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# Is the U.S. Population Behaving Healthier?

David M. Cutler, Edward L. Glaeser, and
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Understanding changes in population health is a key input into public and private decision making. People who live longer have more years of life to enjoy, but also need to prepare for more older years, through increased saving and possibly delayed retirement. Rational decision makers will take into account forecasts of longevity and quality of life in making their work and savings decisions. Public policy must account for this as well. Every additional year of life after age sixty-five is associated with about $15,000 of Social Security and medical care spending, and years spent disabled result in substantially greater medical spending than years spent without disability (Trends in Health and Aging 2007).

Health outcomes are a product of several inputs. Peoples' behaviors and genetic predisposition put them at risk for disease. The medical system then alleviates or treats these risks. Distinguishing the role of behavioral risk factors from medical care is important for several reasons. One reason is the impact on medical spending. Improved behaviors generally lower medical spending, at least in the short term, while treating adverse risk profiles increases costs. Thus, knowing whether health behaviors are

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improving is important in forecasting medical costs. In addition, behavioral trends are essential in predicting future disease burden. A population that behaves in a healthier way will have higher quality of life compared to one with a more adverse behavioral profile, even given length of life. Finally, changes in behaviors are a good guide to the "demand" for better health, which can be used to develop models of health demand and supply. In this chapter, we consider what has happened to the population's health behaviors over time and consider various scenarios for trends in the future.

Past trends in behavioral risk factors have not been in a common direction. Some measures of population risk have improved markedly, while others have deteriorated. Smoking rates have fallen by more than a third since 1960 (Anonymous 1999), and alcohol consumption has declined by 20 percent since 1980 (Lakins, Williams, and Yi 2006), both leading to better health. Demographically, the population is better educated, and better-educated people live longer than less-educated people (Elo and Preston 1996). On the other hand, obesity rates have doubled in the past two decades (Flegal et al. 2002), and diabetes has increased as a result (Gregg et al. 2005). Further, the population has a higher share of minority groups, for whom life expectancy is lower. The net impact of these risk factor trends on population health expectations is uncertain (Preston 2005).

Our analysis has two parts. We start by aggregating these different health trends into a single measure of population risk. We focus on the most common risk factors: smoking, drinking, obesity, hypertension, high cholesterol, and diabetes. We weight the different risk factors by their impact on predicted ten-year mortality, as determined through multiple regression analysis. We show that overall health trends in the past three decades have improved markedly. For the entire population aged twenty-five and older, the age-adjusted probability of dying in ten years, conditional on the same level of medical care, fell from 9.8 percent in the early 1970s to 8.4 percent around 2000, a 14 percent reduction. The largest contributors to this trend were reductions in smoking and improved blood pressure control.

The second part of our analysis considers the impact of a continuation of future trends. Our conclusions here are not as rosy. We show that if current obesity trends continue, the population mortality risk could increase, even with continued reductions in smoking. We estimate that about a third of the past gains would be reversed within twenty years. The increase in obesity is the proximate cause of this. But even given the increase in obesity, the health impact would be substantially blunted if more people took medication to control blood pressure, cholesterol, and diabetes.

Our chapter has five sections. The first section discusses important risk factors; the second section shows trends in risk factors. The third section evaluates mortality risk from the early 1970s through the early 2000s. The

fourth section then considers alternative scenarios for future risk trends. The last section concludes.

## 12.1 Health Behaviors

We are interested in measuring the population's health profile over time. Health is a product of many features: the individual risk factor profile; the disease environment; and the impact of medical care. We focus on individual behaviors because that is (perhaps) the easiest to forecast and tells us the most about the demand for health.

To understand our analysis, consider a simple model. Individuals live for up to two periods; health is defined as the probability that a person survives to period 2. If alive, people get consumption $c$. For simplicity, we assume no borrowing or lending, and no discounting. The lifetime utility function is then:

$$V = U(c) + \pi(b) \cdot U(c), \tag{1}$$

where $\pi(b)$ is the probability of survival to period 2, depending on behavior $b$. Define the behavior as improving health, so $\pi' > 0$. Action $b$ has a cost, $p$ per unit. The cost may be monetary (the cost of a gym membership) or psychological (the implicit cost of dieting). In equilibrium, people will consume item $b$ until the marginal benefit is equal to the marginal cost. This is given by:

$$\pi' \cdot U(c) = p. \tag{2}$$

Equilibrium $b$ will change over time for two reasons. The first is that the population becomes richer. This shows up as increasing $c$. As long as people are not sated in goods consumption, increases in income will raise the utility of living longer and, hence, lead to a greater investment in $b$. The second change is in the cost of better health. This cost may increase or decrease over time. To the extent that $b$ involves hiring people (e.g., a personal trainer), and all wages increase in the economy, the cost of $b$ will increase. Some aspects of technical change will also increase $b$. For example, technology that makes food more readily available will increase the psychological cost of denying ourselves food. Cutler, Glaeser, and Shapiro (2003) suggest that this is why obesity has increased over time. In other cases, $b$ might fall over time, as we develop new medications or ways of improving health.

