Demography (2014) 51:27–49
DOI 10.1007/s13524-013-0246-9

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

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Published online: 23 November 2013

Abstract We estimate 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 estimates. 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 are 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. Estimates of the effects of smoking changes are based on observed relations between cohort smoking patterns and cohort death rates from lung cancer. We find that changes in both smoking and obesity are expected to have large effects on U.S. mortality. For males, the reductions in smoking have larger effects than the rise in obesity throughout the projection period. By 2040, male life expectancy at age 40 is expected to have gained 0.83 years from the combined effects. Among women, however, the two sets of effects largely offset one another throughout the projection period, with a small gain of 0.09 years expected by 2040.

Keywords Projections · Mortality · Longevity · Smoking · Obesity

Introduction

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

Electronic supplementary material The online version of this article (doi:10.1007/s13524-013-0246-9) contains supplementary material, which is available to authorized users.

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singled out as especially damaging to the health of the U.S. population are smoking and the interplay of diet and physical activity that results in obesity. Estimates by the Centers for Disease Control (CDC) suggest that 18 % of deaths in the United States in 2000 were attributable to smoking and 15 % resulted from obesity (Mokdad et al. 2004, 2005). The prevalence of obesity has been rising in the United States, but cigarette smoking has declined (Fig. 1).

Uncertainty about the future effect of these behaviors is a central component of the 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 Old-Age and Survivors Insurance program of the Social Security Administration (SSA) is more sensitive to variation in future mortality rates than it is to any other demographic or economic parameter except real wages (Trustees 2012). A reliable projection of the effects of these two behaviors on future life expectancy would contribute to a better understanding of the fiscal future of the United States (Soneji and King 2012).

In this article, we estimate the effects of declining smoking and increasing obesity on mortality at ages 40+ in the United States over the period 2010–2040. Our estimates incorporate information about cohorts' behavioral histories, allowing mortality rates to be a function not only of current behaviors but also of past behaviors. Prospective cohort studies demonstrate that the history of obesity, in addition to baseline obesity, is an important risk factor in mortality (Abdullah et al. 2011; Preston et al. 2013). Duration of smoking is strongly related to mortality risks among current smokers (Thun et al. 1997). An analytic advantage of incorporating behavioral histories into projections of future mortality levels is that many features of those histories have already been observed and are not themselves products of an uncertain future.

Overview of Analytic Strategy

Our goal is to estimate the effect of changes in the lifetime distributions of smoking and obesity on future death rates. We project body mass index (BMI) distributions

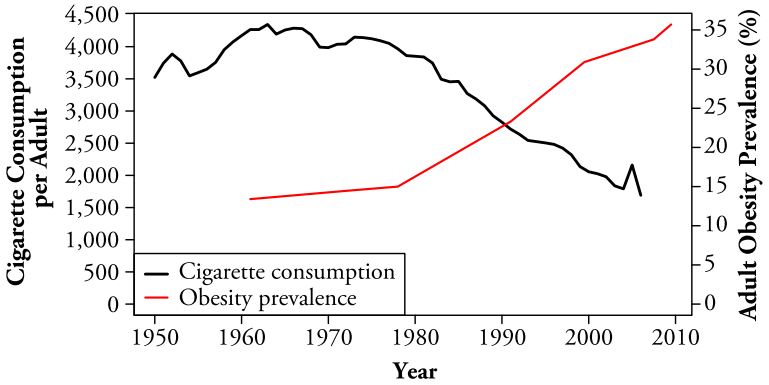

Fig. 1 Trends in smoking and obesity in the United States. *Sources*: Cigarette consumption data per adult per year are extracted from U.S. Department of Agriculture (2007). Obesity data are based on measured body mass index in NHANES from 1960 to 2010

that contain both past and contemporaneous levels of obesity. We then apply multi-dimensional mortality risks that reflect the effect of past and current levels of obesity on mortality. These risks are derived from 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 U.S. men aged 30 and older in 2005, an estimated 90 % of lung cancer deaths are attributable to smoking; for females, the figure is 84 % to 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 and elsewhere. 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 effect on lung cancer and its effect on all-cause mortality.

In the case of both smoking and obesity, our goal is to estimate the proportionate effect of changes in these behaviors on age-specific death rates. Our comparison schedule is simply the age-specific death rates at baseline, 2009–2010, which reflect the behavioral histories that had been accumulated at that point. We are not attempting to project mortality rates themselves, but rather only to estimate the effect of changes in these behaviors on mortality. If there are other sources of future change in mortality, we are implicitly assuming that the effects of changes in these behaviors will be independent of them. King and Soneji (2011) produced Bayesian forecasts of American mortality (also see Soneji and King 2012). Smoking and obesity were integrated into the forecasts, but the analysis does not identify their separate or joint effects.

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 sex- and age-specific BMI transition matrices derived from NHANES. We use a combination of the five 10-year transition matrices that were reported in NHANES between 1999 and 2008. We present evidence that 10-year BMI transition matrices have been nearly constant for the past eight years, and we maintain the assumption of constancy into the future. 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. Third, we apply death rates drawn from NHANES to the distribution of current BMI and BMI at age 25.

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 (Ruhm 2007; Stewart et al. 2009; Wang et al. 2008). 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 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 (see Baum and Chou (2011) for 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 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 that projection of covariate series often involves as much uncertainty as projecting obesity directly.

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 on 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 stable, and we project that stability into the future.

