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Chapter Author(s): Michael Chernew, David M. Cutler, Kaushik Ghosh, Mary Beth Landrum

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Understanding the Improvement in Disability-Free Life Expectancy in the US Elderly Population

Michael Chernew, David M. Cutler, Kaushik Ghosh, and Mary Beth Landrum

Understanding how healthy lifespans are changing over time is central to public policy. For example, policies such as increasing the age of eligibility for Social Security or Medicare only make sense if healthy life expectancy is increasing for the vast bulk of the population. Accurate measurement of healthy life expectancy is thus essential in the welfare evaluation of such policies. Moreover, a good deal of medical spending is predicated on the idea that more intensive treatment improves quality-adjusted life expectancy. Measuring the relationship between medical advances and healthy life expectancy thus contributes to our understanding the value of medical advances and may provide insights into the causes of, and perhaps persistence of, improvements in healthy life expectancy.

Data on life expectancy are easy to obtain, but data on healthy life expectancy are more difficult. To a great extent, this is because there is no single measure of good or bad health commonly accepted in the literature. Our past work (Cutler, Ghosh, and Landrum 2014), along with much of the literature, focuses on disabled and nondisabled life expectancy. We define disability as an indicator for whether an individual has an impairment with

Michael Chernew is the Leonard D. Schaeffer Professor of Health Care Policy and the director of the HealthCare Markets and Regulation (HMR) Lab in the Department of Health Care Policy at Harvard Medical School and a research associate of the National Bureau of Economic Research. David M. Cutler is Harvard College Professor and the Otto Eckstein Professor of Applied Economics at Harvard University and a research associate of the National Bureau of Economic Research. Kaushik Ghosh is a research specialist at the National Bureau of Economic Research. Mary Beth Landrum is professor of health care policy, with a specialty in biostatistics, in the Department of Health Care Policy at Harvard Medical School.

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any Activity of Daily Living (ADL) or Instrumental Activity of Daily Living (IADL). We calculate the number of years a person turning sixty-five in different years can expect to live with and without a disability.

Our previous study shows that disability-free life expectancy has increased significantly at older ages in the United States. Between 1992 and 2005, for example, life expectancy increased by 0.7 years. Disability-free life expectancy increased by 1.6 years; disabled life expectancy fell by 0.9 years. Other results have reached similar conclusions about increases in disability-free life expectancy over time (Crimmins, Saito, and Ingegneri 1997, 2001, 2009; Manton, Gu, and Lowrimore 2008; Cai and Lubitz 2007). However, other work that defines healthy life expectancy based on presence of disease have come to an opposite conclusions finding that length of life with disease has increased (Crimmins and Beltrán-Sánchez 2010). This is consistent with findings from our previous work and others that while disease prevalence has increased, disability conditional on disease has declined (Cutler, Ghosh, and Landrum 2014; Freedman et al. 2007; Crimmins et al. 1993, Crimmins, Saito, and Reynolds 1997; Crimmins 2004; Manton, Corder, and Stallard 1993, 1997; Manton and Gu 2001; Manton, Gu, and Lamb 2006). However, little research has examined why disability-free life expectancy has increased so greatly, and in particular, what role medical advances may have played in this.

We address these issues in this chapter. Our analysis has three specific goals. First, we calculate disabled and disability-free life expectancy for a longer period of time than has been done previously. Our past research examined data from 1992 to 2005. In this chapter, we extend the analysis to 2008. This by itself does not change the conclusions materially, but the additional three years does encompass an era of relatively low growth in medical spending, so it is important to note that even with slow medical care cost increases, disability-free life expectancy kept increasing.

Second, we examine which medical conditions are associated with the greatest additions to disability-free life expectancy. We decompose both mortality and disability into fifteen medical conditions, ranging from acute but recoverable diseases such as heart disease and vision impairment, to chronic degenerative conditions such as Alzheimer's disease and Parkinson's disease, to chronic but nonfatal conditions such as arthritis and diabetes. Our central finding is that the vast bulk of the increase in disability-free life expectancy is accounted for by improvements in acute, recoverable conditions—two in particular: heart disease and vision problems. The prevalence of serious heart disease has declined over time, and for both conditions, people with the condition are in better health than they were formerly.

Our third goal is the most speculative: we seek to understand how much improvements in medical care have contributed to the health improvements associated with heart disease and vision problems. This analysis is the most speculative because we do not have great causal identification. We can ob-

serve trends in treatments and health, but we do not have an ideal way to turn these trends into causal statements. To make a stab at the causal question, we use two methodologies. In the case of cardiovascular disease, we combine trends in treatments over time with clinical trial evidence on the impact of different treatments on mortality and disability. The specific estimates are those used in the IMPACT mortality model, which we parameterize to the elderly population we study. Our results show that use of effective treatments has improved at a rate that the clinical literature suggests would have led to roughly half the health improvements that we observe. Most of the treatment improvements are pharmaceutical—cholesterol-lowering agents and antihypertensives are the major ones, but some are surgical as well.

In the case of vision, we focus primarily on increased use of cataract surgery. Fewer people have vision impairments late in the first decade of the twenty-first century than did in the early 1990s, and this seems proximately related to the increased use of cataract surgery over time. The clinical literature does not suggest a meaningful impact of cataract surgery on health-related quality of life. However, using data on individual transitions between more and less disabled states, we show significant benefits of cataract surgery on both vision and disability trends. People who receive cataract surgery are less likely to experience adverse disability trends than people who do not receive cataract surgery, controlling for the prior year's level of vision impairment. We thus conclude that it is likely that the growing use of cataract surgery explains some of the improvement in health over time.

The outline of the chapter is as follows. In the first section, we examine the overall trends in mortality and disability. Section 5.2 shows the changes in disability-free and disabled life expectancy. In section 5.3, we estimate the impact of medical conditions and demographic variables on disability. In section 5.4, we calculate the disability-free and disabled life expectancy by disease. Section 5.5 examines the pharmaceutical and surgical interventions that may have caused the declines in major cardiovascular events and mortality. Section 5.6 examines the factors responsible for improvements in vision problems. Finally, in section 5.7 we discuss our findings and conclude.

5.1 Health Trends among the Elderly

5.1.1 Life Expectancy

Life expectancy is a function of mortality rates. The mortality data are standard mortality rates from the National Center for Health Statistics. The data on disability comes from the Medicare Current Beneficiary Survey (MCBS), sponsored by the Center for Medicare and Medicaid Services (CMS). We discuss our specific measures of disability below.

Life expectancy in most developed countries increases regularly, and it has continued to do so in recent years. Figure 5.1 shows the change in life

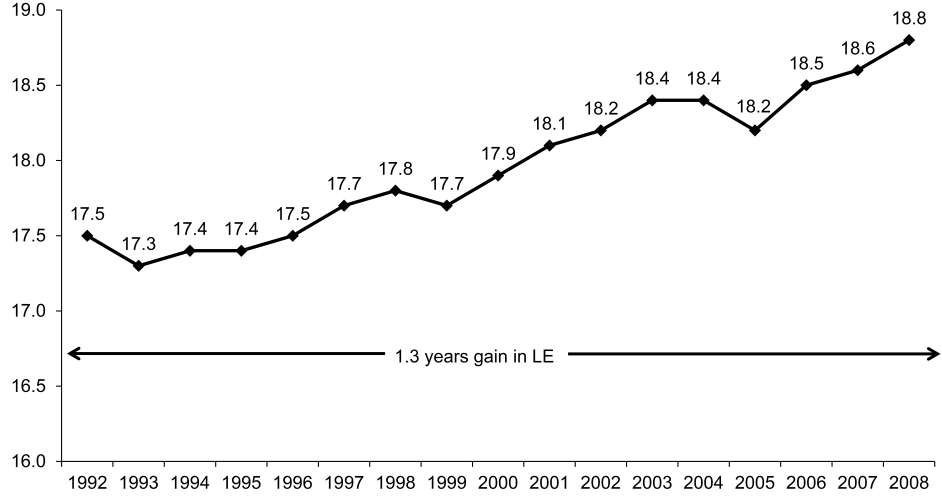

Fig. 5.1 Life expectancy at age sixty-five (total population)
Source: Data are from the Vital Statistics of the United States from the Centers for Disease Control and Prevention/National Center for Health Statistics.

expectancy at sixty-five years of age between 1992 and 2008. Over this time period, life expectancy increased by 1.3 years (17.5 to 18.8), or nearly one year per decade.

Relative to our earlier analysis, which ended in 2005, life expectancy increased by another 0.6 years between 2005 and 2008. Some of this increase is anomalous, given the unusual drop in life expectancy in 2005. Even taking out this year, however, life expectancy increases show no sign of slowing down, even in an era where medical spending increases were very low (Cutler and Sahni 2013).

For our analysis in this chapter, we care about mortality by cause in addition to overall mortality. Cause of death is reported on each death record. These causes are not believed to be wholly accurate. Death is declared when the heart stops, and thus a larger number of deaths are attributed to heart failure than is likely true. Nonetheless, it is not obvious that this will bias trends in mortality reporting over time. Without any alternative, we utilize these causes of death data.

Death codes change over time, and so the mortality rate by cause changes for that reason. Prior to year 1999, deaths were classified by the International Classification of Diseases, Ninth Revision (ICD–9), and from 1999 onward the causes of death are classified by the International Classification of Diseases, Tenth Revision (ICD–10). We use comparability ratio for the cause of death between ICD-9 and ICD-10 to compare causes of death in different periods. Comparability ratios for the broad aggregates of death that we examine are very close to 1.

We look at fifteen specific causes of death. The causes are defined to

match the MCBS. We find the closest mortality cause for the questions that people are asked about directly in the MCBS (e.g., "Has a doctor [ever] told [you/(SP)] that [you/he/she] had a myocardial infarction or heart attack?"). Generally, these are causes that are commonly reported, but not always. For example, the MCBS asks about vision problems. The closest NCHS category is death from "diseases of the eye and adnexa," which is generally not reported separately. We group the fifteen causes into several categories, based on organ system: cardiovascular disease (ischemic heart disease and stroke); cancer (four specific sites and all others); central nervous system (Alzheimer's disease and Parkinson's disease); diseases of the respiratory system; musculoskeletal disease (broken hip, rheumatoid arthritis, and nonrheumatoid arthritis); diabetes; and diseases of the eye and adnexa.

Many chronic diseases have low mortality, but nonetheless contribute to deaths in other ways. For example, very few people have diabetes as the primary cause of death, but diabetes contributes to heart disease, kidney disease, and other conditions that kill many people. A richer model would account for this disease causality, relating chronic diseases to other diseases that ultimately kill them. We do not do that here.

Figure 5.2 shows the NCHS mortality rates per 100,000 (age-sex adjusted) by disease for two time periods: 1991–1994 and 2006–2009. Each data point

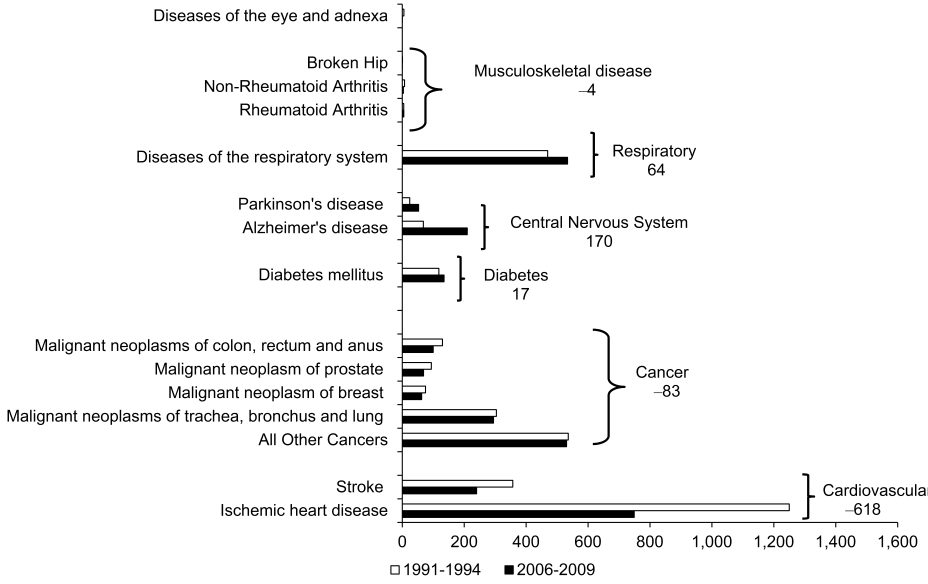

Fig. 5.2 NCHS causes of death for age sixty-five and older: Mortality rates per 100,000 (age-sex adjusted)

Source: Data are from the Centers for Disease Control and Prevention/National Center for Health Statistics on Causes of Death. The change in death rate is for two time periods, 1991–1994 and 2006–2009.

is age and sex adjusted to the population in 2000. Within each interval, we take a simple average of death rates in each of the four years. The age-sex-adjusted death rates for cardiovascular diseases have the biggest decline (–618), followed by cancer (–83). Of the cancers we can attribute, the biggest reduction is in cancer of the trachea, bronchus, and lung—a cause strongly associated with tobacco use. However, mortality from other cancers is declining as well, and preventive efforts and medical treatments likely play a role in declining cancer mortality (Cutler 2008).

