NBER WORKING PAPER SERIES

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Working Paper 22306
http://www.nber.org/papers/w22306

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
June 2016

This research was funded by the National Institute on Aging (P01AG005842) and Pfizer. We are grateful to Jonathan Skinner for helpful comments. The views expressed herein are those of the authors and do not necessarily reflect the views of the National Bureau of Economic Research.

At least one co-author has disclosed a financial relationship of potential relevance for this research. Further information is available online at http://www.nber.org/papers/w22306.ack

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NBER Working Paper No. 22306
June 2016
JEL No. I1,I31

ABSTRACT

Understanding how healthy lifespans are changing is essential for public policy. This paper explores changes in healthy lifespan in the U.S. over time and considers reasons for the changes. We reach three fundamental conclusions. First, we show that healthy life increased measurably in the US between 1992 and 2008. Years of healthy life expectancy at age 65 increased by 1.8 years over that time period, while disabled life expectancy fell by 0.5 years. Second, we identify the medical conditions that contribute the most to changes in healthy life expectancy. The largest improvements in healthy life expectancy come from reduced incidence and improved functioning for those with cardiovascular disease and vision problems. Together, these conditions account for 63 percent of the improvement in disability-free life expectancy. Third and more speculatively, we explore the role of medical treatments in the improvements for these two conditions. We estimate that improved medical care is likely responsible for a significant part of the cardiovascular and vision-related extension of healthy life.

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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 advance and may provide insights to 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 non-disabled life expectancy. We define disability as an indicator for whether an individual has an impairment with any Activity of Daily Living (ADL) or Instrumental Activity of Daily Living (IADL). We calculate the number of years a person turning 65 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 et al. 1989, 1997, 2001, 2009, Manton et al. 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, 2011). 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, 1997, 2004; Manton et al., 1993, 1997, 2001, 2006). However, little research has examined why disability-free life expectancy has increased so greatly, and in particular, what role medical advance may have played in this.

We address these issues in this paper. Our analysis has three specific goals. First, we calculate disabled and disability-free life expectancy for a longer period than has been done previously. Our past research examined data from 1992 to 2005. In this paper, 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 15 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 non-fatal conditions such as arthritis and diabetes. Our central finding is that the vast bulk of the increase in disability-free life expectancy is due to 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 observe 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 anti-hypertensives 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 in the late 2000s 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 growing use of cataract surgery explains some of the improvement in health over time.

The outline of the paper is as follows. In the first section, we examine the overall trends in mortality and disability. Section 2 shows the changes in disability-free and disabled life expectancy. In section 3, we estimate the impact of medical conditions and demographic variables on disability. In Section 4, we calculate the disability-free and disabled life expectancy by disease. Section 5 examines the pharmaceutical and surgical interventions that may have caused the declines in major cardiovascular events and mortality. Section 6 examines the factors responsible for improvements in vision problem. Finally, in section 7 we discuss our findings and conclude.

I. Health Trends Among the Elderly

Life Expectancy

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

Life expectancy in most developed countries increases regularly and it has continued to do so in recent years. **Figure 1** shows the trend in life expectancy in the United States at 65 years of age between 1992 and 2008. Over this 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; Hartman et al., 2015).

For our analysis in this paper, we care about mortality by cause in addition to overall mortality. Cause of death is reported on each death record. On death certificates, the attending physician must write what the underlying cause of death was and what other dominant conditions, if any, led to the immediate cause of death. These causes are not believed to be entirely accurate due to reporting bias (Lakkireddy et al. 2004; Ravakhah, K., 2006; Wexelman et al. 2013). Death is declared when the heart stops, and thus a larger number of deaths are attributed to heart disease than is likely true. Nonetheless, it is not obvious that this will bias trends in mortality reporting over time. Without any better alternative, we utilize the national data on the causes of death data available from the NCHS.

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-CM), and from 1999 onward the causes of death are classified by the International Classification of Diseases, Tenth Revision (ICD–10-CM). We use comparability ratio for the cause of death between ICD-9-CM and ICD-10-CM to compare causes of death in different periods. Comparability ratios for the broad aggregates of death that we examine are very close to unity.