To develop transition probabilities, we use data from NHANES, a series of nationally representative surveys of the noninstitutionalized U.S. population conducted by the National Center for Health Statistics (NCHS). The survey includes an examination component in which extensive medical data, including height and weight, are collected by trained nurses in mobile clinics or at in-home visits. We use data on measured height and weight at the time of the survey to calculate current BMI. Certain NHANES surveys ask respondents to recall their weight 10 years prior to the survey.[1] We combine data on recall weight with current height to estimate "recall BMI" in each period. To reduce bias that may result from inaccurate recall of past weight, we apply an individual-level correction factor based on the proportionate error between measured and self-reported BMI at baseline (Flegal et al. 1995). The combination of corrected recall BMI and current BMI serves 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 use ordered logistic regression models to estimate age- and sex-specific transition probabilities across BMI categories in each of three decadal periods between 1980 and 2010 (i.e., 1980–1990, 1990–2000, 2000–2010). A key question is how well these transition matrices predict changes in obesity that have actually occurred. We show in Online Resource 1 that the transition matrices are highly

[1] These data are available for ages 35 and older, so we are limited to modeling transitions beginning at age 25.

effective in projecting the BMI distributions observed in various NHANES from the beginning to the end of each projection period.

The summary transition matrices for all ages combined, denoted as M, are shown in Table 1 for these three periods; Table 2 shows the changes in transition probabilities in each cell between the two successive matrices. The changes from 1980–1990 to 1990–2000 are large and systematic. The probability of moving up in weight class was significantly higher during 1990–2000 than in 1980–1990. Even the highest weight class, from which no upward movement is possible, contributed to the upsurge in obesity by virtue of a significant increase in the probability of remaining in Obese II–III if a person started there.

In contrast, the changes between 1990–2000 and 1998–2008 were small. Only 1 of the 16 cells in the transition matrix showed a significant change in the transition probability during this period. On the basis of the relative stabilization of the BMI transition matrix over the past two decades, we generate a transition matrix combining data from five NHANES surveys from 1999 to 2008 and assume that this matrix is constant over the three decades starting in 2010. The summary matrix is presented in Table 3. The implication of this assumption of constancy is that the multitude of processes that produce weight change would operate with the same intensity in the future as they did in the recent past.

To project the BMI distribution after 2010, we begin with initial population counts in the 2009–2010 continuous NHANES, 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–III). Sample weights are incorporated so that counts are representative of the U.S. population in that period. In each round of the projection, the first step is 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 on a person-month file 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 produces new population counts, which serve as the initial counts for the next iteration of the projection. A new cohort of 25- to 34-year-olds is assumed to enter the population each decade. The distribution of BMI in these cohorts is predicted through extrapolation of the historical trend.[2]

Figure 2 presents the results of these projections for men and women. By 2040, 47 % of men and 51 % of women are projected to be obese. Some deceleration in the rate of increase in obesity is evident with the passage of time, in contrast to linear extrapolations. The morbidly obese (BMI $\geq$ 35.0) increase as a proportion of the obese for both males and females, to the point where they constitute a majority of obese women by 2020 and thereafter.

[2] We estimate a historical series for ages 25–34 using measured data on height and weight from NHANES continuous waves 1999–2010. We regress the proportion in each BMI category on the logarithm of time (years since 1970), an indicator for sex as well as an interaction between the two, and use the parameters of the model to predict the proportions in each category of BMI for each sex in 2020, 2030, and 2040.

Table 1 Ten-year BMI transition matrices in the United States: Transition probabilities (standard errors) across categories of BMI over three periods (adults aged 25–84)

	1980–1990				1990–2000				1998–2008			
	Normal	Over	Obese I	Obese II–III	Normal	Over	Obese I	Obese II–III	Normal	Over	Obese I	Obese II–III
Normal	.67 (.01)	.30 (.01)	.03 (.00)	.00 (.00)	.61 (.01)	.34 (.01)	.04 (.00)	.01 (.00)	.63 (.02)	.33 (.01)	.03 (.00)	.01 (.00)
Overweight	.12 (.01)	.52 (.01)	.29 (.01)	.08 (.01)	.10 (.01)	.48 (.01)	.32 (.01)	.10 (.01)	.09 (.01)	.51 (.02)	.31 (.02)	.09 (.01)
Obese Class I	.02 (.00)	.22 (.02)	.44 (.02)	.31 (.03)	.02 (.00)	.16 (.01)	.41 (.01)	.41 (.02)	.02 (.00)	.21 (.02)	.42 (.02)	.35 (.02)
Obese Class II–III	.01 (.00)	.07 (.01)	.28 (.02)	.64 (.03)	.00 (.00)	.04 (.01)	.20 (.03)	.76 (.04)	.00 (.00)	.06 (.01)	.24 (.03)	.70 (.04)

Sources: Transition probabilities across periods were predicted fixing age at its population average in 2007–2008. Transition probabilities for 1980–1990 were estimated using data from the National Health and Nutrition Examination Survey (NHANES) III; those for 1990–2000 and 1998–2008 were estimated using data from NHANES continuous waves 1999–2002 and 2007–2008, respectively.

Table 2 Ten-year BMI transition matrices in the United States: Differences in transition probabilities (standard errors) across periods (adults aged 25–84)

	1980–1990 and 1990–2000				1990–2000 and 1998–2008			
	Normal	Over	Obese I	Obese II–III	Normal	Over	Obese I	Obese II–III
Normal	–.057*	.044*	.011*	.002*	.022	–.011	–.009*	–.001
	(.019)	(.016)	(.004)	(.001)	(.020)	(.017)	(.005)	(.001)
Overweight	–.012	–.040*	.030	.022*	–.010	.029	–.014	–.004
	(.011)	(.017)	(.015)	(.010)	(.013)	(.022)	(.021)	(.014)
Obese Class I	–.006	–.061*	–.033	.100*	.003	.046	.010	–.059
	(.004)	(.023)	(.019)	(.035)	(.004)	(.024)	(.022)	(.031)
Obese Class II–III	–.002	–.026*	–.089*	.117*	.001	.015	.041	–.056
	(.001)	(.013)	(.035)	(.047)	(.001)	(.014)	(.039)	(.053)

Sources: Transition probabilities for 1980–1990 were estimated using data from the National Health and Nutrition Examination Survey (NHANES) III; those for 1990–2000 and 1998–2008 were estimated using data from NHANES continuous waves 1999–2002 and 2007–2008, respectively.

