweight (BMI 25-30)	0.131	0.063	2.06	0.043
Obese (BMI>30)	0.309	0.106	2.91	0.005
Baseline				
Obese class 1 (BMI 30-35)	0.355	0.339	1.05	0.299
Obese class 2 (BMI>35)	0.897	0.282	3.18	0.002
Obese class 1 x Age	-0.008	0.009	-0.87	0.385
Obese class 2 x Age	-0.018	0.008	-2.08	0.040
Constant	-9.994	0.186	-53.79	0.000

Notes: Parameters are estimated using a discrete hazards model implemented on person-month data. Age represents the number of attained years beyond age 35. Ages 40-84 are included in the analysis. Baseline obesity terms are derived from measured data on height and weight. The first two years of mortality follow-up are excluded. Estimates reflect sample weights and complex survey design. Sample size=21,734; person-years of follow-up=2,041,354; No. of deaths=3,006.

Sources: Data are derived from pooled NHANES III (1988-1994) and NHANES 1999-2004 with mortality follow-up in the National Death Index through December 2006.

[a]Additional covariates in the model include race/ethnicity, educational attainment and smoking.

[b]BMI at age 25 is corrected for misreporting errors.

Table C2. Sensitivity Analysis Model Parameters for Regression Equation Predicting Mortality as a Function of Current Obesity and Obesity at Age 25. Model implemented without covariate adjustment.

Covariates	Coefficients	SE	t	P>t
Female	-0.396	0.049	-8.12	0.000
Age	0.093	0.003	28.13	0.000
Age 25[a]				
Overweight (BMI 25-30)	0.122	0.060	2.06	0.043
Obese (BMI>30)	0.311	0.108	2.89	0.005
Baseline				
Obese class 1 (BMI 30-35)	0.318	0.322	0.98	0.328
Obese class 2 (BMI>35)	0.812	0.291	2.79	0.007
Obese class 1 x Age	-0.007	0.008	-0.89	0.375
Obese class 2 x Age	-0.016	0.009	-1.83	0.071
Constant	-9.480	0.131	-72.64	0.000

Notes: See Table C1.

Sources: See Table C1.

[a]BMI at age 25 is corrected for misreporting errors.

Table C3. Sensitivity Analysis Model Parameters for Regression Equation Predicting Mortality as a Function of Current Obesity and Obesity at Age 25. Model implemented on uncorrected obesity history data.

Covariates[a]	Coefficients	SE	t	P>t
Female	-0.305	0.051	-5.99	0.000
Age	0.099	0.004	25.64	0.000
Age 25				
Overweight (BMI 25-30)	0.138	0.065	2.10	0.039
Obese (BMI>30)	0.330	0.107	3.09	0.003
Baseline				
Obese class 1 (BMI 30-35)	0.365	0.333	1.10	0.276
Obese class 2 (BMI>35)	0.906	0.284	3.19	0.002
Obese class 1 x Age	-0.008	0.009	-0.89	0.376
Obese class 2 x Age	-0.017	0.008	-2.05	0.043
Constant	-9.998	0.184	-54.20	0.000

Notes: See Table C1.

Sources: See Table C1.

[a]Additional covariates in the model include race/ethnicity, educational attainment and smoking.

Table C4. Sensitivity Analysis Model Parameters for Regression Equation Predicting Mortality as a Function of Current Obesity Only.

Covariates[a]	Coefficients	SE	t	P>t
Female	-0.337	0.049	-6.88	0.000
Age	0.099	0.004	25.60	0.000
Baseline				
Obese class 1 (BMI 30-35)	0.445	0.341	1.31	0.195
Obese class 2 (BMI>35)	1.085	0.298	3.64	0.000
Obese class 1 x Age	-0.009	0.009	-1.00	0.320
Obese class 2 x Age	-0.020	0.009	-2.28	0.025
Constant	-9.954	0.185	-53.82	0.000

Notes: See Table C1.

Sources: See Table C1.

[a]Additional covariates in the model include race/ethnicity, educational attainment and smoking.

Appendix D. Construction of Life Tables

The most common summary measure of age-specific mortality rates is life expectancy, an indicator of how many additional years someone is expected to live after reaching a particular age. Life tables are required in order to estimate life expectancy. The baseline life table that we use for comparison purposes is that for the United States in 2009, which we assume to apply to 2010. The National Center for Health Statistics has not published life tables for 2009 but has provided several preliminary estimates of life expectancy at various ages (Kochanek et al. 2011) We use their value of life expectancy at age 85 and construct our own life table values for ages 40-84 based on age-specific death rates for both sexes in 5-year age intervals that have been released electronically (US National Center for Health Statistics 2012). Our constructed life table has a value of life expectancy at age 40 that is within 0.3 years of the preliminary value published by NCHS (Kochanek et al. 2011: Table 6). To this baseline life table we apply the projected proportionate changes in age-specific death rates under various scenarios and recalculate the life table, including value of e(40), in order to estimate the effects of impending changes in smoking and obesity histories.

We did not estimate the effects of either smoking or obesity on mortality at ages 85+. In the case of obesity, we assume that there is no effect of BMI on mortality above age 85. The BMI/age interaction terms shown in the regression equations of Appendix C produce a negative risk of being either Obese I or Obese II-III by age 87.5. So this assumption seems justifiable. In the case of smoking, we assume that the coefficients of cohort smoking histories estimated on data for ages 40-84 is applicable at ages 85+. The value of the age coefficient at ages 85+ is set at its value for ages 80-84, reflecting the leveling off of age effects at advanced ages.