We look at 15 specific causes of death. These 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 causes 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 15 causes into several categories, based on organ system: cardiovascular disease (ischemic heart disease and

stroke); cancer (4 specific sites and all other); central nervous system (Alzheimer's disease and Parkinson's disease); diseases of the respiratory system; musculoskeletal disease (broken hip, rheumatoid arthritis, and non-rheumatoid 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. 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 2 shows the NCHS mortality rates per 100,000 (age-sex adjusted) by disease for two periods: 1991-1994 and 2006-2009. Each data point 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 deaths rates for cardiovascular diseases have the biggest decline (-618), followed by cancer (-83). Of the cancers we can attribute, one of the biggest reductions 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.

Later in the paper, 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.

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 over-samples the very old (aged 85 or older) and disabled Medicare beneficiaries. Since we are interested in health among the elderly, we restrict our sample to the population aged 65 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 250,000 person-year observations, 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 period, 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.

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 12 months or survived that period, died between 12-24 months or not, 24 and 36 months or not, or survived at least 36 months.

Trends in the distribution of time until death are shown in **Figure 3**.The share of the population that is within one year of death is declines from approximately 5.5% in 1991 to 4.5% in 2009, reflecting the overall reduction in mortality. The share of the population 1-2 years from

death and 2-3 years from death declines as well. Correspondingly, the share of the population that is 3 or more years from death increased by about 0.18 percentage points annually, also shown in **Figure 3**.

The MCBS asks a number of questions about a respondent's ability to function and independently perform basic tasks, shown in **Table 1**. Six questions are asked about each of ADL and IADL limitations. The prevalence of each impairment is also shown in the same 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.

Figure 4 shows the trends in ADL and IADL limitations from 1991-2009. 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 18 years: walking (0.34 percentage point 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 1991-2009 (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% 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 et al., 1998, 2002, 2004; Schoeni et al., 2001; and 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% to 2.5% 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 et al., 1997; Liao et al. , 2001; Manton et al., 2001; Schoeni et al., 2008). Further, an update from the working group (Freedman et al., 2012) found declines in IADL and ADL disability only among those ages 85 and over 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 same time windows used in **Figure 3**: < 12 months to death, 12- 24 months to death, 24-36 months to death, and >36 months to death.

Figure 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 36 months away from death showed an annual decline of 0.5 percentage points between 1991-94 and 2006-09. 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., 2007; Cutler, Ghosh, and Landrum, 2014). In the next section, we combine the NCHS period life tables and MCBS disability data to calculate disability-free and disabled life expectancy.

II. Disability-free and disabled life expectancy

In this section, we extend our previous research (Cutler, Ghosh, and Landrum, 2014) and include some 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:

$$LE(a) = \sum_{S} \left\{ Pr\left[Survive\ a\ +\ s\ \mid\ Alive\ a\right] + 0.5\ *\ Pr\left[\ Die\ at\ a\ +\ s\ \mid\ Alive\ a\right] \right\} \quad (1)$$

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 using life tables in two periods: 1992 and 2008. The life expectancy data from the NCHS life tables are from those exact years. The disability data from MCBS are from 1991-94 and 2006-09. Although, disability data is available for individual years, we used the combined sample to provide estimates that are more reliable using bigger sample size.

We present all of our calculations for a person aged 65 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 and sometimes unstable results, we instead use regression analysis to smooth disability rates by age and time until death. Specifically, we estimate a regression model relating disability to 10 age-sex dummy variables (65-69 male, 65-69 female, 70-74 male, 70-74 female, etc.), and time to death dummy variables. We estimate this regression separately for pooled 1991-94 data and pooled 2006-09 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 6 shows the trend in total life expectancy, disability-free life expectancy, and disabled life expectancy for the overall population at age 65 in 1991-94 and 2006-09. Life expectancy at age 65 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 aged 65, 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.

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

III. 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 Burden of Diseases, Injuries, and Risk Factors study (US Burden of Disease Collaborators, 2013) examined 291 diseases and injuries to identify the leading contributors to morbidity and mortality in the US. 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. (2012).

Landrum, Stewart, and Cutler (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) also examined the onset of disability attributable to medical conditions as coded in the Medicare claims and compared 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; Cutler, Ghosh, and Landrum, 2014; Martin et al., 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, non-rheumatoid 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 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 non-fatal disease over time, as more people report non-rheumatoid 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 (1991-94) and the later time period (2006-09) to demographic and medical factors using a linear probability model:

$$\text{Disabilty}_{it} = \beta_{D,t} * \text{Demographics}_{it} + \beta_{C,t} * \text{Medical Conditions}_{it} + \varepsilon_{it}, \qquad (2)$$

where i denote individuals, and t denotes the period (1991-94 or 2006-09). Demographics include 10 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 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 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 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 1991-94 level. The next column shows the change in disability due to change in prevalence, holding the impact of each coefficient constant at 1991-94 level. The final column shows the net change.