*$p < .05$

Forecasting the Future Distribution of Age-25 BMI

We project age-25 BMI levels for all cohorts who will be aged 25–84 at any time between 2010 and 2040. We begin with initial population counts in 2005 arrayed by sex, age, and BMI at age 25. These are derived from NHANES continuous waves 2003–2006. BMI at age 25 is calculated by combining self-reported weight at age 25 with measured height at baseline for individuals younger than age 50 and by combining self-reported weight at age 25 with self-reported height at age 25 for individuals aged 50 or older. We then survive the initial distribution forward in five-year intervals using sex-, age-, and age-25 BMI-specific life tables. Prior to estimating the initial population distribution and life tables, we apply an individual-specific correction factor to reported age-25 BMI to account for potential errors in reporting.

Table 3 Sex-specific 10-year BMI transition matrices in the United States: Transition probabilities (standard errors) across categories of body mass index and sex (U.S. adults aged 25–84)

	Males				Females			
	Normal	Over	Obese I	Obese II–III	Normal	Over	Obese I	Obese II–III
Normal	.62 (.01)	.34 (.01)	.04 (.00)	.01 (.00)	.60 (.01)	.35 (.01)	.04 (.00)	.01 (.00)
Overweight	.13 (.01)	.53 (.01)	.27 (.01)	.07 (.00)	.07 (.01)	.44 (.01)	.37 (.01)	.13 (.01)
Obese Class I	.02 (.00)	.23 (.01)	.44 (.01)	.31 (.01)	.02 (.00)	.16 (.01)	.41 (.01)	.40 (.02)
Obese Class II–III	.00 (.00)	.06 (.01)	.24 (.02)	.70 (.03)	.00 (.00)	.05 (.01)	.22 (.02)	.73 (.02)

Sources: Transition matrices were developed using combined data from NHANES 1999–2008.

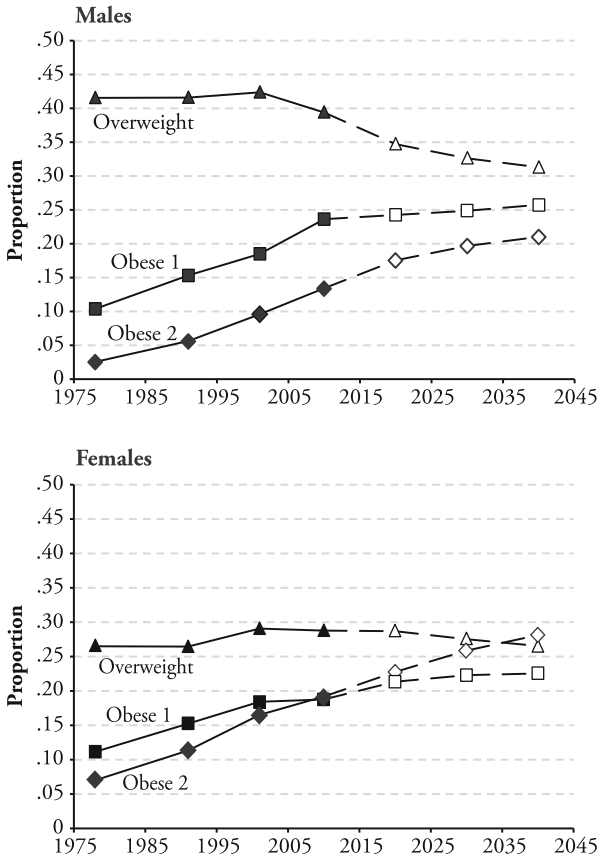

Fig. 2 Actual and projected trends in body mass index. Dotted lines indicate projections. *Sources*: Historical values are calculated using the National Health and Nutrition Examination Survey II, III, 1999–2004 and 2009–2010

Again, this correction factor is calculated as the proportionate error between measured and self-reported BMI at baseline. Details may be found in Online Resource 2.

Estimating Death Rates by Category of BMI

Data for the mortality analysis are derived by pooling the NHANES III (1988–1994) and NHANES continuous 1999–2002 surveys. BMI at baseline is calculated using measured data on height and weight. For purposes of calculating BMI at age 25, measured height at baseline is used for all individuals because self-reported height at age 25 was not available in NHANES III. To reduce bias in estimates of the mortality effects of obesity resulting from reverse causation, we exclude individuals with emphysema or a smoking-related cancer[3] at baseline, and we also eliminate the first three years of exposure. A discrete hazard model is used on a person-month file.

[3] The category of smoking-related cancers is based on relative risks in a recent large study (Pirie et al. 2012) and includes cancers of the lung, larynx, mouth/tongue/lip, esophagus, bladder, kidney, and pancreas.

Information on deaths is available through December 31, 2006. There were 1,894 deaths among 13,737 respondents.

Our BMI categories at age 25 are Overweight (BMI 25.0 to <30.0), Obese (≥30.0), and Normal (<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 age-25 and baseline BMI categories, age attained over follow-up (years), sex, race/ethnicity (white, black, Hispanic, or other), educational attainment (less than high school, high school diploma/GED, some college, or college graduate), and smoking status at baseline (current, former, or never). We include interaction terms between linear attained age (measured from age 40) and the two baseline obese categories because of strong 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. NCHS-supplied survey weights and design elements (strata and primary sampling units) are used.

Coefficients are shown in Table S2 in Online Resource 2. Weight at age 25 as well as baseline weight categories are related to the risk of death in the expected direction. Age interactions with baseline obesity are negative, as expected, and are retained in predictions.