The net impact of economic changes on health behaviors is thus indeterminate, depending on the demand for better health relative to the cost of health improvements.

Empirically, we delineate the risk factors we consider into four groups: demographics, genetics, behaviors, and biological factors. The relations between these are shown in figure 12.1. Demographic factors included age,

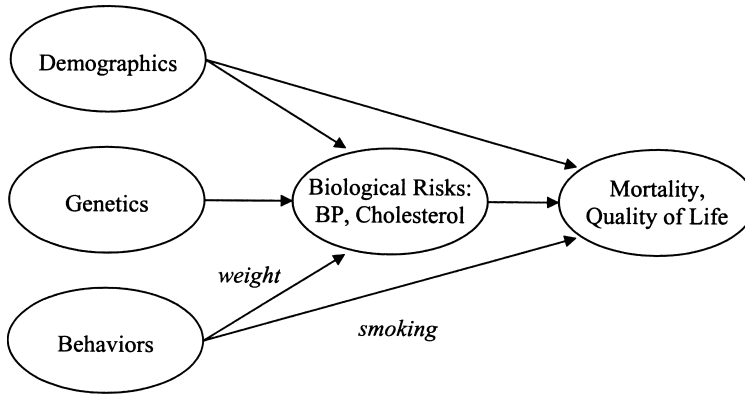


**Fig. 12.1 Conception of risk factors affecting health**

sex, race, and education. Age, sex, and race are standard risk measures. Education is strongly related to health, although the reason for this is unclear (Cutler and Lleras-Muney 2008). Because some evidence suggests that the education effect is causal (Lleras-Muney 2005; Oreopoulos 2007; Arendt 2005; Spasojevic 2003), we consider this as a demographic risk factor. Of course, to the extent that education reflects other underlying characteristics of people such as position in the social hierarchy (Wilkinson 1996; Link and Phelan 1995) or discount rates (Fuchs 1982), we will be overstating the impact of educational changes on health.

A variety of genetic factors predispose people to disease. The data that we have do not render genetic profiles. Because it is unlikely that the population's genetic profile would change markedly in a few decades—particularly controlling for gender and race—we do not consider the possible impact of genetic changes.

There are a number of behavioral risk factors that are important for health. Mokdad et al. (2004) rank the impact of risk factors on mortality; our results largely confirm these rankings. The most important behavioral risk factor is smoking. Mokdad et al. estimate that smoking accounts for about 435,000 deaths annually. Obesity is second in importance, though the impact is controversial (Flegal et al. 2005; Willett et al. 2005). The impact of obesity on mortality ranges from about 100,000 deaths per year to about 400,000 deaths per year.

Other behaviors are of much less quantitative importance than smoking and obesity. Excessive alcohol use is the third important risk factor, accounting for 85,000 deaths. Remaining risk factors include exposure to microbial agents (75,000 deaths) or toxic agents (55,000 deaths), motor vehicle accidents (43,000 deaths), guns (29,000 deaths), sexual behaviors (20,000 deaths), and illicit drug use (17,000 deaths). Many of these latter risk factors disproportionately affect the young. For purposes of Social Se-

curity and Medicare, our focus is primarily on the elderly. Thus, we limit our analysis of behavioral risks to smoking, obesity, and alcohol use.

Finally, we consider two biological risk factors: blood pressure and cholesterol. Both blood pressure and cholesterol are products of other behaviors, most importantly obesity. We consider this link extensively in our forecasting analysis.

Not all important risk factors are included in our analysis of risk. For example, the composition of diet matters as well as overall caloric intake. Among biological risks, the most important omissions are hemoglobin A1c (i.e., diabetes status) and some of the more novel risk factors (such as C-reactive protein or albuminuria). None of these risk factors were measured in the early National Health and Nutrition Examination Survey (NHANES).

## 12.2 Data

Risk factor analysis requires data on physical measures of the population, not just self-reports. Not everyone with high blood pressure knows they are hypertensive, for example, and the share of people with this knowledge will change over time. In the United States, the leading survey with both physical examination and laboratory measurements is the NHANES. More detail on the survey design and operation is reported elsewhere (Miller 1973; NCHS 2006).

We use two NHANES surveys, the first from 1971 to 1975 (NHANES I), and the second from 1999 to 2002 (NHANES IV). Our analysis began with the NHANES I because that is the first population health survey that asked about smoking status, a key variable in health risk.

In each case, our initial sample is the population aged twenty-five to seventy-four. The upper age restriction matches the sampling frame of NHANES I. To focus on the elderly and nonelderly population in specific, we also consider the population aged fifty-five and older.

Table 12.1 shows the characteristics of the sample in the two time periods. The first set of columns are for the entire population, and the second set of columns are restricted to the population aged fifty-five and older. After eliminating people with missing risk factor information, our full age sample includes 6,764 respondents to NHANES I and 6,255 respondents to NHANES IV. The subset of older respondents is about one-third the size.

Age was categorized into ten-year age groups beginning at age twenty-five. Race was defined as white, black, or other. Education was divided into three groups: less than a high school degree; a high school degree; and at least some college. Table 12.1 shows that these risk factors moved in the expected direction over time. In particular, the share of people with at least some college education doubled over those three decades.