Between 1991-94 and 2006-09, 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% of disability decline) as well as its impact on disability (explaining 1.7%). 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 (12-24 months, 24-36 months, >36 months), >36 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.

IV. 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 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 1991-94 data to its 2006-2009 level. We then repredict disability by age and time until death using the new coefficients and disease probabilities. In performing this simulation, we add one additional wrinkle, allowing the disease prevalence to vary by age group. We match the disease prevalence by 10 age-sex groups (65-69 male, 65-69 female, 70-74 male, 70-74 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, i.e. we changed the coefficients and prevalence rates of all time to death variables to their 2006-2009 level jointly, and then re-predicted 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 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 6**, though these estimates are derived by adding across all conditions and thus could differ from the estimates in **Figure 6** because of covariance effects.

The biggest increase in disability-free life expectancy is from cardiovascular disease (0.85 years). Roughly 50% of the increase in disability-free 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 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 bar of the figure 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 bar shows the unexplained change in disability for those 3 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.

V. 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 et al., 2007). Other studies have found that the deaths from cardiovascular disease have greatly declined among the elderly in the United States over the past decades (Rosen et al., 2007; Wilmot et al., 2015). 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 8 and **9** show more detail on death from these two causes. The mortality rate for ischemic heart disease has declined significantly over time (**Figure 8**), from an age-adjusted rate of 1,250 per 100,000 in 1992-94 to 749 per 100,000 in 2006-09 ($p<0.001$). The decline was significantly greater from 2001-09 (35%) than prior to 2001 (17%). **Figure 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 year 1992-94 to 240 per 100,000 in 2006-09 ($p<0.001$). Again, the reduction was greater after 2001 (31%) than before (6%).

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 et al. 1999; Capewell et al., 2010) to gauge the impact of treatment trends on mortality among U.S. adults 65 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 25-84); 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% among hospitalized

patients, 70% among symptomatic patients in the community, and 50% 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 1999-2000 through 2011-2012. **Table 4** reports the trend in cardiovascular risk factors. As 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 10** shows the trends in smoking and obesity in the elderly Medicare population from the MCBS survey. Obesity has increased markedly over time, while smoking has declined. Stewart et al. (2009) found that if past obesity trends continued unabated, the negative effects on the health of the U.S. 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-

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 11 shows the trends in the use of these medications in the elderly community population, and **Figure 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 (1992-94) 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 non-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**.

The remaining rows of **Table 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%) is a result of improved treatment, about slightly less than half is a result of improved risk factors (44%), and a small share is unexplained (5%). Improvement in inpatient treatments only explained 8% of improvement. However, secondary prevention after MI had major effects, particularly, statins (9%), warfarin (1%), beta-blockers (11%), and ACE-inhibitors (6%). Primary prevention was also a major contributor, including statins for lipid reduction (8%) and hypertension treatment (4%). The impact of other treatments was smaller.

Considering risk factor changes, the biggest changes were reduced total cholesterol (30%) and blood pressure (22%). 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 non-treated 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%, while increased BMI and diabetes led to 19% more deaths. Overall, these findings are close to Ford's 2007 study for the adult population aged 25-84, which found a 47% reduction due to treatment and 44% 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 13** shows the results. The red line 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 blue line

shows the actual mortality trend in ischemic heart disease between 1992 and 2009. The green line shows the simulated effect of treatment and improvements in risk factors combined on mortality. The fact that the simulated mortaltiy tracks the actual mortality shows that the model as a whole fits very well. The purple line 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 14** and **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%, which translated into roughly 40,000 fewer deaths.

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% of reduced

deaths accounted for by primary prevention – generally associated with fewer acute events – and indirectly in the secondary prevention after a heart attack. For example, statins and anti-hypertensive 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 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 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.