Uncertainty Analysis

We analyze uncertainty in the effects of obesity on change in life expectancy using a bootstrapping procedure (Efron and Tibshirani 1986). We combine uncertainty originating from three sources: estimation of age-, sex-, and BMI-specific transition rates; age-, sex-, and BMI-specific life tables used to survive the population forward; and parameters of the model relating current BMI and BMI at age 25 to mortality. BMI transition matrices were estimated using an ordered logit model, and the life tables and coefficients relating current BMI and BMI at age 25 to mortality were generated using discrete hazards models. We use a multivariate random normal distribution to simulate the parameters of each of the three models, inputting the mean parameter values and the variance-covariance matrix of each of the regressions. Using the bootstrapped coefficients, we estimate 1,000 sets of transition matrices and life tables, and used these to generate 1,000 predictions of future obesity levels. We then apply each obesity projection to the bootstrapped coefficients of the mortality model to predict mortality rates and life expectancy effects. We extract the 2.5 and 97.5 percentile values as 95 % confidence intervals (CI).

Results

The combined effect of the projected changes in BMI, including age-25 overweight and obesity, and the estimated mortality risks are shown in Fig. 3. For both males and females, the effect of changes in BMI is expected to increase over time and to be proportionately greater for younger people (e.g., younger than age 65) than for older people.

We converted 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, $e(40)$. Online Resource 3 describes the procedures used to translate our estimates of age-specific death rate changes into their effects on life expectancy. As shown in Table 4, when converted into estimates of the effects on life expectancy at age 40, the estimated decline in life expectancy (mean,

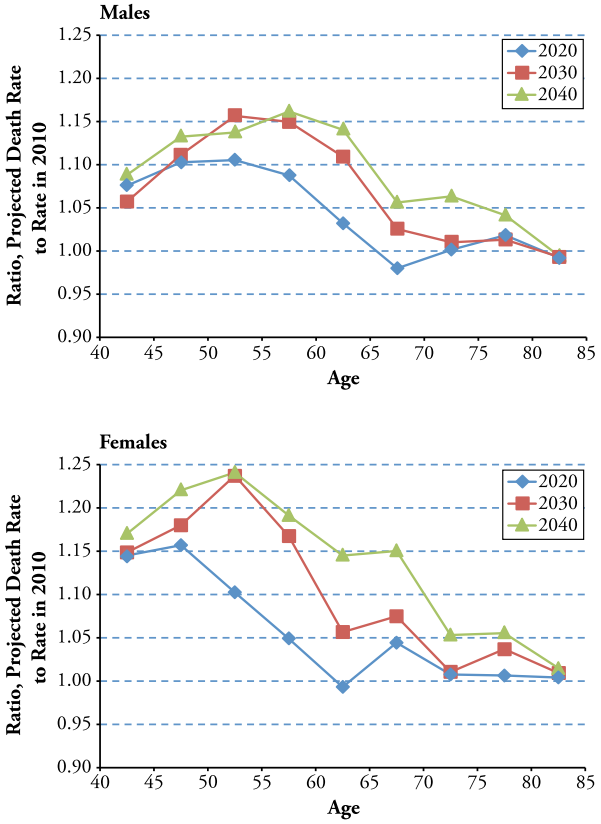

Fig. 3 Effects of projected trends in BMI on age-specific death rates

Table 4 Changes in life expectancy at age 40 resulting from changes in smoking and obesity (confidence intervals in parentheses)

	Changes in Smoking Alone		Changes in Obesity Alone		Changes in Smoking and Obesity	
Year	Males	Females	Males	Females	Males	Females
2015	0.26	–0.03				
	(0.07, 0.47)	(–0.52, 0.46)				
2020	0.54	0.04	–0.30	–0.27	0.24	–0.22
	(0.33, 0.76)	(–0.44, 0.53)	(–0.47, –0.15)	(–0.39, –0.15)	(–0.02, 0.52)	(–0.72, 0.28)
2025	0.81	0.15				
	(0.58, 1.08)	(–0.33, 0.63)				
2030	1.05	0.32	–0.54	–0.56	0.53	–0.21
	(0.78, 1.35)	(–0.15, 0.81)	(–0.79, –0.30)	(–0.81, –0.32)	(0.15, 0.90)	(–0.72, 0.31)
2035	1.31	0.62				
	(1.00, 1.67)	(0.20, 1.13)				
2040	1.54	0.85	–0.73	–0.82	0.83	0.09
	(1.18, 1.94)	(0.41, 1.38)	(–1.04, –0.42)	(–1.17, –0.47)	(0.37, 1.32)	(–0.45, 0.69)

males and females) resulting from rising obesity is 0.28 years by 2020, 0.55 years by 2030, and 0.78 years by 2030.

These estimated effects are smaller than those estimated by Stewart et al. (2009), who projected 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. (2009) 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. The mortality risks associated with obesity have declined in NHANES (Mehta and Chang 2011). On the other hand, unlike Stewart et al. (2009), we have introduced historical data on BMI at age 25. We will show that our estimated effects of increases in obesity on life expectancy would have been smaller had we not incorporated this information.

Projecting the Effects of Changes in Smoking

The risk of death from smoking is a function of many 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 important because of a long lag between smoking behavior and its effects on mortality. 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. Fortunately, another indicator of the health effects of smoking reflects the many dimensions of smoking: the death rate from lung cancer. As noted earlier, smoking is the overwhelming risk factor in death from lung cancer, with 90 % of male and 84 % to 85 % of female lung cancer deaths in the United States attributable to smoking (Oza et al. 2011). 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 more predictably arrayed by birth cohort rather than by period (Janssen and Kunst 2005; Preston and Wang 2006; Willets 2004; Yamaguchi et al. 2000).

Our estimates of the mortality effects of changes in 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. A second stage in the estimation of the effect of changes in smoking patterns is to translate projected changes in lung cancer mortality into changes in all-cause mortality.

Data for Analysis of Cohort Effects in Lung Cancer Mortality

Data on lung cancer deaths by age, sex, and period are drawn from annual volumes of Vital Statistics of United States for periods from 1940 through 1949, from the

Website of the World Health Organization/International Agency for Research on Cancer for 1950 through 1998, and from files of Underlying Cause of Death 1999–2009 on CDC WONDER Online Database for 1999–2009 (National Center for Health Statistics 2012). In this article, lung cancer refers to cancer of lung, bronchus, trachea, and pleura. The International Classification of Diseases (ICD) was used to identify lung cancer deaths. The entire study period of 65 years from 1945 to 2009 are covered by ICD from version 5 to version 10. The corresponding ICD version codes used for each individual time period are listed in the following table.