Deaths from diseases of the central nervous system increased the most by 170, with Alzheimer's disease being particularly important. Death from respiratory disease and diabetes increased as well.

In our work below, we translate these changes in mortality into changes in life expectancy, using standard cause-deletion techniques. To find the increase in life expectancy from one cause, we hold constant death rates from every other cause and change death rates for only the cause we are considering. This step involves an important assumption—that the change in death from one cause does not affect death from other causes. As an example of this, if medical treatment for smokers with cardiovascular disease improves, we might expect age- and sex-adjusted mortality rates for cancers caused by tobacco use to increase. Absent more detailed knowledge of interactions among causes of death, we make the independence assumption.

5.1.2 Disability

To measure disability, we use data from the Medicare Current Beneficiary Survey (MCBS). The MCBS, sponsored by the Centers for Medicare and Medicaid Services (CMS), is a nationally representative survey of aged, disabled, and institutionalized Medicare beneficiaries that oversamples the very old (age eighty-five or older) and disabled Medicare beneficiaries. Since we are interested in health among the elderly, we restrict our sample to the population age sixty-five and older. A number of surveys have measures of disability in the elderly population (Freedman et al. 2004), including the National Health Interview Study and the Health and Retirement Study. Still, the MCBS has a number of advantages relative to these other surveys. First, the sample size is large, about 10,000 to 18,000 people annually. In addition, the MCBS samples people regardless of whether they live in a household or a long-term care facility or switch between the two during the course of the survey period. Third, the set of health questions is very broad, encompassing health in many domains. Fourth, and importantly, individuals in the MCBS have been matched to Medicare death records. As a result, we can measure death for over 200,000 people, even after they have left the survey window. The MCBS started as a longitudinal survey in 1991. In 1992 and 1993, the only supplemental individuals added were to replace people lost to attrition and to account for newly enrolled beneficiaries. Beginning in 1994, the MCBS began a transition to a rotating panel

design, with a four-year sample inclusion. About one-third of the sample was rotated out in 1994, and new members were included in the sample. The remainder of the original sample was rotated out in subsequent years. We use all interviews that are available for each person from the start of the survey in 1991 through 2009. The MCBS has two samples: a set of people who were enrolled for the entire year (the Access to Care sample) and a set of ever-enrolled beneficiaries (the Cost and Use sample). The latter differs from the former in including people who die during the year and new additions to the Medicare population. The primary data that we use are from the health status questionnaire administered in the fall survey, which defines the Access to Care sample. We thus use the Access to Care data. We date time until death from the exact date at which the Access to Care Survey was administered to the person.

To account for demographic changes in the Medicare population over this time frame, we adjust survey weights so that the MCBS population in each year matches the population in the year 2000 by age, gender, and race. All of our tabulations are weighted by these adjusted weights.

The MCBS is matched to death records available in the Medicare denominator files. As a result, we can measure death for all beneficiaries, even after they have left the survey. The death dates are available through 2012. For each individual interviewed between 1991 and 2009, we can determine if they died in the next twelve months or survived that period, died between twelve and twenty-four months or not, twenty-four and thirty-six months or not, or survived at least thirty-six months.

Trends in the distribution of time until death are shown in figure 5.3. The share of the population that is within one year of death declines from approximately 5.5 percent in 1991 to 4.5 percent in 2009, reflecting the overall reduction in mortality. The share of the population one to two years from death and two to three years from death declines as well. Correspondingly, the share of the population that is three or more years from death increased by about 0.18 percentage points annually, also shown in figure 5.3.

The MCBS asks a number of questions about a respondent's ability to function and independently perform basic tasks, shown in table 5.1. Six questions are asked about each of ADL and IADL limitations. The prevalence of each impairment is also shown in the table. The most common ADL impairment is difficulty walking, experienced by one-quarter of the population. The most common IADL impairment is doing heavy housework, which is experienced by one-third of the elderly population.

Figures 5.4A and 5.4B show the trends in ADL and IADL limitations from 1991 to 2009. We show the annual rate in the figure and (in the legend) report the annual percentage point changes between 1991–1994 and 2006–2009 in each impairment. People reporting ADL difficulties in bathing declined the most, by 0.35 percentage points annually. Other ADL difficulties also declined over the eighteen years: walking (0.34 percentage point

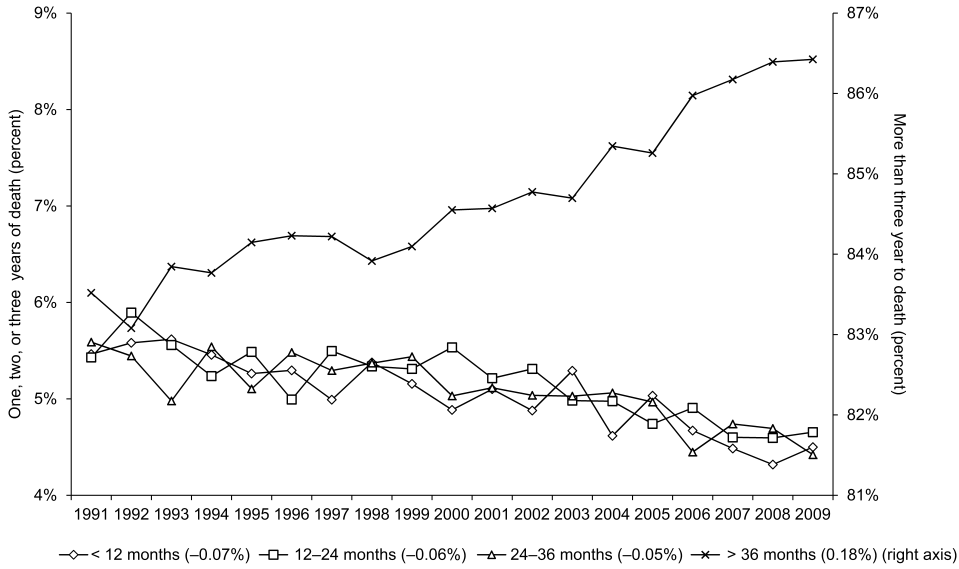

Fig. 5.3 Population distribution by time until death

Source: Data are from the Medicare Current Beneficiary Survey and Medicare denominator files linked to MCBS 1991–2009. Reported statistics is weighted to the population distribution in 2000 by age, sex, and race.

Table 5.1 Health status questions in the MCBS, 1991–2009

Num.	Question	Prevalence (%)
Activities of Daily Living says difficulty doing by himself/herself because of health or physical problem		
1	Bathing or showering	15
2	Going in or out of bed or chairs	15
3	Eating	5
4	Dressing	10
5	Walking	26
6	Using the toilet	8
Instrumental Activities of Daily Living: Difficulty doing the following activities by yourself because of health or physical problem		
7	Using the telephone	10
8	Doing light housework (like washing dishes, straightening up, or light cleaning)	16
9	Doing heavy housework (like scrubbing floors or washing windows)	34
10	Preparing own meals	14
11	Shopping for personal items	18
12	Managing money (like keeping track of expenses or paying bills)	11
Disability (any ADL/IADL difficulty)		45

Source: Tabulations are from the MCBS Access to Care sample for 1991–2009 and use sample weights adjusted to a constant year 2000 population by age, gender, and race.

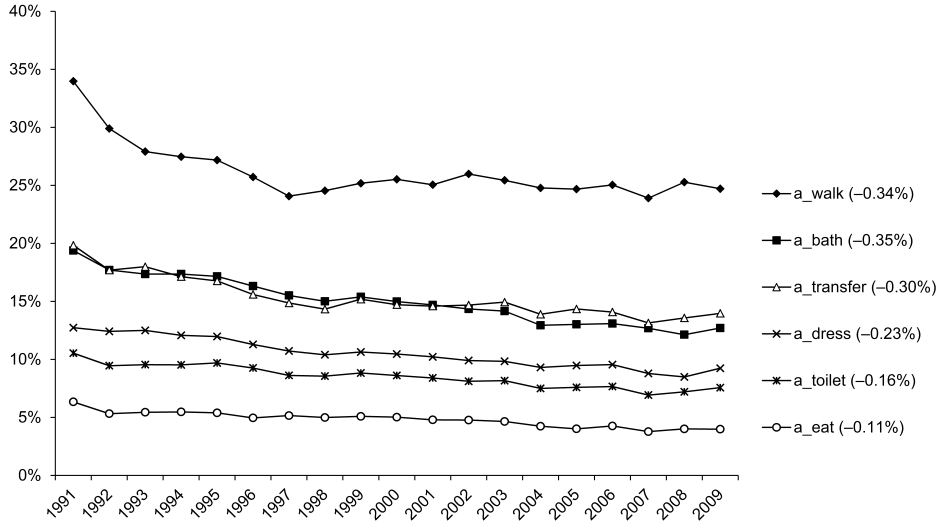

Fig. 5.4A ADL limitations in elderly Medicare beneficiaries

Note: Data are from the Medicare Current Beneficiary Survey 1991–2009, and are weighted to the population distribution in 2000 by age, sex, and race.

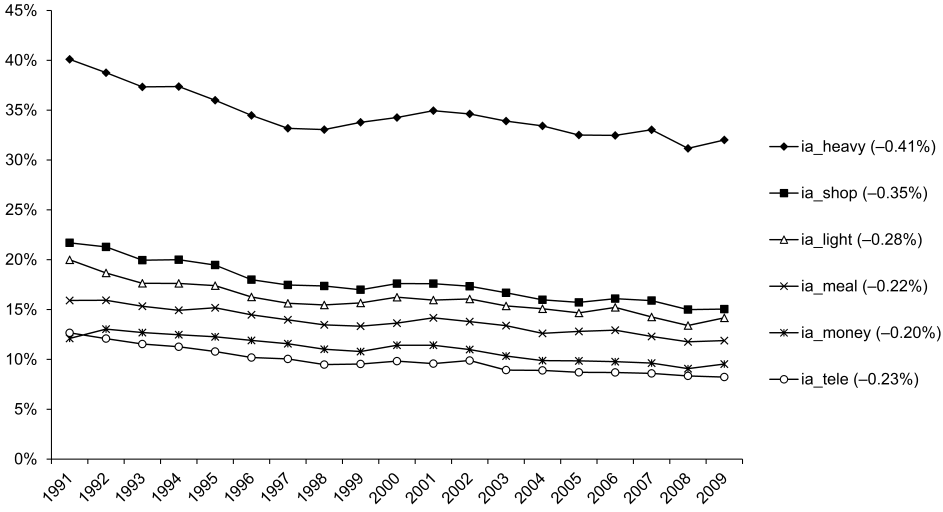

Fig. 5.4B IADL limitations in elderly Medicare beneficiaries

Note: Data are from the Medicare Current Beneficiary Survey 1991–2009, and are weighted to the population distribution in 2000 by age, sex, and race.

annual decline); going in or out of bed or chairs (0.30 percentage point decline); dressing (0.23 percentage point decline); using the toilet (0.16 percentage point decline); and eating (0.11 percentage point decline annually).

Among IADL limitations, doing heavy housework (like scrubbing floors or washing windows) showed the biggest decline from 1991 to 2009 (7 percentage points overall and 0.41 percentage points annually). Again, this decline is significantly greater in the period between 1991and 1998 than later.

The disability metric we use is the share of the population that reports any ADL or IADL limitation. Using this definition, disability was 49.5 percent in 1991–1994 and declined roughly by 7 percentage points between 1991–94 and 2006–2009, or 0.5 percentage points annually.

This pattern of declining disability is found in most previous studies using multiple nationally representative surveys (Freedman and Martin 1998; Freedman, Martin, and Schoeni 2002; Freedman et al. 2004; Schoeni, Freedman, and Wallace 2001; Schoeni et al. 2005; Cutler 2001a, 2001b). For example, a working group analyzing trends in disability from the early 1980s to 2001 across five national data sets found a consistent 1 percent to 2.5 percent annual decline in ADL disability during the mid- to late 1990s (Freedman et al. 2004; Chen and Sloan 2015). A sharp decline in walking problems and heavy housework between 1992 and 1998 is also reported in some other studies (Crimmins 2004).