**Table 12.1 Characteristics of the sample (%)**

| Risk factor | Entire population | | Population 55+ | |
|---|---|---|---|---|
| | NHANES I 1971–1975 (n = 6,764) | NHANES 1999–2002 (n = 6,225) | NHANES I 1971–1975 (n = 2,453) | NHANES 1999–2002 (n = 2,188) |
| Female | 52.5 | 51.1 | 54.1 | 51.9 |
| Race | | | | |
| White | 89.0 | 85.8 | 90.8 | 88.6 |
| Black | 10.0 | 9.9 | 8.5 | 8.0 |
| Other race | 1.0 | 4.3 | 0.7 | 3.5 |
| Married | 79.0 | 64.9 | 72.5 | 70.1 |
| Education | | | | |
| <High school | 34.4 | 19.8 | 55.3 | 31.7 |
| High school | 37.2 | 24.9 | 26.0 | 27.1 |
| At least some college | 28.4 | 55.3 | 18.6 | 48.8 |
| Smoking | | | | |
| Current smoker | 40.3 | 24.8 | 28.5 | 16.3 |
| Former smoker | 21.2 | 26.0 | 27.9 | 40.6 |
| Never smoker | 38.5 | 49.2 | 43.6 | 43.1 |
| Drinking | | | | |
| Heavy drinker | 6.7 | 4.4 | 5.8 | 4.5 |
| Light drinker | 72.3 | 65.3 | 60.3 | 55.1 |
| Nondrinker | 20.9 | 30.3 | 33.9 | 40.5 |
| Body mass index (BMI) | | | | |
| Underweight, BMI $< 18.5$ | 2.8 | 1.7 | 2.9 | 0.9 |
| Optimal weight, $18.5 \leq$ BMI $< 25$ | 47.7 | 30.4 | 40.1 | 25.0 |
| Overweight, $25 \leq$ BMI $< 30$ | 34.6 | 34.7 | 37.5 | 36.4 |
| Obese, $30 \leq$ BMI | 14.8 | 33.2 | 19.5 | 37.7 |
| Blood pressure | | | | |
| Normal blood pressure | 22.4 | 43.4 | 8.9 | 22.5 |
| Prehypertension | 38.2 | 38.9 | 28.1 | 43.6 |
| Stage 1 hypertension | 23.6 | 13.1 | 32.4 | 22.3 |
| Stage 2 hypertension | 15.7 | 4.6 | 30.6 | 11.7 |
| Cholesterol | | | | |
| Normal cholesterol | 35.4 | 47.4 | 19.6 | 35.6 |
| Borderline high | 34.9 | 34.4 | 34.7 | 41.8 |
| High | 29.7 | 18.3 | 45.7 | 22.6 |

*Note:* NHANES is the National Health and Nutrition Examination Survey.

Following standard practice in the literature, smoking status was divided into three groups: current smokers, former smokers, and never smokers. Smoking status was determined by responses to two questions, "Have you ever smoked at least 100 cigarettes in your entire life?" and "Do you smoke cigarettes now?" The share of current smokers fell by a third over the time period, from 40 percent in the early 1970s to 25 percent around 2000. Two-thirds of this was people who never started smoking, and one-third was people quitting.

Drinking status was divided into heavy drinkers, light drinkers, and nondrinkers. In NHANES I, drinking status was assessed with three questions. Nondrinkers were those who answered "no" to the question, "During the past year have you had at least one drink of beer, wine, or liquor?" Among those who answered "yes," subsequent questions included "How often do you drink?" and "When you drink, how much do you usually drink over 24 hours?" Heavy drinkers were those who drink three or more drinks over twenty-four hours and reported drinking "everyday" or "just about everyday." The next possible response was "about 2 or 3 times a week." In NHANES IV, nondrinkers were defined as those who responded "zero" to the question, "In the past 12 months, how often did you drink any type of alcoholic beverage?" A subsequent question asked people, "In the past 12 months, on those days that you drank alcoholic beverages, on the average how many drinks did you have?" Heavy drinkers were those who reported drinking three or more drinks at least four times per week (i.e., four or more times per week, sixteen or more times per month, or 208 or more times per year). Both heavy and light alcohol use declined over time. Heavy drinking fell from 7 to 4 percent of the population; light drinking fell from 72 to 65 percent.

Body mass index (BMI) was based on direct measurement of height and weight. In accordance with conventional guidelines (National Institutes of Health 1998), we classified respondents as underweight ($BMI < 18.5$), normal weight ($18.5 \leq BMI < 25$), overweight ($25 \leq BMI < 30$), and obese ($30 \leq BMI$). The largest change in weight has been the shift from healthy weight to overweight. Overweight and obesity were 49 percent of the population in the early 1970s; today, they are 68 percent. At the other end of the scale, fewer people are underweight now than in the past (2 percent versus 3 percent).