Year (ICD Version)	ICD Codes
1939–1948 (ICD-5)	47b-47f
1949–1957 (ICD-6)	162, 163
1958–1967 (ICD-7A)	162, 163
1968–1978 (ICD-8A)	162
1979–1998 (ICD-9)	162
1999–2009 (ICD-10)	C33, C34

Estimates of population size and counts of deaths from all causes combined are taken from the Human Mortality Database for 1933–2007. These data for 2008 and 2009 are drawn from National Center for Health Statistics (2012).[4]

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 (see Fig. 4). David Burns supplied us with unpublished estimates using the same methodology that incorporates data from three additional National Health Interview Surveys through 2001. We update the series using NHIS data through 2009. We convert 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 annual proportion of cohort members who were estimated to be current cigarette smokers.

For cohorts that had not reached age 40 in 2010, we estimate the future cumulative years of smoking by age 40 based on observed cumulative years smoked at younger ages. For this purpose, we use regressions predicting the mean cumulative years of smoking by age 40 with independent variables representing cumulative smoking indexes by age 35, age 30, age 25, and 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 1985–1990 cohort. The resulting series are presented in Fig. 4.

[4] Estimates pertaining to birth cohorts are created by organizing a data matrix in five-year age groups and five-year time blocks. In order to align cohort mortality data with cohort smoking data, we define five-year birth cohorts that are centered on birth years 1900–1904, 1905–1909, and so on. For example, mortality rates in the birth cohort of 1905–1909 were composed of death rates at ages 40–44 in 1947–1951, death rates at ages 45–49 in 1952–1956, and so on. The final mortality observations for cohorts still alive are death rates in 2007–2009.

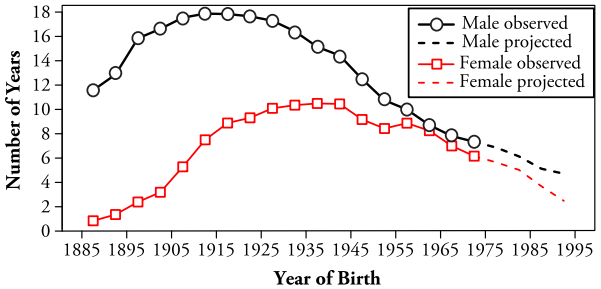

Fig. 4 Mean number of years spent as a cigarette smoker before age 40 by cohort. *Sources*: Data are derived from the National Health Interview Survey

Methods for Projecting the Mortality Effects of Smoking

Our initial goal is to identify how lung cancer mortality varies 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 relate lung cancer mortality to a cohort smoking variable that had proven useful in prior research on 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^c for cohort c. For each sex, we estimate an equation of the form

$$\ln(M_a^c) = A + \beta_a X_a + \beta_s \ln(S^c) + \varepsilon, \tag{1}$$

where M_a^c is the lung cancer death rate at age a in cohort c, X_a is an indicator of age category a, β_a is the coefficient of age category X_a, and β_s is the coefficient of $\ln(S^c)$. We estimate this model using negative binomial regression on death counts on all observations at ages 40–44 to 80–84 for periods beginning in 1947–1951. This starting period was chosen because it produced the best fit to actual death rates in 2009 among all potential start years from 1937 to 1987. The coefficients of $\ln(S^c)$ are 1.279 for males and 0.929 for females. Greater sensitivity of males than females to their respective smoking histories was also found by Preston and Wang (2006) and Wang and Preston (2009) based on all-cause mortality. It is also a common finding in prospective cohort studies, perhaps because women smokers on average consume fewer cigarettes per day, inhale less frequently, and smoke cigarettes lower in tar content (Thun et al. 1997). 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.[5] Using negative binomial regression on death counts, we estimate the parameters of a straightforward age/cohort model,

$$\ln(M_a^c) = A + \beta_a X_a + \beta_c X_c + \varepsilon, \tag{2}$$

[5] 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 (Fienberg 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. A second reason for not invoking an age/period/cohort model is that we had no strong hypothesis about period effects on lung cancer mortality, given that we considered such mortality to be primarily a function of cohort smoking histories.

where M_a^c is the lung cancer death rate in cohort c at age a, β_a and β_c are the coefficients of age category a and cohort c, and X_a and X_c are indicators of age and cohort membership.

Figure 5 plots cohort effects estimated from Eq. (2) and the mean number of years of smoking before age 40 for each cohort, used to estimate Eq. (1). The two series for women obviously track each other closely, including a bump for female cohorts born 1955–1964. For men, both series are hill-shaped, although the peak of the smoking series occurs earlier than the peak cohort coefficient. Figure 5 illustrates that cohort effects in lung cancer are dominated by smoking histories.

Our projections are based on Eq. (1), which uses the smoking series. A main advantage of this approach is that we are able to observe smoking behavior for cohorts as young as ages 15–19. In contrast, the cohort coefficients from Eq. (2) are not robustly estimated until a cohort has reached the 40s, when substantial numbers begin to die from lung cancer. Furthermore, the smoking-based analysis produces predicted death rates in 2009 that are much closer to the actual death rates in that year than the analysis using cohort coefficients, which significantly underestimate mortality for older cohorts.