That said, the literature is not entirely uniform. Crimmins (2004) reported that trends in ADL disability is not consistent across studies (Crimmins et al. 2001; Crimmins, Saito, and Reynolds 1997; Liao et al. 2001; Manton and Gu 2001; Schoeni, Freedman, and Martin 2008). Further, an update from the working group (Freedman et al. 2013) found declines in IADL and ADL disability only among those ages eighty-five and older between 2000 and 2008.

To measure lifetime disability, we need to know disability by time until death. A decline in disability matters less for healthy life expectancy if it occurs at the very end of life than if it represents a sustained period prior to death. To understand the change in disability by time until death, we use the time periods in figure 5.3: < twelve months to death, twelve to twenty-four months to death, twenty-four to thirty-six months to death, and >thirty-six months to death.

Figure 5.5 shows the trend in disability by time until death. This figure is similar to that in our earlier paper (Cutler, Ghosh, and Landrum 2014), but updating the data through 2009. The vast bulk of the reduction in disability is among people a few years away from death. People who are more than thirty-six months away from death showed a decline of 0.5 percentage points between 1991–94 and 2006–2009. Disability is high and has remained so for people within one year of death; about 80 percent of this population is disabled, and that has not changed over time.

The reduction in disability farther away from death implies that there is a compression of morbidity into the period just before death (Cai and Lubitz

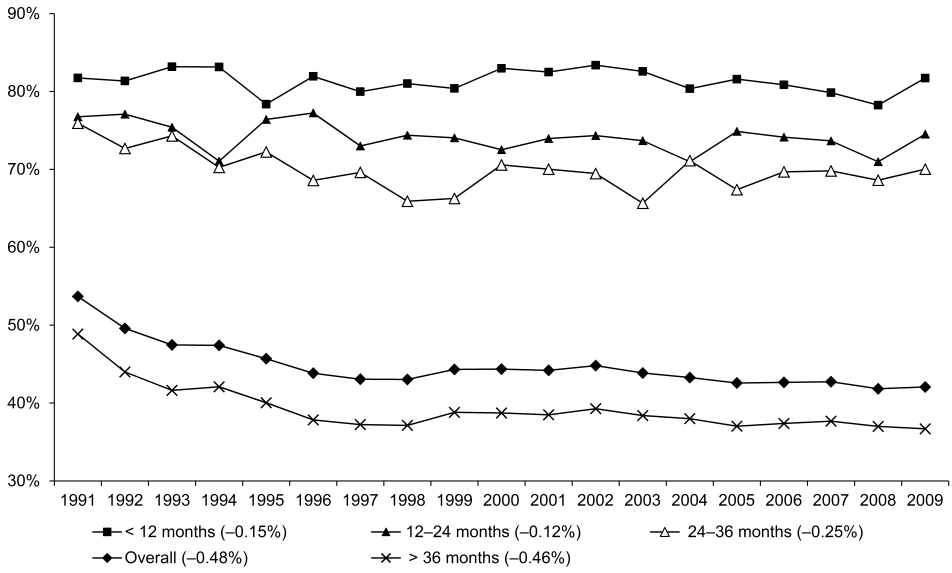

Fig. 5.5 ADL/IADL disability by time until death

Source: Data are from the Medicare Current Beneficiary Survey and Medicare denominator files linked to MCBS 1991–2009, and are weighted to the population distribution in 2000 by age, sex, and race.

2007; Cutler, Ghosh, and Landrum 2014). In the next section, we combine the NCHS period life tables and disability data to calculate disability-free and disabled life expectancy.

5.2 Disability-Free and Disabled Life Expectancy

In this section, we extend our previous research (Cutler, Ghosh, and Landrum 2014) and include more recent years of data to measure the changes in disability-free and disabled life expectancy.

The starting point for our analysis is the standard measure of life expectancy:

$$(1)\quad \mathrm{LE}(a) = \sum_{s} \left\{ \Pr\left[\text{Survive } a+s \,\middle|\, \text{Alive } a \right] + .5 * \Pr\left[\text{Die at } a+s \,\middle|\, \text{Alive } a \right] \right\}.$$

Starting at age a, every (probabilistic) year that the average person survives adds one year to life expectancy. A person who dies in a year is assumed to live half the year, and thus adds half that amount to life expectancy.

To account for disability, we modify equation (1). For those in the last year of life, we weight the half year they expect to live by the share of the people in that half year who are not disabled. Similarly, we weight the years lived by those one year away from death, two years away from death, three

years away from death, and more than three years away from death by the share of population in those intervals who are not disabled. Adding this up over all future ages yields disability-free life expectancy. Disabled life expectancy is the difference between total life expectancy and disability-free life expectancy. We can form disability-free life expectancy and disabled life expectancy for any year in which we have mortality and disability data. To match our results above, we estimate these values in two time periods: 1992 and 2008. The mortality data are from those exact years. The disability data are from 1991–1994 and 2006–2009. Although, disability data is available for individual years, we used the combined sample to provide more reliable estimates.

We present all of our calculations for a person age sixty-five in those years. Relative to our calculations in the previous section, we make one additional refinement. Where our aggregate trends were on an age-adjusted basis, here we need to disaggregate disability by age and time until death. Rather than calculating means across single-year age by time-until-death cells, which would involve many small cells, we instead use regression analysis to smooth disability rates by age and time until death. Specifically, we estimate a regression model relating disability to ten age-sex dummy variables (sixty-five to sixty-nine male, sixty-five to sixty-nine female, seventy to seventy-four male, seventy to seventy-four female, etc.), and time to death dummy variables. We estimate this regression separately for pooled 1991–1994 data and pooled 2006–2009 data. We use these regression results to predict disability rates for each person and then average predictions by single year of age. We match these to life tables in 1992 and 2008 and calculate disability-free and disabled life expectancy.

Figure 5.6 shows the trend in total life expectancy, disability-free life expectancy, and disabled life expectancy for the overall population at age sixty-five in 1991–1994 and 2006–2009. Life expectancy at age sixty-five was 17.5 years in 1992. Reflecting the fact that about half the elderly population is disabled, about half of those years were disabled. As noted earlier, life expectancy increased by 1.3 years between 1992 and 2008. The increase in disability-free life expectancy was greater than the total increase in life expectancy—1.8 years in total. The residual was a reduction in disabled life expectancy of 0.5 years. Thus, according to both metrics (the change in disabled life expectancy as well as the share of life that is spent disability free), morbidity is being compressed into the period just before death.

These results are consistent with our early findings (Cutler, Ghosh, and Landrum 2014). In our previous research, we found that for a typical person age sixty-five, life expectancy increased by 0.7 years between 1992 and 2005. Disability-free life expectancy increased by 1.6 years, while disabled life expectancy fell by 0.9 years. In the last three years, then, disability-free life expectancy increased by 0.2 years, although the disabled life expectancy increased by 0.4 years.

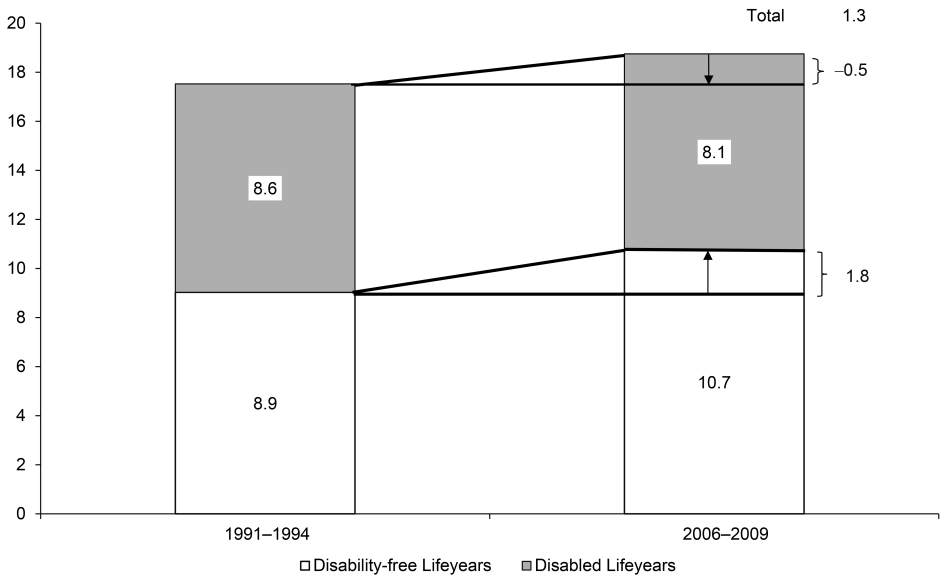

Fig. 5.6 Trend in disabled and disability-free life expectancy at age sixty-five
Note: The figure combines life expectancy data from the NCHS with imputed disability rates by age and time until death from MCBS data linked to Medicare.

In the next section, we examine the prevalence of self-reported diseases in the MCBS and how medical conditions affect disability.

5.3 Medical Conditions Affecting Disability

There is an extensive literature documenting the medical conditions that have the greatest impact on mortality and morbidity in older Americans. The Global Burden of Disease study (JAMA 2013) examined 291 diseases and injuries to identify the leading contributors to morbidity and mortality in the United States. This effort is the most exhaustive report. Ischemic heart disease, lung cancer, stroke, and chronic lung disease were the largest contributors to mortality, while musculoskeletal and mental illness were major contributors to disability. However, few results are reported by age group, and many of the top conditions are less relevant in elderly populations (for example, road injuries). Other studies looking at the burden of diseases include Wang et al. (2012), Salomon et al. (2012), and Murray et al. (2013).

Cutler, Landrum, and Stewart (2008) used data from the National Long-Term Care Survey and found that the probability of being disabled because of the cardiovascular disease fell from 9.4 percent in 1989 to 8.0 percent in 1999. Landrum, Stewart, and Cutler (2008) examined the onset of disability attributable to medical conditions as coded in the Medicare claims and com-

pared these results to respondents' self-report of the cause of their disability. Because of their high prevalence and strong association with disability onset, they found that arthritis, dementia, and cardiovascular disease were the most important contributors to disability. Several studies have examined respondents' self-reported cause of their disability in national surveys (Landrum, Stewart, and Cutler 2008; Martin, Schoeni, and Andreski 2010). Arthritis, back pain, heart disease, diabetes, mental illness, and vision problems are the most common reported causes. Similar patterns are documented in all studies: cardiovascular disease, diabetes, lung disease, and Alzheimer's are a major contributor to death and disability, while musculoskeletal, mental illness, and vision problems are major contributors to morbidity. Cancer remains a major source of mortality, but is relatively minor in its contribution to disability.

The MCBS asks extensive medical condition questions, which we use to classify diseases. The questions are generally of the form, "Has a doctor (ever) told (you/[SP]) that (you/he/she) had a myocardial infarction or heart attack?" The first set of health questions is about medical events the person has experienced. These include cancers (lung cancer, breast cancer, prostate cancer, colorectal cancer, and other cancer); cardiovascular conditions (heart disease, stroke), diseases of the central nervous system (Alzheimer's disease, Parkinson's disease), musculoskeletal problems (rheumatoid arthritis, nonrheumatoid arthritis, broken hip), pulmonary disease, diabetes, and vision problems. The prevalence of these conditions is asked about, not the incidence rate.

Trends in age-sex-adjusted disease prevalence are reported in table 5.2. The prevalence of self-reported breast and prostate cancer is increasing, respectively, at 0.04 and 0.13 percentage points annually. Breast and prostate cancer screenings are increasingly common among the elderly and are mostly paid for by Medicare. Thus, the likelihood of early detection and treatment of these cancers may be becoming more common. Cardiovascular disease prevalence has declined markedly, including both ischemic heart disease (0.44 percentage point decline annually) and stroke (0.03 percentage point decline annually). Alzheimer's disease is increasing by 0.07 percentage points annually. There has been an increase in the prevalence of nonfatal disease over time, as more people report nonrheumatoid arthritis (0.18 percentage points annually) and, particularly, diabetes (0.51 percentage points point annually). People reporting vision problems have declined substantially (0.91 percentage points annually). The prevalence of pulmonary disease has also increased (0.17 percentage points annually).