Blood pressure and total cholesterol were measured according to standard protocols used in the medical examination component of each survey (Burt et al. 1995; Hajjar and Kotchen 2003; Carroll et al. 2005). Blood pressure was divided into four groups following the recommendations of the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VII): normal blood pressure (systolic blood pressure [SBP] $\leq$ 120 mmHG and diastolic blood pressure [DBP] $\leq$ 80 mmHG); prehypertension ($120 \leq SBP <$ 140 or $80 \leq DBP < 90$); stage 1 hypertension ($140 \leq SBP < 160$ or $90 \leq DBP < 100$); and stage 2 hypertension ($160 \leq SBP$ or $100 \leq DBP$). Cholesterol levels were divided into three groups based on the recommendations of the Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (NCEP 2001): normal cholesterol (total cholesterol $< 200$); borderline high cholesterol ($200 \leq$ total cholesterol $< 240$); and high cholesterol ($240 \leq$ total cholesterol).

Even with the increase in obesity, substantial gains have been made in blood pressure and cholesterol control. The share of people with stage 2 hypertension fell from 16 percent of the population in the early 1970s to 5 percent around 2000. The share with stage 1 hypertension fell nearly in half as well. Rates of high cholesterol declined by over one-third, almost certainly a result of improved medications.

## 12.3 The Health Profile, 1971–1975 versus 1999–2002

To gauge the impact of these differing health trends, we need to weight the various risk factors. The optimal weights to use will depend on the question being asked. One could use longevity weights, quality of life weights, or medical spending weights. In practice, the NHANES does not have data on medical spending, and quality of life data are not great. Thus, we use mortality weights.

To estimate the impact of these risk factors on mortality, we use the epidemiological follow-up conducted as part of the 1971 to 1975 NHANES. Epidemiological follow-ups were conducted at periodic intervals after the initial survey, going into the 1990s. We estimated a logit model for death from any cause within the ten years subsequent to the initial survey. We choose ten years to get the long-term impact of these risk factors, but to avoid a situation where most everyone will have died. Previous evidence shows that prediction equations from NHANES are broadly similar to those from other data sources such as the Framingham Heart Study, with the possible exception of increased importance of smoking and diabetes in NHANES data (Liao et al. 1999; Leaverton et al. 1987).

Table 12.2 shows the odds ratios for death in the subsequent ten years. The coefficients are all in the expected direction, and most are statistically significant. Among demographic factors, blacks are more likely to die than whites (OR = 1.4; $p = .010$), and marriage is protective of future longevity (OR = 0.68; $p = .001$). People with less than a high school degree have 27 percent higher mortality than people with a high school degree ($p = .036$).

Behavioral risk factors are also important. Being a current smoker increases the odds of death in the next ten years by 113 percent ($p < .001$). Heavy drinking is associated with higher mortality, and light drinking is associated with lower mortality; the net impact is thus unclear, though as we show in the following, these changes are relatively small.

Without controlling for hypertension or high cholesterol, obesity increases the odds of death by 44 percent ($p = .018$); however, this drops to 28 percent and is no longer statistically significant ($p = .112$) once blood pressure and cholesterol are controlled for. This finding parallels other research from the Framingham Heart Study, which does not include obesity in the risk equations (Anderson et al. 1991; Wilson et al. 1998), and data showing that the impact of obesity on mortality is declining in more recent

**Table 12.2 Effect of risk factors on 10-year mortality**

| Variable | Odds ratio | Standard error |
|---|---|---|
| Race (relative to white) | | |
| Black | 1.402** | .195 |
| Other race | .245 | .221 |
| Married | .682** | .077 |
| Education (relative to high school graduate) | | |
| <High school | 1.269** | .144 |
| At least some college | 1.062 | .191 |
| Smoking status (relative to never smoker) | | |
| Current smoker | 2.126** | .250 |
| Former smoker | 1.233 | .165 |
| Drinking status (relative to nondrinker) | | |
| Heavy drinker | 1.021 | .175 |
| Light drinker | .771** | .094 |
| Body mass index (BMI; relative to optimal) | | |
| Underweight, BMI < 18.5 | 2.408** | .582 |
| Overweight, 25 ≤ BMI < 30 | .762** | .089 |
| Obese, BMI ≥ 30 | 1.278 | .197 |
| Blood pressure (relative to normal) | | |
| Prehypertension | .904 | .166 |
| Stage 1 hypertension | 1.131 | .201 |
| Stage 2 hypertension | 1.535** | .289 |
| Cholesterol (relative to normal) | | |
| Borderline high | 1.029 | .130 |
| High | 1.150 | .148 |
| *N* | 6,525 | |

*Source:* Data from National Health and Nutrition Examination Survey I.
*Note:* The regression includes 10-year age dummy variables interacted with gender.
**Significant at the 5 percent level.

surveys (Flegal et al. 2005). Indeed, it is likely that some of the obesity effect we find would be reduced still further if we were able to control for diabetic status. Being underweight is associated with significantly higher mortality, likely because of the loss of lean body mass (and, therefore, weight) associated with chronic or severe illnesses (Willett et al. 2005).