We test the predictive validity of Model 1 by estimating the parameters of the model on data through 1995–1999 and using the age and cohort coefficients to project mortality in 2005–2009. Comparing the projected mortality level to the actual level in the prime ages of 50–84, the mean error in projected rates is 1.54 % for males and 1.17 % for females. The mean absolute error is 4.64 % for males and 5.64 % for females. A prediction of "no change" between 1995–1999 and 2005–2009 produces a mean error of –28.23 % for males (i.e., an overprediction) and –9.65 % for females. The mean absolute errors for a

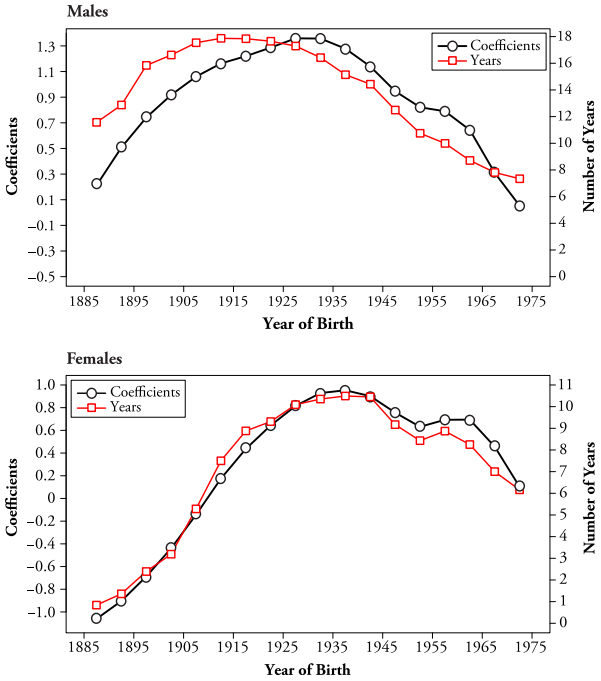

Fig. 5 Cohort coefficients predicting lung cancer mortality and cumulative cohort smoking by age 40. *Sources*: Data on smoking are derived from the National Health Interview Survey. Coefficients are derived from age/cohort model of lung cancer mortality

no-change prediction are 28.23 % for males and 14.36 % for females. These are obviously many times greater than errors produced by our model, which performs well in predicting changes in lung cancer mortality between 1995–1999 and 2005–2009. We conclude that our model proves effective in out-of-sample prediction. It is worth noting that at these ages, a 10 % error in all age-specific death rates would produce an error of less than 3 % in life expectancy (Keyfitz and Golini 1975).

Translating Changes in Lung Cancer Mortality into Changes in All-Cause Mortality

Although lung cancer mortality serves as an excellent marker of the health effects of smoking, lung cancer does not account for a majority of deaths attributable to smoking. Cardiovascular diseases, other cancers, 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) converted observed lung cancer death rates into an estimate of smoking "prevalence" by referring to the difference between lung cancer death rates for smokers and nonsmokers in Cancer Prevention Study II (CPS-II). They then used this estimate of smoking prevalence to estimate the risk attributable to smoking for other smoking-related causes of death by employing the cause-specific relative risks for smokers versus nonsmokers from CPS-II.

The second method also uses lung cancer mortality as the basic indicator of the damage caused by smoking (Preston et al. 2010, 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 data set of 21 countries covering the period 1950 to 2006, including 9.9 billion person-years of exposure and 284 million deaths. In addition to lung cancer mortality, the statistical model includes age, sex, period, and country effects as well as interactions among them.

The two methods of translating lung cancer mortality into all-cause mortality give very similar results (Preston et al. 2010). 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. This assumption appears reasonable: using the Peto et al. (1992) approach, Oza et al. (2011) found that the estimated number of deaths attributable to smoking differed by only 1.7 % when cause-specific lag structures were incorporated compared with when they were not.

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 et al. (2011).[6] Later, we explore the sensitivity of results to this choice of translation factors.

Uncertainty Analysis

We analyze uncertainty in our estimates of the effects of smoking on change in life expectancy using a bootstrapping procedure similar to that used in the analysis of obesity.

[6] Preston et al. (2011) did not estimate coefficients for ages below 50. We assume that the coefficients for ages 50–54 apply to ages 40–49. Because coefficients decline with age, this choice probably produces an underestimate of smoking-attributable deaths, but there are very few smoking-related deaths in the age interval 40–49, so results are little affected by this assumption.

We generate 1,000 sets of bootstrapped coefficients for the lung cancer mortality and Preston/Glei/Wilmoth models. We then apply the 1,000 sets of age-specific lung cancer mortality rates to the 1,000 sets of PGW coefficients to calculate mortality from all causes and life expectancies at age 40. The 2.5 and 97.5 percentile values from the simulated life expectancy estimates are extracted as the 95 % confidence interval. The uncertainty estimates for the combination of smoking and obesity are obtained in a similar manner.

Results

Figure 6 presents the results of the smoking analysis. Male age-specific death rates are expected to decline at every age throughout the projection period. The heaviest-smoking male cohorts are already aged 80+ in 2010, and the effect of persistent declines in smoking from cohort to cohort is to produce a steady decline in relative death rates as time advances. In contrast, female rates are expected to rise in the oldest age intervals during the early years as heavier-smoking cohorts replace lighter-smoking ones. Projected male declines are larger than female declines in nearly all comparisons, reflecting the more gradual changes in cohort smoking propensities among women.

Table 4 converts the age-specific projections of mortality change into estimates of the effect on life expectancy at age 40. Males show a relatively steady improvement in life