To determine the impact of each disease on disability, we relate disability in the early time period of the sample (1991–1994) and the later time period (2006–2009) to demographic and medical factors using a linear probability model:

$$(2)\quad \text{Disability}_{it} = \beta_{D,t} \bullet \text{Demographics}_{it} + \beta_{C,t} \bullet \text{Medical Conditions}_{it} + \varepsilon_{it},$$

Table 5.2 Self-reported medical event questions in the MCBS

Num.	Ever told have	Prevalence (%)	Annual % point change (1991–1994 to 2006–2009)
	Cancer		
1	Lung cancer	0.9	0.02
2	Breast cancer	4.4	0.04
3	Prostate cancer	3.4	0.13
4	Colorectal cancer	2.5	–0.04
5	Other cancer	7.0	–0.13
	Cardiovascular disease		
6	Ischemic heart disease	25.6	–0.44
7	Stroke	11.2	–0.03
	Central Nervous system		
8	Alzheimer's disease	5.2	0.07
9	Parkinson's disease	1.6	–0.01
	Musculoskeletal disease		
10	Rheumatoid arthritis	10.4	–0.11
11	Non-rheumatoid arthritis	46.0	0.18
12	Broken hip	4.1	–0.11
13	Pulmonary disease	14.0	0.17
14	Diabetes	18.7	0.51
15	Vision problems	31.4	–0.91

Source: Tabulations are from the MCBS Access to Care sample for 1991–2009 and use sample weights and use sample weights adjusted to a constant year 2000 population by age, gender, and race.

where i denotes individuals, and t denotes the period (1991–1994 or 2006–2009). Demographics include ten age-sex dummy variables and time-to-death dummy variables. Individuals may show up multiple times in the regression, depending on how frequently they are interviewed. For accurate standard errors, this should be accounted for. In the regression, we have clustered by individual id and reported the robust standard errors.

Table 5.3 shows the results of the regression. Columns (1) and (2) in show the average prevalence and regression coefficients obtained by regressing disability on demographic variables for the 1991–1994 period. Columns (3) and (4) show the same results for 2006–2009. Both the demographic and clinical covariates are strongly associated with disability. Older age is associated with higher disability, although this relationship decreased slightly over our study period. People are less disabled the further away they are from death. All of the clinical covariates are associated with higher disability rates, as we would expect. In most cases the coefficients are smaller in the 2006–2009 cohort, suggesting that these conditions are less disabling over time. Two exceptions are Alzheimer's disease and Parkinson's, which are more strongly associated with disability in the later time period.

We perform an Oaxaca decomposition to understand how much of the reduction in disability can be explained by changes in the prevalence of

Table 5.3 **Regressions explaining disability**

					Oaxaca decomposition		
	Prev. 91–94 (%)	Coeffs. (rob. se) 91–94	Prev. 06–09 (%)	Coeffs. (rob. se) 06–09	Effect of change in beta X * DBETA (%)	Effect of change in X BETA * DX (%)	Net effect (%)
Total					*–5.6*	*–1.8*	*–7.4*
Central nervous system					***0.3***	***0.2***	***0.5***
Alzheimer's	4.7	0.25 (0.01)	5.8	0.28 (0.01)	0.1	0.2	0.4
Parkinson's	1.8	0.18 (0.02)	1.6	0.24 (0.02)	0.1	0.0	0.1
Cardiovascular disease					*–1.7*	*–0.8*	*–2.5*
Ischemic heart disease	29.5	0.11 (0.01)	22.9	0.06 (0.01)	–1.5	–0.7	–2.2
Stroke	11.3	0.16 (0.01)	10.9	0.14 (0.01)	–0.2	–0.1	–0.3
Pulmonary disease							
Pulmonary	13.3	0.14 (0.01)	15.9	0.13 (0.01)	**–0.02**	**0.4**	**0.3**
Diabetes	16.2	0.11 (0.01)	23.8	0.12 (0.01)	***0.1***	***0.9***	***0.9***
Musculoskeletal disease					**–0.3**	**–0.2**	**–0.5**
Rheumatoid arthritis	12.3	0.22 (0.01)	10.7	0.20 (0.01)	–0.3	–0.4	–0.6
Nonrheumatoid arthritis	43.5	0.13 (0.01)	46.2	0.12 (0.01)	–0.2	0.4	0.2
Broken hip	5.2	0.13 (0.01)	3.5	0.16 (0.01)	0.2	–0.2	–0.1
Cancer					**–0.3**	**–0.03**	**–0.3**
Lung cancer	0.7	0.09 (0.03)	1.1	0.08 (0.02)	0.0	0.0	0.0
Brest cancer	4.2	0.04 (0.02)	4.8	0.00 (0.01)	–0.2	0.0	–0.1
Prostate cancer	2.2	0.02 (0.02)	4.1	–0.01 (0.01)	–0.1	0.0	0.0
Colorectal cancer	2.9	0.03 (0.02)	2.3	0.03 (0.02)	0.0	0.0	0.0
Other cancer	8.4	0.05 (0.01)	6.5	0.05 (0.01)	–0.1	–0.1	–0.2

Vision problem							
Vision problem	38.4	0.13 (0.01)	24.7	0.13 (0.01)	***0.1***	*–1.7*	*–1.7*
Time to death					*–4.5*	*–0.4*	*–4.9*
12–24 months	5.5	–0.04 (0.01)	4.8	–0.04 (0.01)	0.0	0.0	0.0
24–36 months	5.4	–0.04 (0.01)	4.6	–0.07 (0.01)	–0.1	0.0	–0.1
> 36 months	83.6	–0.19 (0.01)	86.2	–0.24 (0.01)	*–4.4*	*–0.5*	*–4.9*
Other demographics					**0.9**	**0.0**	**0.9**
Male 70 to 74 years	11.9	0.01 (0.01)	11.9	0.00 (0.01)	–0.1	0.0	–0.1
Male 75 to 79 years	9.5	0.06 (0.01)	9.5	0.04 (0.01)	–0.2	0.0	–0.2
Male 80 to 84 years	5.6	0.14 (0.01)	5.6	0.13 (0.01)	–0.1	0.0	–0.1
Male 85 years +	3.8	0.28 (0.02)	3.8	0.22 (0.01)	–0.2	0.0	–0.2
Female 65 to 69	12.7	0.10 (0.01)	12.7	0.08 (0.01)	–0.3	0.0	–0.3
Female 70 to 74	14.5	0.12 (0.01)	14.5	0.09 (0.01)	–0.4	0.0	–0.4
Female 75 to 79	13.1	0.19 (0.01)	13.1	0.15 (0.01)	–0.5	0.0	–0.5
Female 80 to 84	9.2	0.28 (0.01)	9.2	0.22 (0.01)	–0.5	0.0	–0.5
Female 85 years +	9.1	0.37 (0.01)	9.1	0.35 (0.01)	–0.2	0.0	–0.2
Constant	100.0	0.27 (0.01)	100.0	0.30 (0.01)	3.3	0.0	3.3

Note: The table is a decomposition of changes in the measure of disability indicated in the columns. We estimate equations of the form: $D_{it} = X_{it}\beta_t + \varepsilon_{it}$, for two time periods: 1991–1994 and 2006–2009. The table shows Oaxaca decomposition, the predicted percentage point change in D_{it} resulting from changes in the X variables, decomposed into demographics and condition prevalence, and changes in the βs, decomposed into those for conditions, those for demographics, and the constant term. Robust standard errors are reported in parentheses.

the covariates versus changes in the impact of covariates on disability (the coefficients). The Oaxaca decomposition is reported in the last three columns of the table. The first column in the Oaxaca decomposition shows the change in disability due to change in the impact of covariates (coefficients), holding prevalence constant at its 1991–1994 level. The next column shows the change in disability due to change in prevalence, holding the impact of each coefficient constant at the 1991–1994 level. The final column shows the net change.

Between 1991–1994 and 2006–2009, disability decreased by 7.4 percentage points. Out of that, 5.6 percentage points is associated with a change in the impact of covariates on disability, and the remaining 1.8 percentage points is due to change in prevalence holding the impact constant. The biggest contributors to the total disability decline are cardiovascular disease (2.5 percentage points) and vision problems (1.7 percentage points). Both the prevalence of cardiovascular diseases decreased (explaining 0.8 percent of disability decline) as well as its impact on disability (explaining 1.7 percent). Vision problems remained equally disabling in the later period, but declined in prevalence. Cancers (0.3 percentage points) and musculoskeletal diseases (0.5 percentage points) both have declined marginally. In contrast, Alzheimer's disease (0.5 percentage points) and diabetes (0.9 percentage points) have increased disability points.

Even given these conditions, people are less disabled further away from death. Among the time-to-death dummies (12 to twenty-four months, twenty-four to thirty-six months, >thirty-six months), >thirty-six months have the biggest decline in disability (about 5 percentage points). The disability changes attributed to the time-to-death dummy variables are mostly factors that remained unexplained. This may include medical conditions not captured in the MCBS, environmental factors (ramps, disability accessible buildings), changes in living conditions (married, assisted living), other medical treatments, or unmeasured changes in the severity of conditions that are occurring over time. Understanding these other factors is an important issue for future research.

5.4 Disability-Free and Disabled Life Expectancy by Disease

The results in the previous section show us which diseases are affecting disability. In this section, we calculate disability-free and disabled life expectancy by disease.

To calculate the disability-free life expectancy by disease, we used a simulation method based on regression coefficients reported in table 5.3. For each disease, we simulate the impact of changes in the disease prevalence and impact on disability by changing the prevalence and coefficient for that particular disease in the 1991–1994 data to its 2006–2009 level. We then re-predict disability by age and time until death using the new coefficients and disease probabilities. In performing this simulation, we add one addi-

tional wrinkle, allowing the disease prevalence to vary by age group. We match the disease prevalence by ten age-sex groups (sixty-five to sixty-nine male, sixty-five to sixty-nine female, seventy to seventy-four male, seventy to seventy-four female, etc.).

On the demographic side, all age-sex dummy variables are adjusted to 2000 level. So, the only other variable for which we did the simulation are the time-to-death dummy variables. We simulated these variables all at once, that is, we changed the coefficients and prevalence rates of all time-to-death variables to their 2006–2009 level jointly, and then repredicted disability.

Once we have the change in disability due to each disease, we combine this with the change in life expectancy due to that disease, using the methodology described in the previous section. The result is a calculation of the change in disability-free and disabled life expectancy due to each disease.

Figure 5.7 shows the change in disability-free and disabled life expectancy resulting from changes in each medical condition. Adding across all conditions, disability-free life expectancy increased by 1.8 years and disabled life expectancy decreased by 0.5 years. These are the same as in figure 5.6, though these estimates are derived by adding across all conditions and thus could differ from the estimates in figure 5.6 because of covariance effects.

The biggest increase in disability-free life expectancy is from cardiovascular disease (0.85 years). Roughly 50 percent of the increase in disability-free

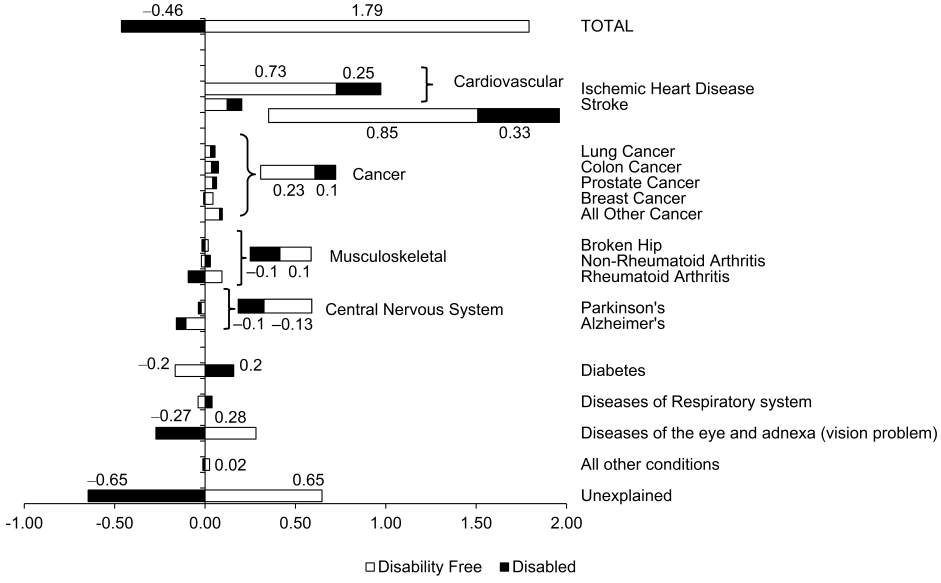

Fig. 5.7 Change in disabled and disability-free life expectancy at age sixty-five by disease (1991–1994 versus 2006–2009)

Note: The figure combines life expectancy data from the NCHS combined with causes of death data and imputed disability rates by age and time until death from MCBS data linked to Medicare.

life expectancy is from the cardiovascular disease, primarily heart disease. However, improvements in survival in those with cardiovascular disease also led to a modest increase in disabled life expectancy. Consistent with previous literature (Landrum, Stewart, and Cutler 2008) cancer remains a major source of mortality and contributes modestly to disability. Improvements in survival rates among those with cancer led to an increase in disability-free life expectancy of about 0.23 years. Vision problems show a significant impact on disability-free life expectancy (0.28 years). There is no increase in life expectancy from vision impairment, so all of this change comes from a reduction in disabled life expectancy.