VI. 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 17.** Current vision problems have declined from about 40 percent of the elderly population to about 25 percent. This decline has been noted in other studies (Freedman and Martin 1998; Cutler 2001; 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 treatment for cataracts in the U.S. **Figure 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% to 33%. The decline in current vision problems looks like a mirror image of increase in cataract surgery, both in number (16% decline vs. a 13% 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 macular 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 as for cardiovascular disease, in particular by examining the literature on the impact of cataract surgery on disability. **Table 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 4-6 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 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 non-vision 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:

$$VI_{it} = \beta_C * \underset{\text{Surgery}}{Cataract_{it}} + \beta_D * Demogs_{it} + \beta_M * \underset{\text{Conditions}}{Medical_{it}} + \beta_S * \underset{\text{Factors}}{Social_{it}} + \beta_V * VI_{it-1} + \varepsilon_{it}, \quad (3)$$

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), non-fatal 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 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%. 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 problem 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% to 62.0%, a little vision problem falls from 29.3% to 27.9%, and the probability of having a lot of vision problems falls from 11.3% to 10.1%.

As expected, difficulty with vision increases with age and diseases – the non-fatal 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%. 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 55.5% to 52.0%, the predicted probability of an IADL limitation only increases from 14.3% to 14.9%, the predicted probability of having 1-2 ADL limitations increases from 18.8% to 20.2%, and the predicted probability of having 3+ ADL limitations increases from 11.4% to 12.9%.

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 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 3** shows that disability fell by 1.7 percentage points due to fewer vision impairments. Thus, the increase in cataract surgery explains 27% 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% 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.