Both hypertension and high cholesterol are associated with substantially increased risk. People with stage 2 hypertension have a 54 percent increase in risk ($p = .023$) above those with normal blood pressure. High cholesterol is associated with a 15 percent higher mortality risk, though this is not statistically significant ($p = .277$).

We use these coefficients to estimate the mortality risk for every person in the 1971 to 1975 and 1999 to 2002 NHANES surveys. These risks will vary with all of the risk factors. To standardize the risk assessment, we present age- and sex-adjusted risks, using the age and sex distribution of the population in 1999–2002 as weights.

**Table 12.3 Impact of risk factors on predicted 10-year mortality**

| | Total population | Population 55+ |
|---|---|---|
| Predicted mortality | | |
| 1971–1975 | 9.8% | 25.7% |
| 1999–2002 | 8.4 | 21.7 |
| Change | −1.4 | −3.9 |
| Effect of: | | |
| Smoking | −0.9 | −1.2 |
| Blood pressure | −0.6 | −2.1 |
| Education | −0.2 | −0.9 |
| Cholesterol | −0.2 | −0.6 |
| Drinking | 0.1 | 0.2 |
| Body mass index | 0.3 | 0.6 |

*Notes:* Estimates are adjusted to the age and sex distribution of the population in 1999–2002. Effects of changes in race and marital status are not reported.

Table 12.3 reports the risk profile in the two time periods, for the population as a whole and for the near elderly and elderly populations. For the entire population, the ten-year mortality risk declined from 9.8 percent in 1971 to 1975 to 8.4 percent in 1999 to 2002 ($p < .001$), an absolute reduction of 1.4 percentage points, and a relative risk reduction of 14 percent. Among the population aged fifty-five and older, the absolute risk fell from 25.7 percent to 21.7 percent ($p < .001$), a relative reduction of 16 percent.

The lower rows of the table show which risk factor changes were most important in this health improvement. We calculate these by taking derivatives of the prediction equation evaluated at the mean risk level (in a logit model, $dp/dx = p[1 - p]\beta$). We evaluate this equation at the average probability in the population.

For the population as a whole, the largest risk factor change was the reduction in smoking, which contributed to a 0.9 percent absolute decrease in mortality risk. Better risk factor control was second in importance. Improved blood pressure control led to a reduction of 0.6 percent in risk, and better cholesterol control accounted for 0.2 percent. The increase in obesity offset some, but not all, of these risk reductions.

In the population aged fifty-five and older, the patterns were the same, although the magnitudes were larger. The most important factor for the older population was better control of medical risk: lower blood pressures contributed a 2.1 percent absolute reduction in mortality risk, and lower cholesterol contributed 0.6 percent. Second in importance was decreased smoking, accounting for a 1.2 percent reduction in risk. Improved education among the older group led to a nearly 1 percent reduction in risk. The impact of obesity was to raise risk by 0.6 percentage points.

The factors responsible for better control of hypertension and high cholesterol likely include increased use of medications and, to a lesser extent,

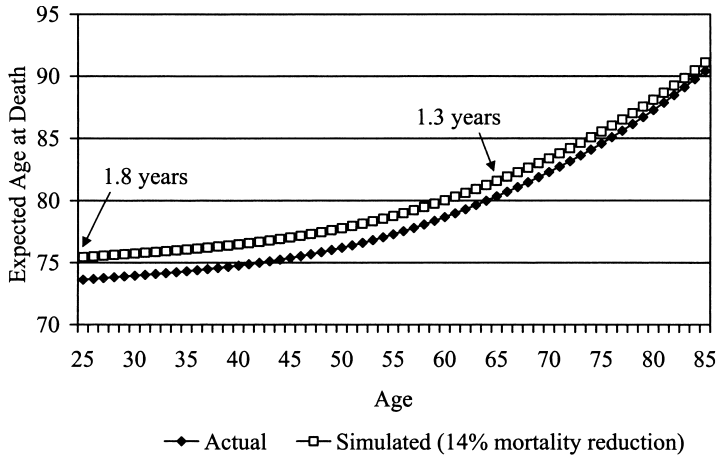


**Fig. 12.2 Effect of mortality reduction on expected age at death**

behavioral change. Use of antihypertensive medications rose markedly after the early 1970s (Burt et al. 1995), and use of HMG-CoA Reductase Inhibitors (i.e., statins) to control cholesterol increased markedly in the 1990s (Ma et al. 2005; Ford et al. 2003). Other possible factors include reduced fat and salt intake (Cutler and Kadiyala 2003).

The relatively small impact of obesity on mortality risk is in part a reflection of the fact that we control for blood pressure and cholesterol in our mortality equation. As noted in the preceding, the estimate of obesity on mortality nearly doubles without controlling for these risk factors.

Life expectancy is easier to understand than mortality rates. We simulate the impact of risk factor changes by considering how a 14 percent reduction in risk at every age would affect mortality rates at each age. Figure 12.2 shows the impact. The lower line in the figure is the expected age at death for a person alive at each age, using the 1970 Social Security life table for the United States. The upper line is the expected age at death for people at those same ages, but with a 14 percent lower mortality rate. The expected increase in longevity is 1.8 years at age twenty-five, 1.6 years at age forty-five, 1.4 years at age sixty-five, and 0.7 years at age eighty-five.