Increased prevalence and impact of diseases of the central nervous system (Alzheimer's and Parkinson's) have reduced disability-free life expectancy by 0.13 years. The diseases of the central nervous system are very important as they have significant impacts on both morbidity and mortality. For diabetes, the disability-free life expectancy declined by 0.2 years.

The penultimate row of the table shows the impact of causes of death we have not separately delineated. These residual causes of death have a small aggregate effect on disability-free life expectancy. The final row shows the unexplained change in disability for those three or more years from death, which translates into 0.65 years of disability-free life expectancy and—since this is not associated with any mortality reduction—a reduction in disabled life expectancy of the same amount.

Overall, the most important gains in disability-free life expectancy are from cardiovascular disease and vision problems. In the next two sections, we explore the factors that may have caused the decline in mortality and morbidity for these two conditions. We examine the importance of medicines and revascularization in preventing primary and secondary cardiovascular events. We also explore the impact of surgical procedures like cataract surgery on improving vision problem and its impact on vision-related measurements and quality of life.

5.5 Pharmaceutical and Surgical Interventions in Reducing Cardiovascular Incidence, Mortality, and Morbidity

The question we address in this section is how much of the reduction in cardiovascular mortality can be explained by increased use of medications and procedures. Previous research has shown for conditions such as musculoskeletal problems and circulatory disorders, higher rates of surgery are plausibly related to reduced disability (Cutler 2005). There are also studies showing how pharmaceutical agents play an important role in the prevention of cardiovascular disease (Downs et al. 1998; Weisfeldt and Zieman 2007). And deaths from cardiovascular disease have greatly declined among the elderly in the United States over the past decades (Rosen et al. 2007). We examine how these trends are related.

We have two measures of cardiovascular disease: ischemic heart disease and stroke. Ischemic heart disease happens when there is reduced blood flow to the heart. Acute myocardial infarction or heart attack is the most serious form of ischemic heart disease, when the blood flow to the heart is abruptly interrupted, causing part of the heart muscle to die. A stroke happens when poor blood flow to the brain or a hemorrhage in the brain leads to death of part of the brain. Historically, heart attack and strokes are a major cause of death in the United States.

Figures 5.8 and 5.9 show more detail on death from these two causes. The mortality rate for ischemic heart disease has declined significantly over time (figure 5.8), from an age-adjusted rate of 1,250 per 100,000 in 1992–1994 to 749 per 100,000 in 2006–2009 ($p < 0.001$). The decline was significantly greater from 2001–2009 (35 percent) than prior to 2001 (17 percent). Figure 5.9 shows the trends in stroke mortality. Stroke mortality also declined significantly over time, from an age-adjusted rate of 357 per 100,000 in years

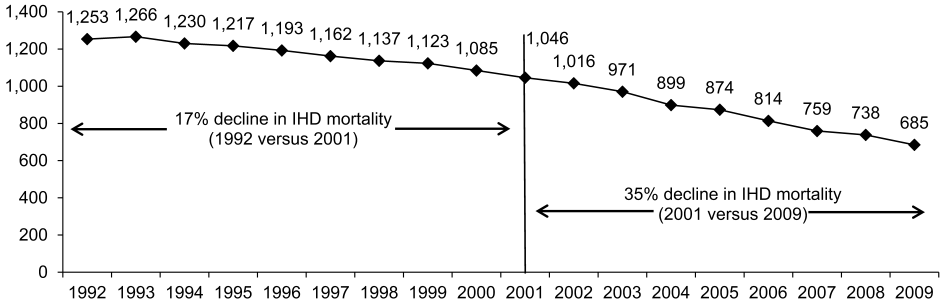

Fig. 5.8 IHD mortality rates per 100,000 (age-sex adjusted)

Source: Data are from the Centers for Disease Control and Prevention/National Center for Health Statistics on Causes of Death and microdata on mortality available at the National Bureau of Economic Research.

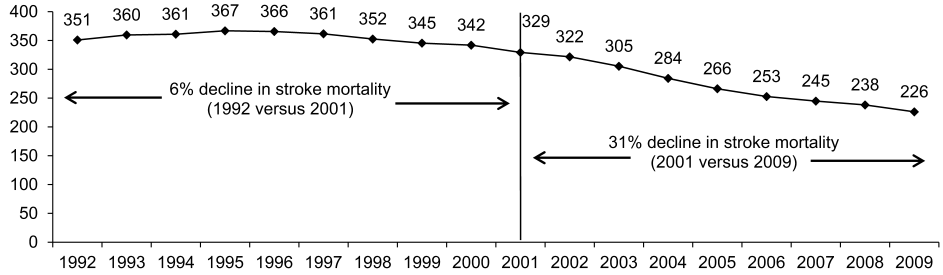

Fig. 5.9 Stroke mortality rates per 100,000 (age-sex adjusted)

Source: Data are from the Centers for Disease Control and Prevention/National Center for Health Statistics on Causes of Death and microdata on mortality available at the National Bureau of Economic Research.

1992–1994 to 240 per 100,000 in 2006–2009 ($p < 0.001$). Again, the reduction was greater after 2001 (31 percent) than before (6 percent).

Understanding how medical treatments or other changes influence these trends is challenging. The natural econometric technique is to relate receipt of the technology to reduced mortality. This is problematic, however, because receipt of different therapies is not random. For example, people who are more severely ill are more likely to receive more intensive technologies. Those same people are also more likely to die. Thus, receipt of intensive technologies is often associated with higher mortality in a cross section, even if the technology is actually effective.

A natural solution to the endogeneity problem is to instrument for technology receipt. In preliminary analysis, we spent some time evaluating potential instruments, including area-level treatment rates and their changes. However, there were no characteristics of areas or their changes that led to plausible instruments for technology receipt.

As a result, we follow a different path. We use the IMPACT model (Ford et al. 2007; Capewell, Morrison, and McMurray 1999; Capewell et al. 2010) to gauge the impact of treatment trends on mortality among US adults sixty-five years and older between 1992 and 2009. The IMPACT model is a multistate model explaining coronary heart disease mortality. The model divides the population into two groups: patients receiving medical and surgical treatments for heart disease and those who are not. It then estimates the contribution of treatment and risk factor changes (smoking, high systolic blood pressure, elevated total blood cholesterol, obesity, diabetes, and physical inactivity) to mortality. Within each disease state, clinical literature is used to parameterize the impact of different treatments and risk factors on mortality. The model was developed for the population as a whole (ages twenty-five to eighty-four); we parameterize the model to estimate the causes of mortality reduction in the elderly.

The rates of medical and surgical treatments and risk factors are calculated using various data sources, including NHDS (National Hospital Discharge Survey), Medicare data, MCBS, and NHANES (National Health and Nutrition Examination Survey), following the methodology of Ford et al. (2007). Similarly, we follow the assumptions of Ford et al. (2007) in assuming that the proportion of treated patients actually taking medication is 100 percent among hospitalized patients, 70 percent among symptomatic patients in the community, and 50 percent among asymptomatic patients in the community.

We start by presenting general trends in risk factors and the use of medications among the population overall, and for those with prior heart disease. We use data from NHANES, which measures cardiovascular risk factors such as total cholesterol, HDL cholesterol, blood pressure, body mass index, Hemoglobin A1c, body mass index, and smoking status. We use several years of data: 1988–1994 and biennial data from 1999 to 2000 through 2011 to 2012. Table 5.4 reports the trend in cardiovascular risk factors. As

Table 5.4 **Prevalence of cardiovascular risk factors in NHANES subjects age ≥ sixty-five years during years and Medicare enrolled 1988–1994, 1999–2000, 2001–2004, 2005–2008, and 2009–2012**

	Men					Women				
	1988–1994	1999–2000	2001–2004	2005–2008	2009–2012	1988–1994	1999–2000	2001–2004	2005–2008	2009–2012
Age	73	73	74	73	73	74	74	74	74	74
Diabetes (%)	12	14	18	20	22	12	14	17	19	20
HbA1c > 6.5%	11	13	12	13	15	10	10	10	12	13
Current smoker (%)	14	12	9	9	7	11	9	6	6	6
Systolic blood pressure	139	138	134	134	132	142	147	142	140	136
Total cholesterol	209	202	194	181	178	231	224	216	207	205
HDL cholesterol	46	47	48	49	50	56	57	61	60	60
Body mass index	26	28	28	29	28	27	28	28	28	29

Notes: The table combines NHANES 2001–2002 and 2003–2004; NHANES 2005–2006 and 2007–2008; and NHANES 2009–2010 and 2011–2012.

is well known, the elderly population has become more obese over time. Even still, total cholesterol levels have decreased in both men and women, and HDL cholesterol (good cholesterol) has increased. This is quite plausibly a result of greater statin use. Systolic blood pressure has also been decreasing marginally in both men and women. The prevalence of diabetes has increased in both men and women, and the prevalence of high HbA1c levels has increased.

Since smoking and obesity are the two most significant risk factors for cardiovascular disease, we focus on them in some detail. Figure 5.10 shows the trends in smoking and obesity in the elderly Medicare population. Obesity has increased markedly over time, while smoking has declined. Stewart, Cutler, and Rosen (2009) found that if past obesity trends continued unabated, the negative effects on the health of the US population will increasingly outweigh the positive effects gained from declining smoking rates.

The elderly population is now treated more aggressively to control cardiovascular risk. Statins are one well-known example. Statins help reduce the level of low-density lipoproteins (LDL) in the blood and also help with modulation of oxidative stress (Beltowski 2005) that may eventually lead to heart attack. Antihypertensive drugs include beta-blockers, angiotensin-

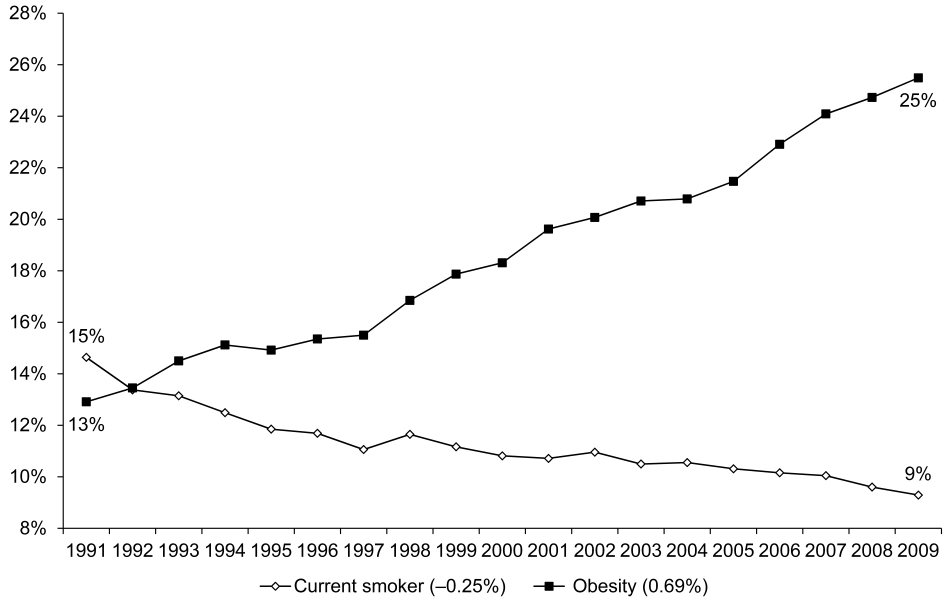

Fig. 5.10 Smoking and obesity prevalence in age sixty-five and older Medicare beneficiaries

Source: Data are from the Medicare Current Beneficiary Survey and Medicare denominator files linked to MCBS, 1991–2009, and are weighted to the population distribution in 2000 by age, sex, and race.