VII. 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 (US Burden of Disease Collaborators, 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 et al., 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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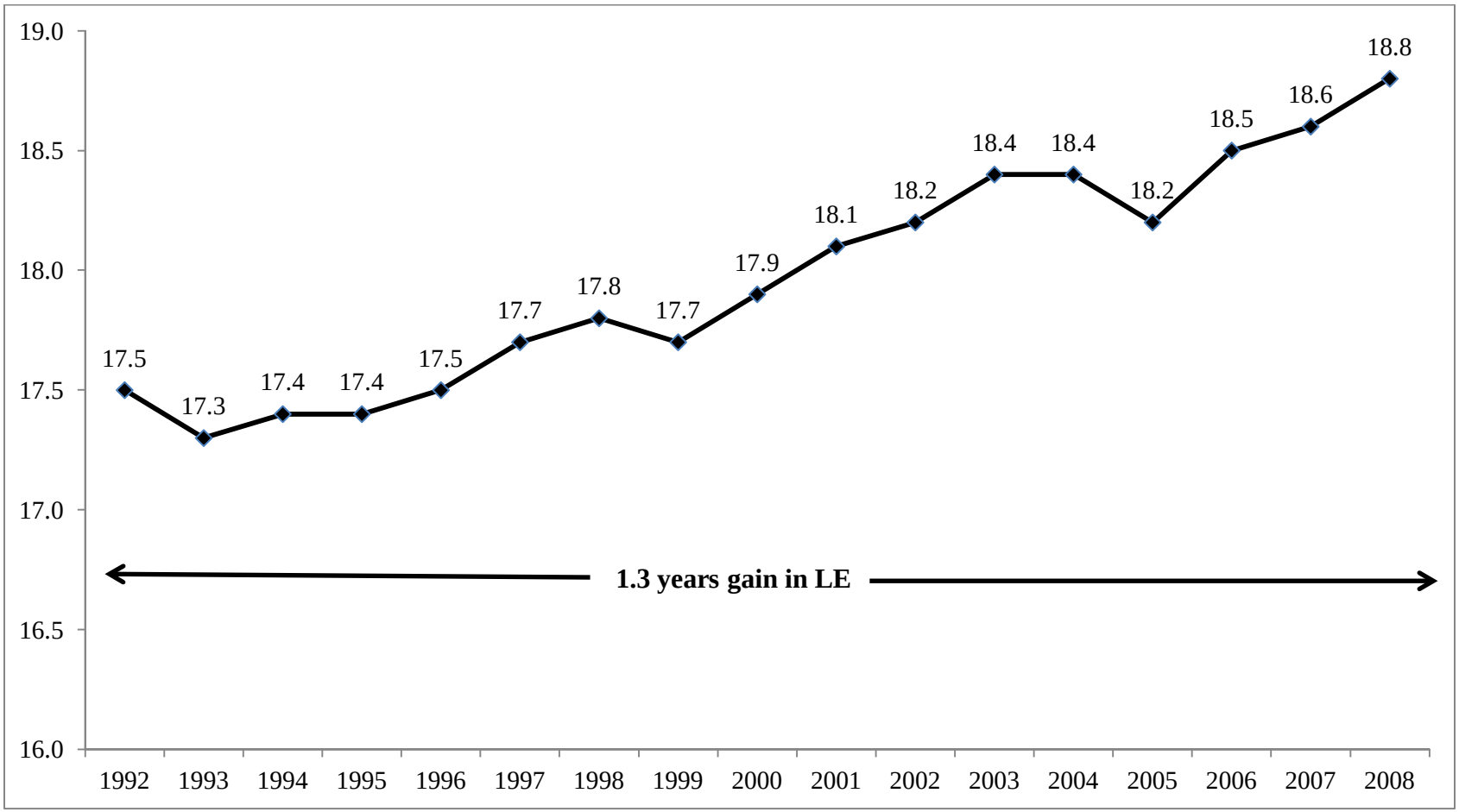
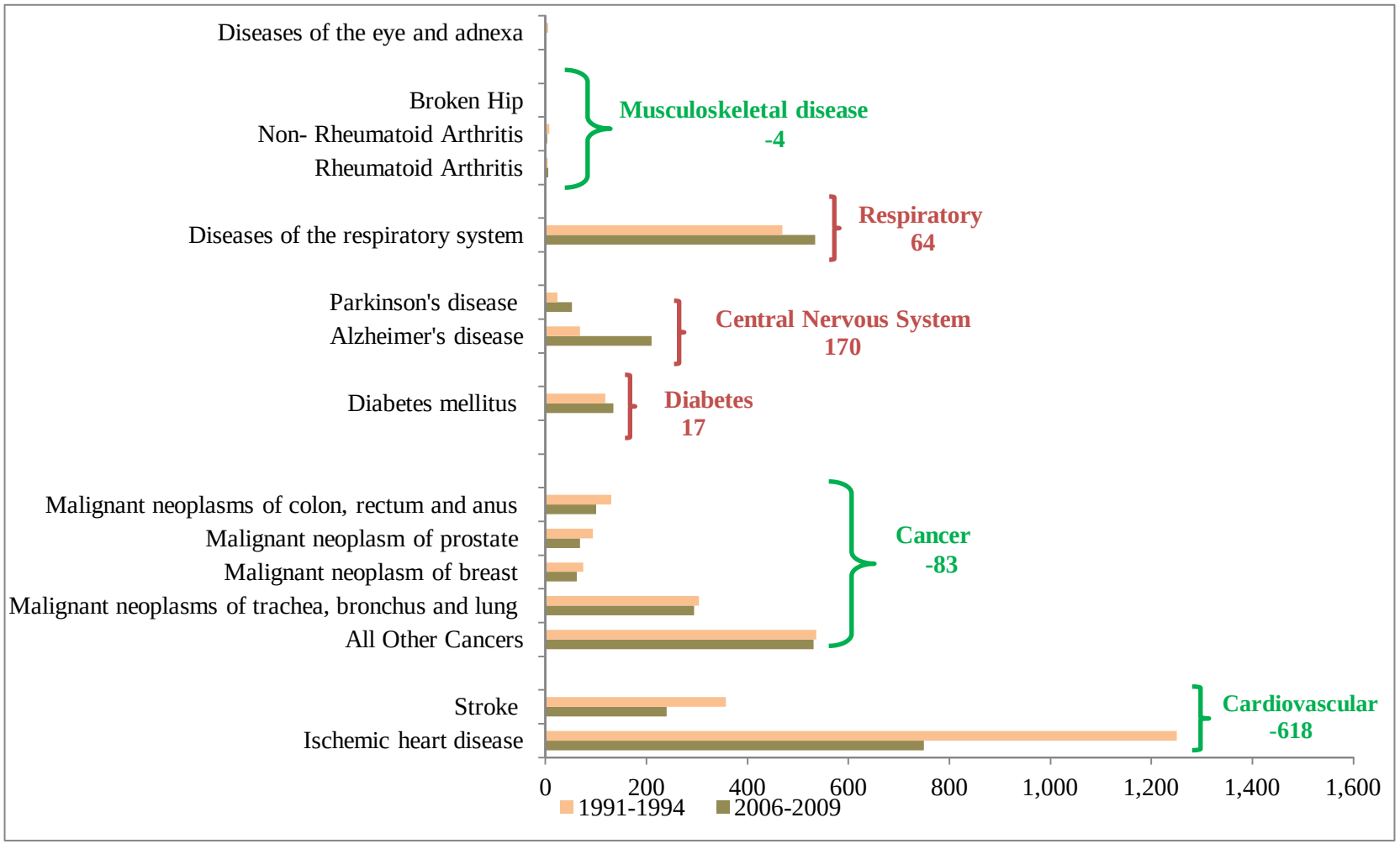
Figure 1: Life expectancy in the US at age 65