## 12.4 Forecasts of Future Risk

Forecasting in any field is difficult, but behaviors are particularly difficult to forecast. Still, forecasting is important in this case for two reasons. First, we want to understand how the disparate trends we have observed will play out in the future. Will the increase in obesity become significant enough to overwhelm reductions in smoking and improved risk factor control? If so, it suggests that longevity forecasts should not be as optimistic as

they currently are. Second, forecasting can help evaluate the impact of different interventions. How much would increased use of medications for hypertension and high cholesterol mitigate the impact of rising obesity?

We develop a forecasting model based on the pathways laid out in figure 12.1. We forecast the impact of educational changes and behaviors for the early 2020s, twenty years after the most recent NHANES. As the horizon extends further out, the forecast becomes more speculative.

Our forecasting methodology is explicitly extrapolative. We want to understand what will happen if current trends continue. This is not a "best guess" about the future health profile, which would be based on explicit consideration of the demand for and supply of health behavioral changes. We describe each component of the forecast.

### 12.4.1 Education

We have reasonable data to guide our education simulation because education rarely increases after age twenty-five. Still, differential mortality by education makes the forecast difficult. For people that will be aged twenty-five to fifty-four in two decades, assume that completed education for those ages will match those observed for those same ages in 1999 to 2002. For age and sex groups aged fifty-five and older, we assume that education will be at the highest level for the pre-fifty-five cohorts. These assumptions yield a twenty-year forecast of 17 percent of people with a high school degree or less (compared to 20 percent in 1999 to 2002) and 59 percent of people with at least some college education (compared to 55 percent currently).

### 12.4.2 Smoking

We also have good data to guide our smoking simulation. Because people rarely start smoking after age twenty-five, the share of elderly people in the future that smoke is bounded by the share of people who smoke currently. Specifically, for people who will be age forty-five and older in two decades, we assume that the share who will be ever smokers is the same as the share for that age and sex group in 1999 to 2002. To forecast the division between current and former smokers, we use data on the trend in current smoking rates. As shown in table 12.2, current smoking rates fell by 2.7 percent per year (demographically adjusted) between 1971 to 1975 and 1999 to 2002. We assume this rate continues within each age and sex group. We then subtract the forecast of current smokers from the forecast of ever smokers to estimate the share of former smokers.

For the population twenty-five to forty-four, we do not have past experience to guide our forecasts because we do not view them as adults in 1999 to 2002. For these groups, we assume that the current smoking rate is equal to the smoking rate in 1999 to 2002 among that age group, adjusted down by 2.7 percent per year (the historical trend). We assume the same ratio of

former to current smokers in those age groups as we observe in 1999 to 2002. Thus, the share of ever smokers is trending down as well.

The net impact of our forecast is that current smoking rates would decline from 25 percent of the population in 1999 to 2002 to 15 percent two decades later. The share of former smokers would be relatively constant, falling from 26 percent to 23 percent. Among the population aged fifty-five and older, current smoking rates would fall from 16 to 10 percent, and the share of former smokers would remain constant.

It is worth reiterating that our forecast is designed to extrapolate past trends, not to provide a best guess about the future. Still, some data suggest this is reasonable. Future generations of Americans will have grown up with stronger warnings about the harms from cigarettes than current generations and may, thus, smoke less. In addition, recent price increases as a result of tobacco taxes and the Master Settlement Agreement should lead additional people to stop smoking (Chaloupka and Warner 2000).

### 12.4.3 Drinking

We assume that heavy and light drinking will each change at the same annual rate in the next two decades as they did in the period from 1971 to 1975 to 1999 to 2002 (a decline of 1.5 percent per year for heavy drinking and 0.3 percent per year for light drinking). This leads to a forecast of 3.3 percent of the population being heavy drinkers in two decades (compared to 4.4 percent currently) and 61.2 percent being light drinkers (compared to 65.2 percent currently).

### 12.4.4 Obesity, Hypertension, and High Cholesterol

Forecasting obesity is difficult because obesity can change rapidly at any age (Cutler, Glaeser, and Shapiro 2003). Further, obesity is a key input into hypertension and high cholesterol, so we cannot forecast those without understanding obesity trends. Our forecast of these factors is done in several steps.

We start by extrapolating past changes in weight. Between 1971 to 1975 and 1999 to 2002, average BMI increased by 11 percent in total (from 25.6 to 28.3), or 0.4 percent annually. We assume that this annual change in BMI will continue for the next twenty years. We account for this by increasing each person's BMI in the 1999 to 2002 data uniformly by 7.4 percent for twenty years. We then calculate for each person their obesity status: underweight, normal weight, overweight, or obese. This forecast suggests that 0.6 percent of the population will be underweight (compared to 1.7 percent currently), 20.1 percent of the population will be normal weight (compared to 30.4 percent currently), 33.9 percent of the population will be overweight (compared to 34.7 percent currently), and 45.4 percent will be obese (compared to 33.2 percent currently).