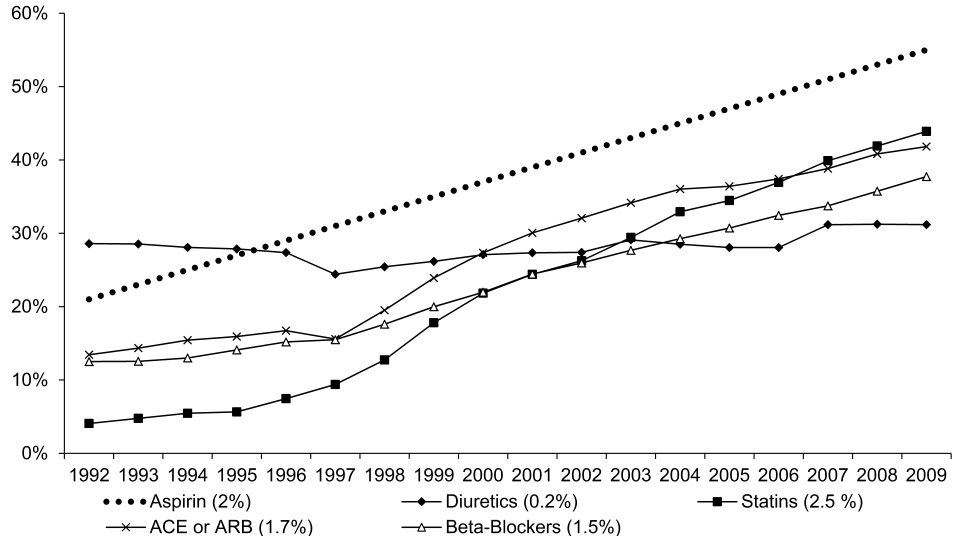

Fig. 5.11 Trends in cardiovascular medication usage in age sixty-five and older Medicare beneficiaries

Sources: Data on medication usage (statins, ACE or ARB, beta-blockers, and diuretics) is from Prescribed Medicine Events in the MCBS data. Rates are adjusted to 2000 population by age, sex, and race. The aspirin usage from the 1992 to 1994 period is from NHANES III, and the later period is from MEPS 2007. We did a linear interpolation for the intermediate years.

converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), and diuretics. Aspirin use is also increasingly common. Literature suggests that low-dose aspirin helps reduce cardiovascular disease incidence and recurrence.

Figure 5.11 shows the trends in the use of these medications in the elderly community population, and figure 5.12 shows similar trends among patients with ischemic heart disease. The data on medication usage is from the Prescribed Medicine Events file in the MCBS that contains cost and utilization of prescribed medicines for the community population. Statin usage increased the most (2.5 percentage points annually in the population without IHD), though the use of beta-blockers (1.5 percentage points annually) and ACE inhibitors (1.7 percentage points annually) also increased markedly. The use of diuretics increased marginally (0.2 percentage point annually). Aspirin is available over the counter and thus is not in the prescribed medicine file. We obtain usage in the earlier time period (1992–1994) from NHANES III, with later data from the 2007 Medical Expenditure Panel Survey (MEPS). We used a linear interpolation to fill in the intermediate years. For this reason, we show the plots for aspirin use in dotted lines. Use among the population with IHD increased even more rapidly. In addition,

procedure rates increased rapidly in the IHD population with a 2.0 percentage point annual increase in primary percutaneous interventions (PCI).

To estimate the impact of these changes on cardiovascular disease mortality, we first calculate the difference between the observed and expected number of deaths from ischemic heart disease in 2009. Compared to what would have happened had age-specific mortality rates remained constant at its 1992 level, the decline in age-adjusted death rate resulted in 228,910 fewer deaths from ischemic heart disease in 2009. This is shown in the first row of table 5.5.

The remaining rows of table 5.5 shows how this reduction in mortality distributes across treatments and risk factors. All told, the IMPACT model estimates that about half of reduced ischemic heart disease mortality (51 percent) is a result of improved treatment, about slightly less than half is a result of improved risk factors (44 percent), and a small share is unexplained (5 percent). Improvement in inpatient treatments only explained 8 percent of improvement. However, secondary prevention after MI had major effects, particularly, statins (9 percent), warfarin (1 percent), beta-blockers (11 per-

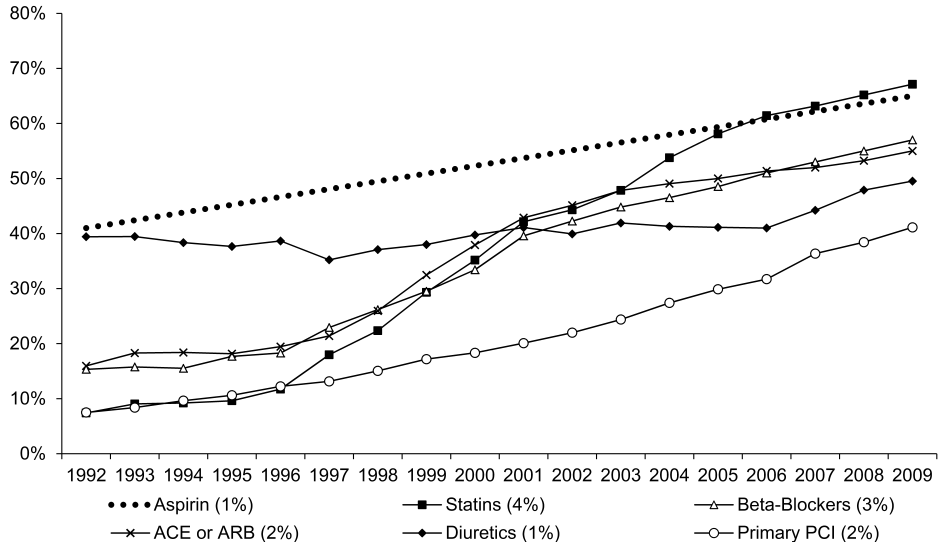

Fig. 5.12 Trends in medication usage in ischemic heart disease patients (sixty-five years and older)

Sources: Data on medication usage is from (statins, beta-blockers, ACE or ARB, diuretics) Prescribed Medicine Events in the MCBS data. Rates are adjusted to 2000 population by age, sex, and race. Aspirin usage for the earlier period is from NHANES III and the later year is from MEPS. The intermediate years are linear interpolations. Primary PCI usage is from 5 percent Medicare sample for people hospitalized for ischemic heart disease (410.X—414.X). Primary PCI is defined as having a PCI on the same day or the next day of an ischemic heart disease hospitalization.

Table 5.5 **IMPACT mortality model estimated deaths prevented or postponed in the elderly United States population in 2009**

	Number of deaths	Percent of total change
Total change relative to expectations	**–228,910**	
Treatments	**–117,521**	**51**
Ischemic heart disease hospitalization (aspirin, beta-blockers, ACE inhibitors, primary PCI and CABG)	–18,158	8
Secondary prevention after MI		
Aspirin	–2,399	1
Beta-blocker	–25,476	11
ACE inhibitor	–12,752	6
Statins	–19,827	9
Warfarin	–2,195	1
Rehabilitation	–9,299	4
Secondary prevention after CABG or PTCA (aspirin, beta-blockers, ACE inhibitor, statins, rehabilitation)	–2,270	1
Chronic angina (CABG, angioplasty, aspirin, statins)	1,268	–1
Antihypertensive for hypertension treatment	–8,895	4
Statins for lipid reduction treatment	–17,538	8
Risk factors	**–100,511**	**44**
Smoking prevalence (%)	–19,299	8
Systolic blood pressure (mm hg)	–51,270	22
Total cholesterol (mmol/liter)	–68,787	30
Physical inactivity (%)	–3,924	2
Body mass index (BMI)	13,254	–6
Diabetes prevalence (%)	29,515	–13

Note: In the risk factor calculations for systolic blood pressure, the number of deaths excludes people receiving treatment for hypertension and for total cholesterol. The number of deaths excludes patients receiving statins. Risk factor estimates are from NHANES. The treatment data comes from several sources including NHDS, Medicare data, MCBS and some other studies.

cent), and ACE inhibitors (6 percent). Primary prevention was also a major contributor, including statins for lipid reduction (8 percent) and hypertension treatment (4 percent). The impact of other treatments was smaller.

Considering risk factor changes, the biggest changes were reduced total cholesterol (30 percent) and blood pressure (22 percent). These are each separate from treatment in that the estimated decline in blood pressure and cholesterol is among those who do not report taking medication. That said, the Ford et al. (2007) study does not adjust its estimate of population trends among the nontreated for the fact that increased numbers of people—likely with high levels of cholesterol and blood pressure—are being treated. Thus, it is possible that selection effects contribute to the magnitude of the risk factor estimates, making these estiamtes overstated. Smoking reduction contributed 8 percent, while increased BMI and diabetes led to 19 percent

more deaths. Overall, these findings are close to Ford's 2007 study for the adult population ages twenty-five to eighty-four, which found a 47 percent reduction due to treatment and 44 percent due to risk factors.

Using the IMPACT model, we simulated the annual impact of treatment and risk factor changes for ischemic heart disease mortality rates between 1992 and 2009. Figure 5.13 shows the results. The line with squares shows the conterfactual mortality rate per 100,000 if the mortality rate by age remained constant at its 1992 level and only the population totals changed. The line with diamonds shows the actual mortality trend in ischemic heart disease between 1992 and 2009. The line with triangles shows the simulated effect of treatment and improvements in risk factors combined on mortality. The fact that the simulated mortality tracks the actual mortality shows that the model as a whole fits very well. The line with x-marks divides the total effect found by the model into a treatment component (the upper part) and a risk factor component (the lower part). Almost all of the changes in the 1990s are due to treatment; those after 2000 are a mix of treatment and risk factor changes. Figures 5.14 and 5.15 show the effect of individual medications and risk factors on mortality. Between 1992 and 2009, increased use of statins for primary and secondary prevention saved roughly 48,000 lives. Increased use of ACE inhibitors and beta-blockers in IHD patients saved another 43,000 lives. Other studies have found similar impact of greater satin use. For example, Grabowski et al. (2012) found that statin therapy reduced low-density lipoprotein levels by 18.8 percent, which translated into roughly 40,000 fewer deaths.

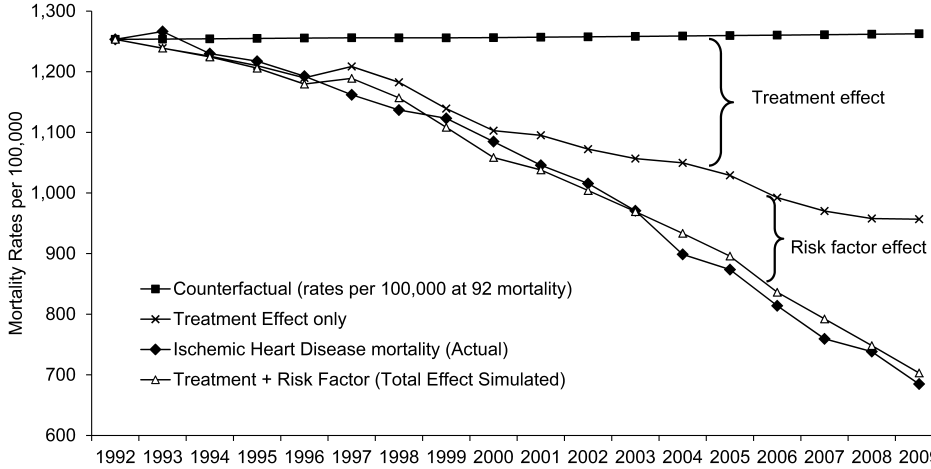

Fig. 5.13 Ischemic heart disease mortality rates per 100,000: Actual versus simulated

Source: Data are from the Centers for Disease Control and Prevention/National Center for Health Statistics on Causes of Death and the US Census of population.