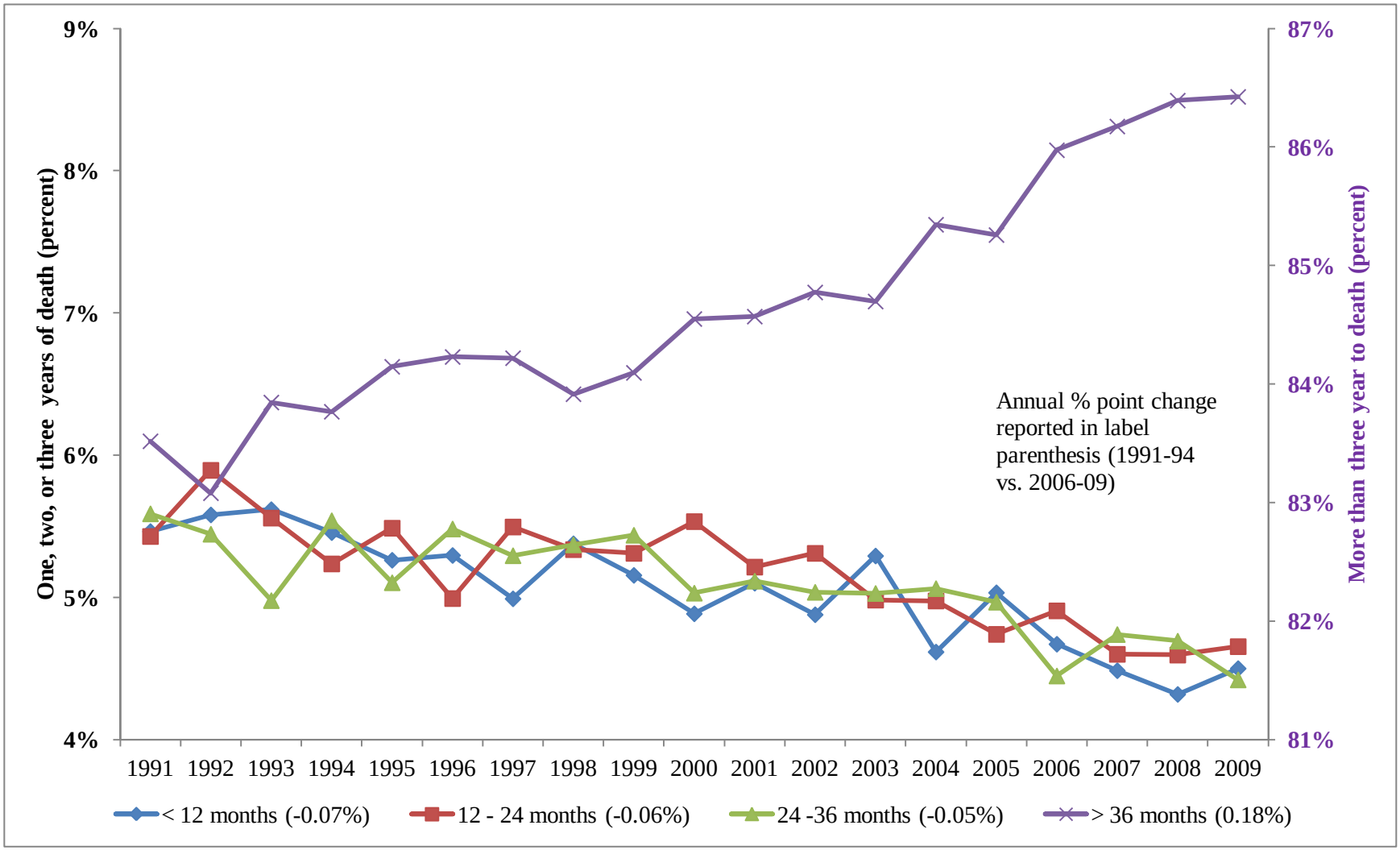
Note: Data are from the Vital Statistics of the United States from the Centers for Disease Control and Prevention/National Center for Health Statistics (NCHS). The annual life tables published by NCHS give life expectancy for the United States by age, gender and race.