It is important to note a key assumption of this weight forecast. We assume that weight increases by the same percent annually, not the same number of pounds. An increase of the same number of pounds would translate into a reduced growth rate of obesity over time. However, time series data from the Behavioral Risk Factor Surveillance Survey do not show a reduction in the rate of obesity increase in the past two decades. If anything, the rate is increasing over time.

The second step is to use these forecasts to simulate the population's blood pressure and cholesterol in two decades if there were no treatment. To do this, we use data from the 1959 to 1962 National Health Examination Survey (NHES). The NHES data were gathered from a period when blood pressure and cholesterol treatments were very scarce. They thus provide a good structural model for these risks. Following Cutler et al. (2007), we relate systolic blood pressure, diastolic blood pressure, and total cholesterol to age and age squared, interacted with gender, race dummy variables, and BMI and its square. These regressions are shown in table 12.4.

**Table 12.4 Prediction equations for blood pressure and cholesterol**

| | Blood pressure | | |
|---|---|---|---|
| | Systolic | Diastolic | Total cholesterol |
| Age | −.355** | .963** | 4.57** |
| | (.148) | (.089) | (.35) |
| Age$^2$ | .010** | −.009** | −.010** |
| | (.002) | (.001) | (.004) |
| Female | −8.55** | .918 | 35.95** |
| | (4.27) | (2.578) | (10.14) |
| Female · Age | −.116 | −.162 | −2.31** |
| | (.201) | (.121) | (0.48) |
| Female · Age$^2$ | .006** | .002* | .034** |
| | (.002) | (.001) | (.005) |
| Black | 6.31** | 4.63** | −7.88** |
| | (0.77) | (0.46) | (1.83) |
| Other race | −7.72** | −1.40 | −19.54** |
| | (1.78) | (1.08) | (4.20) |
| BMI | 1.57** | 1.42** | 8.05** |
| | (0.34) | (0.20) | (0.80) |
| BMI$^2$ | −.006 | −.010** | −.124** |
| | (.006) | (.004) | (.014) |
| Constant | 90.50** | 26.23** | −14.81 |
| | (5.46) | (3.30) | (13.01) |
| $N$ | 6,257 | 6,257 | 6,098 |
| $R^2$ | .373 | .240 | .244 |

*Source:* Data are from the 1959–1962 National Health Examination Survey.

*Note:* BMI = body mass index.

**Significant at the 5 percent level.

The general fit of the models is good, with $R^2$s ranging from 24 percent to 37 percent. The coefficients are all in the expected direction; most important, BMI is related to blood pressure and cholesterol.

We use these equations and the forecast of BMI from the 1999 to 2002 population to simulate systolic blood pressure, diastolic blood pressure, and total cholesterol. In performing the simulation, we first find the expected value of blood pressure and cholesterol for each person. We then add in a random normal error term, drawn from the same variance as in the 1959 to 1962 data. The latter step allows us to capture heterogeneity in actual values of blood pressure and total cholesterol.

The next step in the simulation is to consider the impact of treatment. In our benchmark simulation, we assume that treatment will be taken by the same share of people and have the same efficacy as medication use does in 1999 to 2002. The share of people taking medication is known from the 1999 to 2002 NHANES, which asks explicitly about use of antihypertensive and cholesterol-lowering medication. In those data, 60 percent of people with hypertension report taking antihypertensive medication, and 35 percent of people with high cholesterol report taking cholesterol-lowering medication.

For those taking medication, we draw values of blood pressure and cholesterol from the distribution of medication users, using the mean and standard deviation of each. This simulation suggests that people taking antihypertensive medication have a reduction of 7.9 (9.2) mmHg in systolic (diastolic) blood pressure (to mean levels of 143 [89] in systolic [diastolic] blood pressure) and that people taking cholesterol-lowering medication have a reduction of 30.5 mg/dL in total cholesterol (to a mean level of 244 mg/dL).

These simulations rest on the assumption that the structural equations for blood pressure and cholesterol are similar over time. Consideration of this assumption suggests that it is reasonable. One issue is whether there are other risk factors that would have changed over time. For hypertension, the other likely risk factor is salt intake, but this has not changed greatly (Cutler and Kadiyala 2003). For cholesterol levels, the share of fat and cholesterol in the diet is also important, but this, too, did not change greatly (Cutler and Kadiyala 2003). Thus, Cutler et al. (2007) conclude that the early data are a good guide to nontreatment blood pressure for the later population, and the same seems likely for cholesterol.

Table 12.5 shows the predicted changes in ten-year mortality risk for each of these simulations. We consider the different changes independently, although the effects will generally be additive. Continued reductions in smoking will reduce mortality risk, by roughly the same amount as changes over the past thirty years. The mortality risk for the entire population aged twenty-five and older would decline by 0.7 percent, or 8 percent of the baseline rate. The impact on the older population would be an ab-

**Table 12.5 Impact of possible future risk factors on predicted 10-year mortality**

| | Total population | Population 55+ |
|---|---|---|
| Predicted mortality, 1999–2002 | 8.4 | 21.7 |
| Effect of: | | |
| Continued reduction in smoking | −0.7 | −1.0 |
| Continued increase in education | 0.0 | −0.5 |
| Continued reduction in drinking | 0.1 | 0.2 |
| Continued increase in obesity | 1.1 | 1.3 |
| Continued increase in obesity and more effective medications | 0.0 | 0.1 |

*Notes:* Estimates are adjusted to the age and sex distribution of the population in 1999–2002. Effects of changes in race and marital status are not reported.

solute mortality reduction of 1.0 percent, or 5 percent of the baseline rate. Education changes would have a modest impact on mortality, larger for the older population than for the population as a whole.