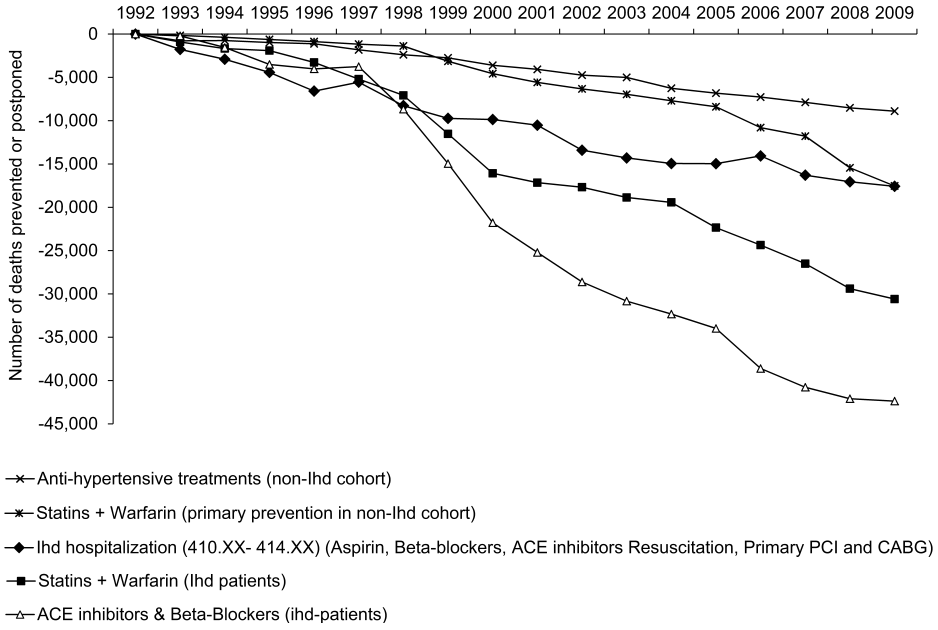

Fig. 5.14 Estimated deaths prevented or postponed in the elderly United States population: Treatment effect

Source: The treatment effects are calculated using data from NHDS, MCBS, Medicare and other sources cited in Ford et al. (2007).

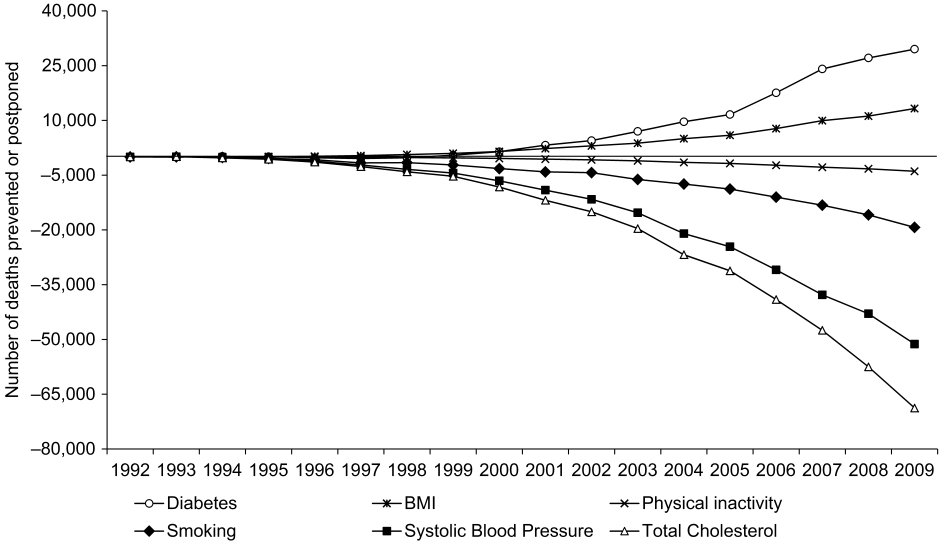

Fig. 5.15 Estimated deaths prevented or postponed in the elderly United States population: Risk factors

Source: The risk factors are calculated using data from NHANES and MCBS.

One central question is how these mortality changes are related to the overall improvement in disability-free life expectancy we noted above. Linking these two estimates is not completely straightforward, as the disability-free life expectancy estimates include changes in both disability and mortality, while the IMPACT model includes mortality only. To understand how these mortality changes contribute to the overall improvement in disability-free life expectancy, we need to understand whether the improvement in mortality is accompanied by a reduction in disability or whether it keeps more people alive in a disabled state. The former would add much more to disability-free life expectancy than the latter.

By and large, the interventions shown to be important in reducing mortality are those that reduce the incidence of adverse events and enable improved functioning after an event, not just prolong survival for those who are very disabled. This is shown directly in the 12 percent of reduced deaths accounted for by primary prevention—generally associated with fewer acute events—and indirectly in the secondary prevention after an MI. For example, statins and antihypertensive agents decrease cardiovascular symptoms in addition to reducing heart attacks and strokes. Still, to be conservative, we assume that medical treatments reduce mortality and the prevalence of acute cardiovascular events, but leave unaffected disability for those who have had a cardiovascular event. We model this empirically as treatment affecting mortality and the prevalence of disease, but not disability conditional on having ischemic heart disease.

Considering only the reduction in mortality and cardiovascular disease prevalence yields an increase in disability-free life expectancy of 0.53 years between 1992 and 2008 (compared to 0.73 years including changes in disability conditional on ischemic heart disease as well, as shown in figure 5.7). If half of this is a result of medical treatments, this yields an increase of 0.26 years associated with medical advance. This is a very large increase; by itself, it accounts for 15 percent of the total increase in disability-free life expectancy over this time period. Figure 5.16 shows the impact of cardiovascular disease treatment on disability-free life expectancy.

The obvious follow-up question is whether these benefits exceed the cost of the therapies. Costing out the impact of the treatment changes is somewhat complex because the lifetime costs of any therapy include what people will suffer who do not die of cardiovascular disease. For this reason, we defer the cost-effectiveness calculation for future research.

5.6 Vision Impairment in the Elderly Population

We now conduct an analysis of possible factors that may explain the change in disability-adjusted life expectancy associated with vision impairment. The trend in having a current vision problem is shown in figure 5.17. Current vision problems have declined from about 40 percent of the elderly

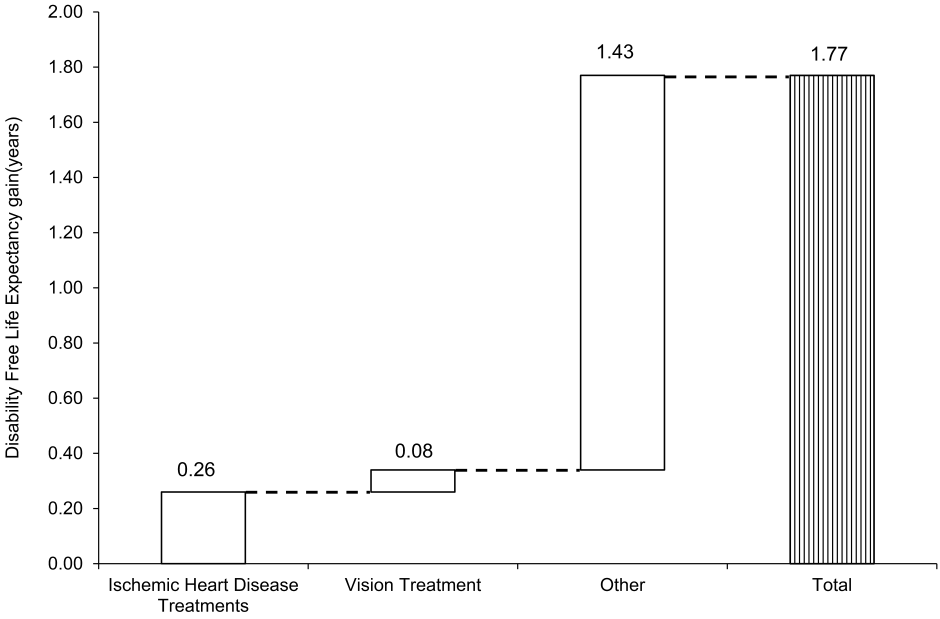

Fig. 5.16 Impact of treatments on changes in disability-free life expectancy at age sixty-five

Note: The figure combines life expectancy data from the NCHS with imputed disability rates by age and time until death from MCBS data linked to Medicare.

population to about 25 percent. This decline has been noted in other studies (Freedman and Martin 1998; Cutler 2001a, 2001b; Freedman et al. 2007; Cutler, Ghosh, and Landrum 2014).

There are several reasons why people may have vision problems, and thus several treatments for them. The most prevalent source of vision problems in the elderly is cataracts, a condition in which the lens of the eye becomes progressively opaque. Most cataracts are a natural process of aging. Other possible causes of vision impairment include glaucoma, diabetic retinopathy, and macular degeneration (Kasper 1989).

Cataract surgery is the most common treatment for cataracts in the United States. Figure 5.17 also shows the percentage of people who have had cataract surgery in the elderly Medicare population. This is from a self-reported question the first year that an individual is in the survey. Self-reported cataract surgery increased from 20 percent to 33 percent. The decline in current vision problems looks like a mirror image of increase in cataract surgery, both in number (16 percent decline versus a 13 percent increase) and in timing. It is thus plausible that people are reporting fewer vision problems as a result of greater use of cataract surgery.

For comparison, the bottom line of the figure shows treatment for macu-

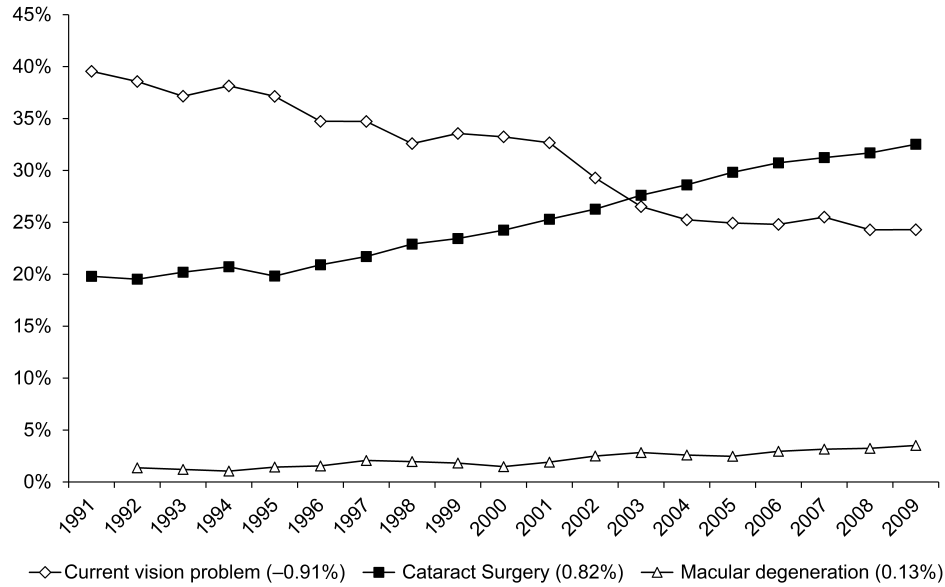

Fig. 5.17 Cataract surgery and vision problems

Source: Data are from the Medicare Current Beneficiary Survey and Medicare denominator files linked to MCBS 1991–2009, and are weighted to the population distribution in 2000 by age, sex, and race.

lar degeneration, measured by claims for macular degeneration drugs. This is also increasing over time, though the rates are much lower.

The question is whether the increased use of cataract surgery can explain the reduction in vision impairment, and thus reduced disability. We started in the same way for cardiovascular disease, in particular by examining the literature on the impact of cataract surgery on disability. Table 5.6 contains a brief literature review of studies documenting vision changes and broader changes in health-related quality of life after cataract surgery. The first part of the table shows clear evidence that cataract surgery results in fewer vision problems. Studies show improvements in Snellen visual acuity, improvements in self-reported trouble with vision, and also improvements in VF-14 scores and NEI-VFQ25 scores in a period four to six months after cataract surgery.

Despite these improvements in vision, however, studies of health-related quality of life, shown in the lower panel of the table, indicate no significant change in periods after cataract surgery. This is true for measures such as the Euroqual-5D (EQ-5D), the SF-12, and the SF-36. This result is confusing, since the evidence presented earlier shows that vision problems are a significant cause of disability. That said, none of these survey instruments are a perfect match for our measure of ADL and IADL disability.