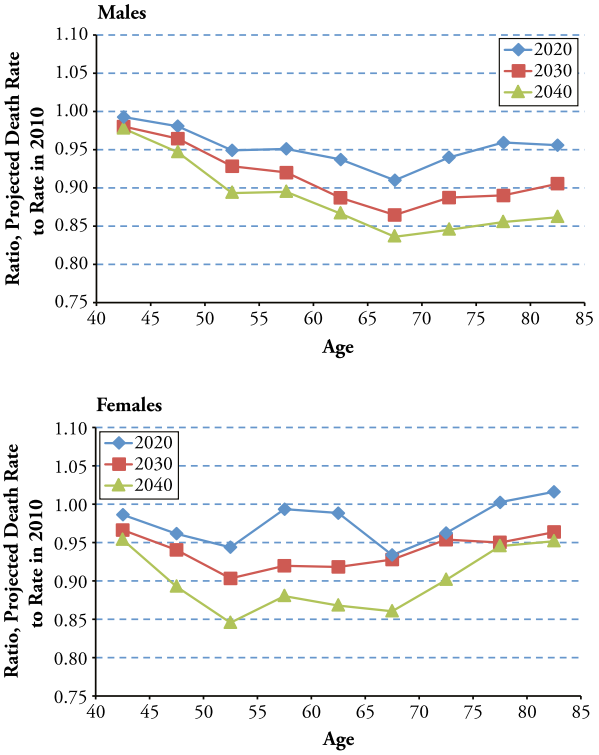

Fig. 6 Effects of projected trends in smoking on age-specific death rates

expectancy from smoking reductions and a total gain of 1.52 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 continue moving into the prime ages of dying. There is projected to be virtually no gain in female life expectancy as a result of smoking reductions between 2010 and 2020. However, female gains accelerate after 2025 as the heaviest smoking cohorts begin to disappear. By 2040, women are projected to have gained 0.85 years in $e(40)$ from smoking reductions.

Two other projections have been made of anticipated changes in mortality as a result of changing smoking patterns. Wang and Preston (2009) added a cohort smoking term to the conventional Lee-Carter model of mortality change from all causes of death combined. They summarized 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 estimated that reductions in smoking will increase the probability of male survival by 15.8 % and of female survival by 7.2 %. In the present set of projections, changes in this probability between 2010 and 2035 are 13.4 % for males and 4.7 % for females. The proportion of lung cancer deaths attributable to smoking is in the range of 85 % to 90 % (Oza et al. 2011), whereas the proportion of all-cause deaths attributable to smoking is in the neighborhood of 20 % (Mokdad et al. 2004, 2005). Accordingly, mortality from lung cancer is a much more sensitive indicator of the damage from smoking than is all-cause mortality. As a result, we believe the present estimates are more reliable.

Stewart et al. (2009) also projected 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 did not differentiate between the sexes. They estimated that in a 15-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 0.80 years for males and 0.15 years for females, with an average gain of 0.47 years.[7] Although 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 0.28 years compared with their 0.31 years over the preceding 15-year period. Thus, our results appear reasonably consistent with theirs over this short projection period.

Combining Obesity and Smoking

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 may be relevant. One refers to behavioral associations 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 et al. (1995) 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)

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

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, and there is no obvious reason why the relation between the prevalence of the two risk factors over the next decades would differ from that in the recent past.

The second type of interaction is interaction between the two mortality 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 number of deaths attributable to obesity. On the other hand, such an interaction would imply that the mortality risk from smoking is lower among the obese, which would result in a reduction in the number of deaths attributable to smoking as obesity increases. Because the prevalence of the two risk factors is moving in opposite directions, any such interaction would produce effects that are at least partially offsetting. Unfortunately, our research design does not allow us to investigate such interactions.

Assuming independence between the mortality risks of obesity and smoking, we multiply the effects of changes in obesity and smoking presented in Figs. 3 and 6. Results are shown in Fig. 7. The preponderant downward slope of both sets of results when smoking and obesity are considered independently is accentuated when the effects are multiplied. The obesity effect dominates the smoking effect below age 60, where death rates are projected to be higher than baseline throughout the projection

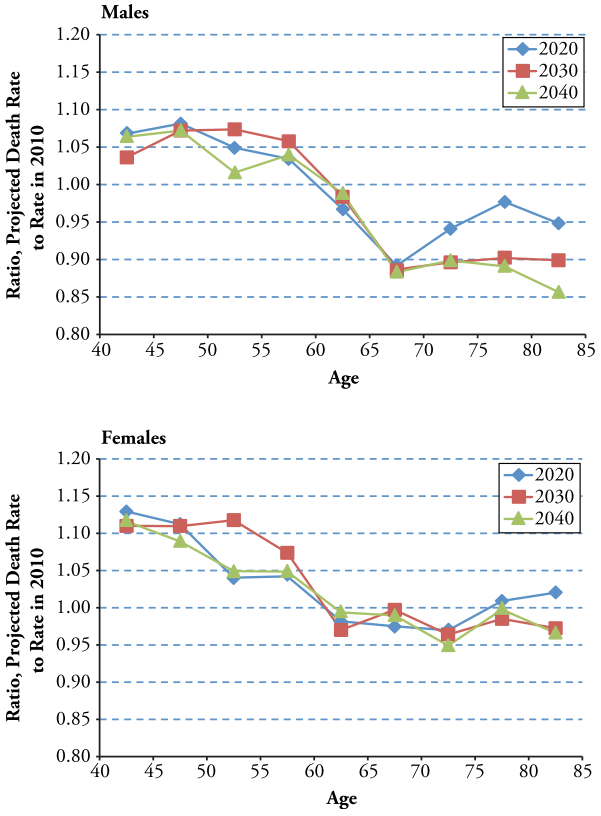

Fig. 7 Combined effects of projected trends in smoking and obesity on age-specific death rates

period. Above age 60, the smoking effect is clearly dominant and produces reductions in mortality.

Table 4 summarizes these changes in the form of life expectancy at age 40. The combined effect of changes in smoking and obesity is expected to produce steady improvements in male life expectancy through 2040, with a total gain of 0.83 years by that date. On the other hand, women's life expectancy is expected to be lower as a result of the combined changes through 2030. Thus, the combined effects of changes in smoking and obesity are expected to maintain the pattern of reductions in the female advantage in life expectancy that has been evident in national life tables since 1979. By 2040, life expectancy is anticipated to be 0.09 years higher for females as a result of these combined behavioral changes.

Sensitivity Analyses

We perform six analyses of the effect of changes in procedures on outcomes. In each case, we estimate the effect of an alternative procedure on age-specific death rates and convert those rates into estimated effects on life expectancy at age 40. Results for life expectancy at age 40 are shown in Table 5, where values 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 with smoking results.