The most surprising finding in table 12.5 is the impact of future changes in obesity on mortality risk. Even with existing degrees of medication use, the impact of increases in obesity, hypertension, and high cholesterol would lead to a 1.1 percent increase in mortality risk for the total population, or 13 percent of the baseline rate. In the population fifty-five and older, the increase in risk is 1.3 percent, or 5 percent of the baseline risk.

The reason for this large impact is the nonlinear relationship between BMI and weight increase, and between BMI and health risk. At higher levels of BMI, a given percent increase in weight is a greater number of pounds. And because weights are so high to begin with, further increases in weight push many more people into the obese category, where health impacts are particularly severe. Thus, the impact of BMI changes on health is becoming increasingly large.

Lack of good hypertension and cholesterol control is a major reason why increases in BMI have such large impacts on mortality risk. The last row of the table shows an alternative simulation where BMI increases the same amount, but all people with hypertension or high cholesterol are assumed to be on medication and medication is assumed to bring people to the 75th percentile of effectiveness. This is an additional reduction of 14 (7) mmHg in systolic (diastolic) blood pressure and 18 mg/dL in cholesterol. In this simulation, the impact of weight changes on mortality risk is virtually nil and is significantly smaller than the impact of continued smoking reductions. The key in this simulation is the effectiveness of medications more than getting more people to take them. Because even the typical person taking medication has high risk factor levels, increasing the share of people taking medication to 100 percent lowers the risk to only 0.1 percent for the population aged fifty-five and over (relative to 1.3 percent at the cur-

rent level). If medications can be made more effective or used more regularly, however, the benefits would be much greater.

## 12.5 Conclusions

The impact of trends in health behaviors on longevity has not been uniform across the different behaviors over the past three decades. Fewer people smoke than used to, but more people are obese. The net impact is important, but not clear a priori. Examining these factors as a whole, we show significant improvements in the health risk profile of the U.S. population between the early 1970s and the early 2000s. Reduced smoking, better control of medical risk factors such as hypertension and cholesterol, and better education among the older population have been more important for mortality than the substantial increase in obesity.

Our results suggest substantial caution about the future, however. Where smoking reductions can be expected to have continued impacts on improved health, future changes in obesity might more than overwhelm this trend. Two-thirds of the U.S. population is overweight or obese. As a result, continued increases in weight from current levels have a bigger impact on health than did increases in weight from lower levels of BMI (Olshansky et al. 2005). A large part of the impact of BMI is moderated through its effect on hypertension and high cholesterol. Given that not everyone with these conditions takes medications, or is controlled by the medication they do take, the resulting impact of rising weight on health can be significant. The optimistic side of this picture, however, is the potential for better control. If the effectiveness of risk factor control can be increased, much of the impact of obesity on mortality risk can be blunted.

Effectiveness, as we are using the term, captures several factors. One is the effect of the medication when taken as directed. Studies show that the reduction in blood pressure from medication is about the level we predict, and that people taking antihypertensive medication in the NHANES have average blood pressures about the level of people treated in clinical trials (e.g., Cushman et al. 2002). Our predictions of cholesterol reduction, in contrast, are only half those in clinical trials (LaRosa et al. 1999). Other evidence shows that physicians do not always prescribe evidence-based therapies, and not everyone prescribed these medications takes them as directed (Lenfant 2003; Osterberg 2005). Some people take their medication sporadically, others take only part of the dosage, and still others take drug "holidays."

Understanding how to improve utilization of and adherence to recommended medications are key issues. Research has focused on two possible avenues. The first is through performance-based payment. Physicians are paid for office visits, but not for ensuring follow-up with their recommendations. The idea behind pay-for-performance systems is to reward physi-

cians (or insurance companies) for successful efforts to increase utilization and possibly adherence. Such efforts might involve having nurse outreach, automatic medication refills, or more convenient office hours to monitor side effects. The second strategy involves use of information technology. Patients can receive electronic reminders about medication goals, information such as blood pressure can be transmitted and monitored electronically, and automated decision tools can help with dosing and medication switches. Whether these or other strategies offer the greatest promise of improved adherence is uncertain; our results suggest that evaluating these strategies in practice is a high research priority.

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## Comment James P. Smith

In their excellent chapter, David M. Cutler, Edward L. Glaeser, and Allison B. Rosen make several salient points. First, trends in most behavioral risk factors are strongly positive. These would include education, smoking, heavy drinking, hypertensive control, and total cholesterol. In contrast, only a few behavioral risk factors are strongly negative, most notable among them are obesity and drinking abstention. When combined into a

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