To better understand the impacts of cataract surgery on vision problems

Table 5.6 Effectiveness and use of cataract surgery on vision problems and disability

Findings	Study
Vision problems	
Improvement in Snellen visual acuity	Steinberg et al. (1994) Mangione et al. (1994) Javitt et al. (1993)
Improvement in self-reported trouble with vision	Steinberg et al. (1994)
Improvements in VF-14 score	Steinberg et al. (1994) Owsley et al. (2007)
Improvements in NEI-VFQ25 score	Groessl et al. (2013)
Health-related quality of life (HRQOL)	
EQ-5D shows insignificant change	Foss et al. (2006)
SF-12 shows insignificant change	Castells et al. (2006)
No significant impact on SF-36 physical functioning	Mangione et al. (1994) Owsley et al. (2007)

Note: **Snellen visual acuity** test is decimal acuity with 1.0 representing 20/20 vision. **VF-14** is a method for assessing the quality of the visual function of those with cataracts in daily living from the patient's viewpoint, developed in 1994 by Steinberg et al. **VFQ-25** is the product of an item-reduction analysis of the longer field test version of the survey called the fifty-one-item National Eye Institute Vision Function Questionnaire (NEI-VFQ). **EQ-5D** is a standardized instrument for use as a measure of health outcomes. **SF-12** is a short-form 12 health survey that was developed for the Medical Outcomes Study (MOS). **SF-36** is a thirty-six-item, patient-reported survey of patient health commonly used to determine the cost effectiveness of medical treatments.

and disability, we look at trends in vision-problem reporting and disability within individuals who have and have not received cataract surgery. The idea is that if cataract surgery changes the trend in vision degredation over time, this might be apparent by following individual health trends. Of course, such an effect is not guaranteed to be found. For example, if people who have cataract surgery are at the poor end of the vision distribution, their vision might deteriorate even if cataract surgery prevents a more rapid deterioration. Conversely, if the nonvision health of people who receive cataract surgery is better, their health transitions may have been relatively better even without the cataract surgery. This is the endogeneity issue noted above. In the case of vision, we do not have a disease model we can use for validation.

Our regressions for vision impairment are of the form:

$$(3)\ VI_{it} = \beta_C * \text{Cataract Surgery}_{it} + \beta_D * \text{Demogs}_{it} + \beta_M * \text{Medical Conditions}_{it} + \beta_S * \text{Social Factors}_{it} + \beta_V * VI_{it-1} + \varepsilon_{it},$$

where VI is the degree of vision impairment (ordered, as described below) and VI_{it-1} is a set of dummy variables representing the answers in the prior year of the survey. Cataract Surgery$_{it}$ is a dummy variable indicating a claim for cataract surgery between the previous interview date and the current interview date (interviews are generally in the fall). Demographics include

age-sex dummy variables and a time trend. We also control for four groups of medical conditions: chronic disabling (Alzheimer's, Parkinson's, and pulmonary), recoverable acute events (ischemic heart disease, stroke, and broken hip), nonfatal chronic conditions (diabetes and arthritis), and cancer. Social factors may influence disability as well. We address this with dummy variables for whether the person is married and whether they live alone. The latter variable is partly endogenous—people who are less healthy may not be able to live alone; the former is plausibly more exogenous. Since 2002, the MCBS has asked about three levels of vision impairment: no vision problem, a little vision problem, and a lot of vision problem. We order them in that fashion (healthiest is 0 and a lot of vision problems is 2) and estimate an ordered probit model. Prior to 2002, the MCBS also included a category for whether the individual was blind. The share of people reporting blindness is small, so we include this with the group reporting a lot of vision problems. Because the relationship between past vision impairment and current vision impairment may change in the year that the survey questionnaire changes, however, we omit data from 2002 from the regression.

We estimate a separate but similar model for disability. In this case, we form an ordered variable for no disability, IADL disability only, 1–2 ADLs, and 3+ ADLs (in this case, from 0 to 3, where higher numbers indicate worse health). We also include dummies for lagged disability status and lagged vision impairment as independent variables.

To measure cataract surgery during the course of our sample, we use MCBS fee-for-service cost and use data (recall that self-reports of cataract surgery receipt are asked only in the first year of the survey). Eligible CPT codes include simple cataract surgery (66984), complex cataract surgery (66982), removal of lens material (66840, 66850, 66852, 66920, 66930, 66940), and intracapsular cataract surgery (66983).

The results of the two regressions are reported in table 5.7. The model for vision impairment is in the left panel, and the model for disability is in the right panel. Each model estimates the coefficients on the indicated variables and a series of cut points for the different variables; we report the coefficient estimates, but not the cut point estimates. The fit of the vision impairment model is reasonable, with a pseudo-R^2 of 19 percent. All the variables have expected signs and most are statistically significant. The cataract surgery dummy variable is negative and significant ($p < 0.001$) indicating an improvement in reporting of vision problems in people having cataract surgery. To interpret the magnitude of the coefficient, we repredicted the probability of having no vision problem, a little vision problem, and a lot of vision problems for those who received cataract surgery under the counterfactual that they had not received surgery. The predicted impact of cataract surgery for no vision problem rises from 59.4 percent to 62.0 percent, a little vision problem falls from 29.3 percent to 27.9 percent, and the probability of having a lot of vision problems falls from 11.3 percent to 10.1 percent.

Table 5.7 Vision problem acuity and disability states: Ordered probit model

	Vision problem		Disability	
	Coef.	Robust std. err.	Coef.	Robust std. err.
Cataract surgery receipt (t –1 to t)	–0.092	0.024	–0.126	0.021
Year trend	–0.026	0.001	–0.004	0.001
Age 70–74 male	0.009	0.023	0.042	0.023
Age 75–79 male	0.139	0.023	0.175	0.024
Age 80–84 male	0.171	0.023	0.359	0.024
Age 85+ male	0.213	0.025	0.593	0.024
Age 65–69 female	0.103	0.026	0.163	0.027
Age 70–74 female	0.091	0.022	0.172	0.022
Age 75–79 female	0.147	0.022	0.298	0.022
Age 80–84 female	0.208	0.022	0.434	0.022
Age 85+ female	0.217	0.022	0.675	0.022
Nonwhite	–0.017	0.014	0.035	0.012
Married	0.019	0.013	–0.214	0.011
Living alone	0.042	0.013	–0.190	0.011
Ischemic heart disease	0.117	0.010	0.102	0.009
Stroke	0.127	0.013	0.307	0.012
Alzheimer's disease	–0.250	0.020	0.671	0.018
Parkinson's disease	0.129	0.031	0.416	0.031
Broken hip	0.044	0.019	0.268	0.019
Pulmonary disease	0.117	0.012	0.205	0.011
Diabetes	0.135	0.011	0.213	0.010
Arthritis	0.208	0.010	0.168	0.009
Cancer	0.052	0.011	0.056	0.010
A little vision problem (previous year)	1.086	0.010	0.057	0.010
A lot of vision problem (previous year)	1.954	0.021	0.143	0.016
IADL limitation only (previous year)	—	—	0.891	0.012
1–2 ADL limitations (previous year)	—	—	1.437	0.013
3+ ADL limitations (previous year)	—	—	2.746	0.022
N	109,728		109,728	
Pseudo-R^2	0.188		0.303	

Source: Data are from the MCBS cost and use sample for 1992–2009 and use sample weights adjusted to a constant year 2000 population by age, gender, and race. The ordinal dependent variable for vision problem is: no vision problem, a little vision problem, and a lot vision of problem. The ordinal dependent variable for disability is no IADL/ADL limitations, IADL limitations only, 1–2 ADL limitations, and 3+ ADL limitations.

As expected, difficulty with vision increases with age and diseases—the nonfatal chronic conditions (arthritis, diabetes) have the biggest impact on vision acuity. Also interestingly, there is no trend in vision impaiment for married people, but vision worsens for people who are living alone.

The right columns of the table examine the impact of cataract surgery on disability. The model fit is again reasonable, with a pseudo-R^2 of 30 percent. A good share of this is a result of the fact that disability does not change

greatly over time, and we include prior year's disability in the model. The cataract surgery dummy is also negative and significant ($p < 0.001$), implying a reduction in the extent of disability after cataract surgery. Again, to better interpret the coefficient, we repredicted the probability of the various levels of disability for those who received cataract surgery if they had not received surgery. The predicted probability of having no limitations falls from 55.5 percent to 52.0 percent, the predicted probability of an IADL limitation only increases from 14.3 percent to 14.9 percent, the predicted probability of having 1–2 ADL limitations increases from 18.8 percent to 20.2 percent, and the predicted probability of having 3+ ADL limitations increases from 11.4 percent to 12.9 percent.

One way to gauge the magnitude of these coefficients is to compare them with other variables. We focus on two other malleable variables: marital status and living alone. Married people have better trends in health than unmarried people. Roughly speaking, the impact of being married is twice the impact of having cataract surgery. Also interestingly, those living alone have improved health over time. We suspect this is a result of selection; those with materially worse health will move in with relatives or move to an institution. The correlation between cataract surgery and each of these variables is small; the coefficient on cataract surgery is essentially unchanged controlling for marital status and living arrangements. This lends some support to the idea that the coefficient on cataract surgery is picking up the true effect of medical care changes, not just other attributes of the individual.

A second way to gauge the magnitude of this coefficient is to consider its implication for the time series. As figure 5.17 shows, the share of people receiving cataract surgery increased by 13 percentage points over our time series. If each cataract surgery operation reduces the probability of being disabled by 3.5 percentage points, the implied reduction in disability is 0.5 percentage points. Table 5.3 shows that disability fell by 1.7 percentage points due to fewer vision impairments. Thus, the increase in cataract surgery explains 27 percent of the improved health related to vision impairment over time. This translates into 0.08 years gain in disability-free life expectancy due to increase in cataract surgery, or roughly 5 percent of the total increase in disability-free life expectancy.

Even this estimate, while large, is likely to be an underestimate, as cataract surgery may explain the trend in vision and thus disability in years beyond its receipt. Thus, we conclude that cataract surgery has an important impact on disability trends over time.

5.7 Conclusion

Our analysis of disability-free life expectancy yields three important conclusions. First, we show that over the 1991–2009 period, disability-free life expectancy rose and disabled life expectancy declined. These results mirror our earlier findings, but extend the years for which we have this information.

Second, we identify the diseases that contribute most to the improvement in disability-free life expectancy. Quantitatively, the largest contributions come from cardiovascular disease and vision problems. Cardiovascular disease contributes to both mortality and morbidity improvements; the impact of vision impairment is entirely through morbidity. Our results attribute 63 percent of the improvement in disability-free life expectancy to these two conditions.

Third, and more speculatively, we consider the factors that lead to improvements in these conditions. For neither condition can we do the type of rigorous empirical research that would identify a population effect with a very high degree of reliability. Nonetheless, our methodologies have strengths. In the case of cardiovascular disease, we use a well-validated model to identify the role of medical treatments versus social factors in improved health. These results show that a bit under half of the mortality reduction from cardiovascular disease is a result of improved medical treatments, translating into about 0.26 years of disability-free life, or roughly 15 percent of the overall increase in disability-free life expectancy.

Our results on vision problems are less certain, since no validated models for vision impairment exist that are comparable to those for cardiovascular disease. The major medical treatment change for people with vision impairment over this time period is the increased use of cataract surgery. Cataracts are the primary source of vision impairment in the elderly population, and cataract surgery has diffused widely. Our results on within-person changes in vision impairment and disability show that receipt of cataract surgery is associated with improved vision and disability trends. We estimate that one-quarter of the reduction in disability due to poor vision results from greater use of cataract surgery. This translates into about 5 percent of the overall increase in disability-free life expectancy. The result on improved vision after cataract surgery mirrors the clinical literature. The finding of reduced disability is novel; studies have not shown a very large improvement in disability after cataract surgery. It is unclear if the difference in results is due to our larger sample sizes, to having measures more focused on disability, or to a tendency to perform cataract surgery in the healthiest members of the population. To the extent that these findings are not driven by selection, however, they indicate real and large benefits of diffusion of cataract surgery.

The important question raised by our results is to identify the other contributors to improved population health over time. There are some conditions that our data do not ask about—mental illness and musculoskeletal issues (back pain, for example)—that have been shown to be major contributors to disability in other studies (JAMA 2013). Other data that have information on these conditions would be a valuable addition to what we present here.

In addition, recent work has documented a slowdown or even reversal of improvements in morbidity and mortality in more recent periods, particularly in the near elderly (Martin, Schoeni, and Andreski 2010; Chen and

Sloan 2015; Case and Deaton 2015). Moreover, improvements in health have been concentrated in high socioeconomic populations (Chetty et al. 2016). The combination of medical, social, and environment factors that have led to better health is a major topic for future research.

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Comment Jonathan Skinner

The chapter by Chernew, Cutler, Ghosh, and Landrum is an ambitious one that covers considerable ground, ranging from updated measures of disability compression in the United States to the key question of how much the diffusion of health care technology has contributed to improving health outcomes. First, the authors have revisited the questions posed in Cutler, Ghosh, and Landrum (2014) to test whether the decline in disability (and increase in disability-free days) has continued through 2008; the reassuring answer is yes. But they go beyond this question to dig in more as to the

Jonathan Skinner is the James O. Freedman Presidential Professor of Economics at Dartmouth College, professor of community and family medicine at the Dartmouth Institute for Health Policy and Clinical Practice, and a research associate of the National Bureau of Economic Research.

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