Table 5 Sensitivity of results to changes in procedures

	Effect on Life Expectancy at Age 40 Relative to Main Projection[a]					
	2020		2030		2040	
Change in Procedure	Male	Female	Male	Female	Male	Female
Use of Measured Data on Obesity at Age 25	–0.038	–0.061	–0.067	–0.095	–0.059	–0.103
No Future Growth in Obesity Among Initial Cohorts of 25- to 34-Year-Olds	0.000	0.000	0.004	0.009	0.029	0.040
Use of Mortality Rates With No Control Except Age and Sex	0.017	0.010	0.026	0.015	0.026	0.012
Use of Mortality Rates Without Inclusion of BMI at Age 25	0.026	0.056	0.095	0.164	0.199	0.289
No Exclusion Criteria Applied to Mortality Modeling	0.046	0.072	0.099	0.141	0.145	0.202
Use of Alternative Series Translating Lung Cancer Into All-Cause Mortality	–0.067	0.012	–0.173	–0.107	–0.322	–0.360

[a] A positive value means that the alternative procedure resulted in a gain in projected life expectancy relative to the main procedure.

Five of the six sensitivity analyses are made with respect to obesity. In our baseline analysis, we use retrospective data as the basis for projecting age-25 BMI distributions for individuals aged 25–84 in 2010–2040. Rather than using self-reported age-25 data, the first sensitivity analysis uses *measured* data derived (NHES I; 1959 from National Health Examination Survey, Cycle I –1962), NHANES I–III (1970–1974, 1976–1980, and 1988–1994), and continuous NHANES (1999–2010) as the basis for projecting future age-25 BMI distributions. Details are presented in Online Resource 2. Table 5 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 0.11 years.

Second, we examine the sensitivity of results to the projected changes in current BMI at ages 25–34 as well as changes in age-25 BMI. Instead of extrapolating recent trends in the BMI distribution at these ages (as in the main analysis), in the sensitivity analysis, we assume the distribution to remain constant at its level in 2010. Table 5 shows that this change has almost no effect on results because so much of the dying is concentrated at older ages that are unaffected by such a compositional change at younger ages.

The other three changes in obesity procedures pertain to the regression equation linking mortality to BMI. Model parameters are available from the authors upon request. To show the impact of controlling educational attainment, race/ethnicity, and smoking in the mortality estimates, we repeat the mortality analysis for obesity without these controls. Table 5 shows that results are insensitive to this change in procedure: the effect on $e(40)$ never reaches 0.03 years for either sex.

The next sensitivity analysis examines the effect of omitting information about age-25 BMI. We reestimate the regression equation predicting mortality based on BMI after excluding terms representing BMI at age 25. Table 5 shows that including age-25 information has an important effect on results. By 2040, losses in life expectancy are 0.20 years greater for men and 0.29 years greater for women when age-25 BMI is included than when it is not. These represent increases in the impact of obesity of 37 % for men and 54 % 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 final sensitivity analysis involving obesity uses death rates by BMI estimated on a data set that does not exclude those with emphysema and smoking-related cancers at baseline and does not exclude the first three years of observation. The results demonstrate what would happen to our estimates if we had not made efforts to eliminate the biases produced by reverse causation. The effect is sizable: the use of the exclusion criteria raises the estimated impact of changes in obesity on life expectancy by 0.15 years for men and 0.20 years for women by 2040.

We interpret these results as evidence of the importance of correcting for reverse causation. At the same time, they demonstrate considerable sensitivity of our projections to the set of mortality rates by BMI, about which there can be substantial disagreement (Preston et al. 2013).

The sensitivity analysis involving smoking uses an alternative set of relations between lung cancer mortality and all-cause mortality. The main results presented in this article are based on relations estimated across 21 countries from 1950 to 2006. Fenelon and Preston (2012) instead estimated coefficients relating lung cancer to all-

cause mortality that are based on variations across the 50 states of the United States between 1996 and 2004; coefficients predicting mortality from other causes of death on the basis of lung cancer mortality were very similar for men to those in Preston et al. (2011), but they were lower for women at younger ages.[8]

Results in Table 5 show that the sensitivity of results is minor for the first 10 years of projection, modest for the second 10 years, and sizable by 2040. Of the projected 1.52 years of gain in life expectancy from reductions in smoking by 2040 for males in Table 4, 0.32 years would be eliminated if the alternative relations were used. Of the 0.85-year gain for women, 0.36 years would be eliminated if the alternative relations are used. The alternative results have the virtue of being based on contemporary relations in the United States, 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 by 2040.

Conclusion

The combined effects of past and future changes in obesity and smoking are likely to result in an improvement in U.S. 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 forecast 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. We find relatively small net effects of the two forces between 2010 and 2020; more precisely, gains in life expectancy for men are largely offset by 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. (2009) reflect a 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.

Are the changes that we have projected large or small? One useful metric is provided by projections made by the SSA (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, somewhat smaller gains than forecast by most other analysts (Wilmoth 2005). Relative to projections by the SSA, the mean of male and

[8] Neither approach estimated a coefficient for ages 85+. Preston et al. (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).

female gains that we estimate from reduced smoking (1.54 years among men and 0.85 years among women) would themselves account for about one-half of the projected mean gain in life expectancy. These gains will be partially offset by the consequences of increases in obesity. As a percentage of the life expectancy increases projected by the SSA, growing obesity is expected to impose a penalty of 29 % for men and 38 % for women. These two behaviors clearly exert a major influence on American mortality and warrant continued monitoring and analysis.

Acknowledgements This research was supported by National Institute of Aging Grant R01AG040212, and by the U.S. Social Security Administration through Grant #5RRC08098400-04-00 to the National Bureau of Economic Research (NBER) as part of the SSA Retirement Research Consortium. The findings and conclusions expressed are solely those of the authors and do not represent the views of SSA, any agency of the Federal Government, or the NBER. We are grateful to Haidong Wang for technical assistance and to Christopher Ruhm, Andrew Fenelon, Douglas Ewbank, Lucia Tiererova, and reviewers for this journal for their comments and suggestions.

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