Figure 2: NCHS causes of death for people aged 65 years and older: Mortality rates per 100,000 (age-sex adjusted)



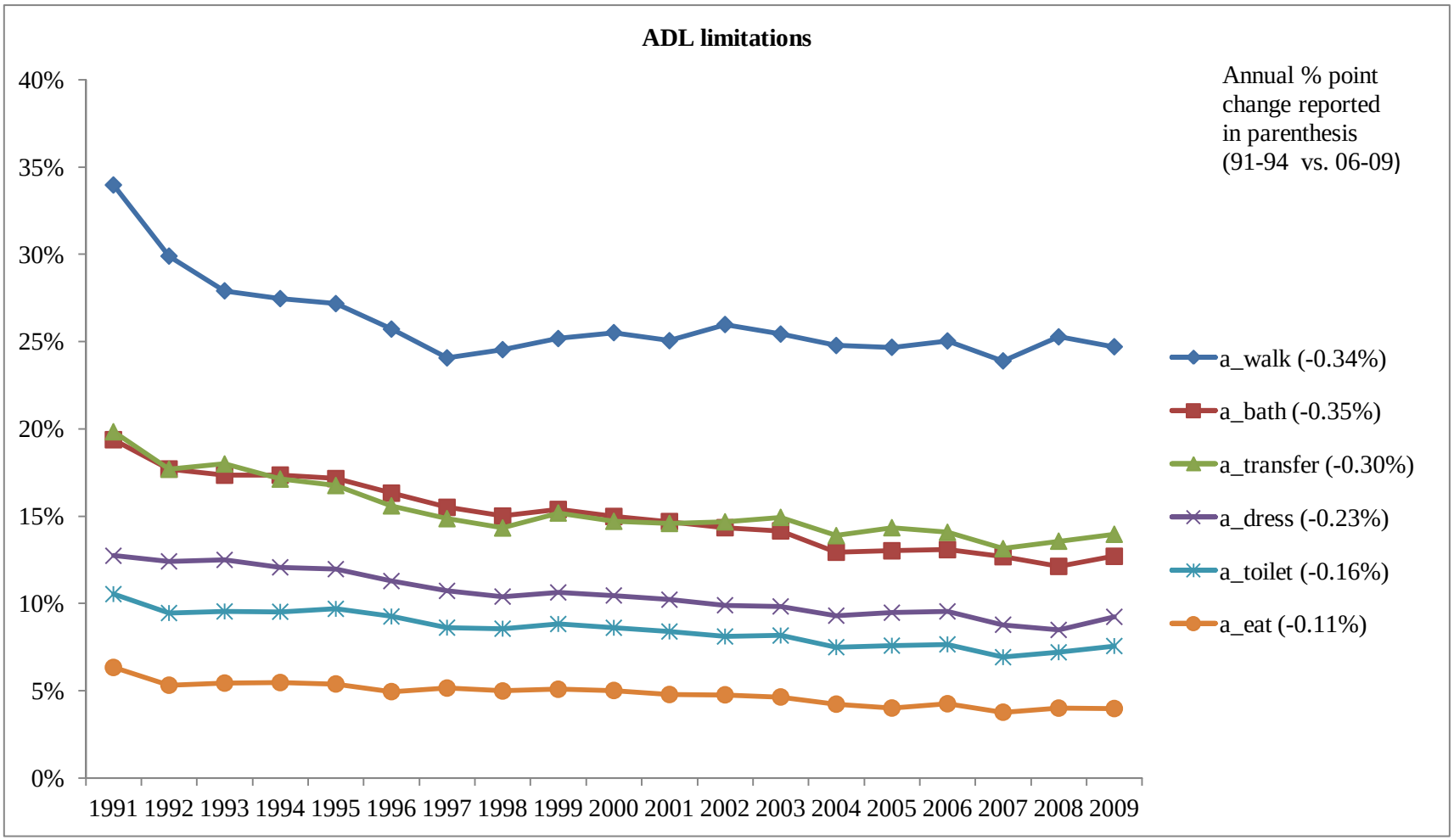
Note: Data are from the Centers for Disease Control and Prevention/National Center for Health Statistics (NCHS). The change in death rate is for two time periods 1991-1994 and 2006-2009.

Figure 3: Population distribution by time until death in elderly Medicare beneficiaries



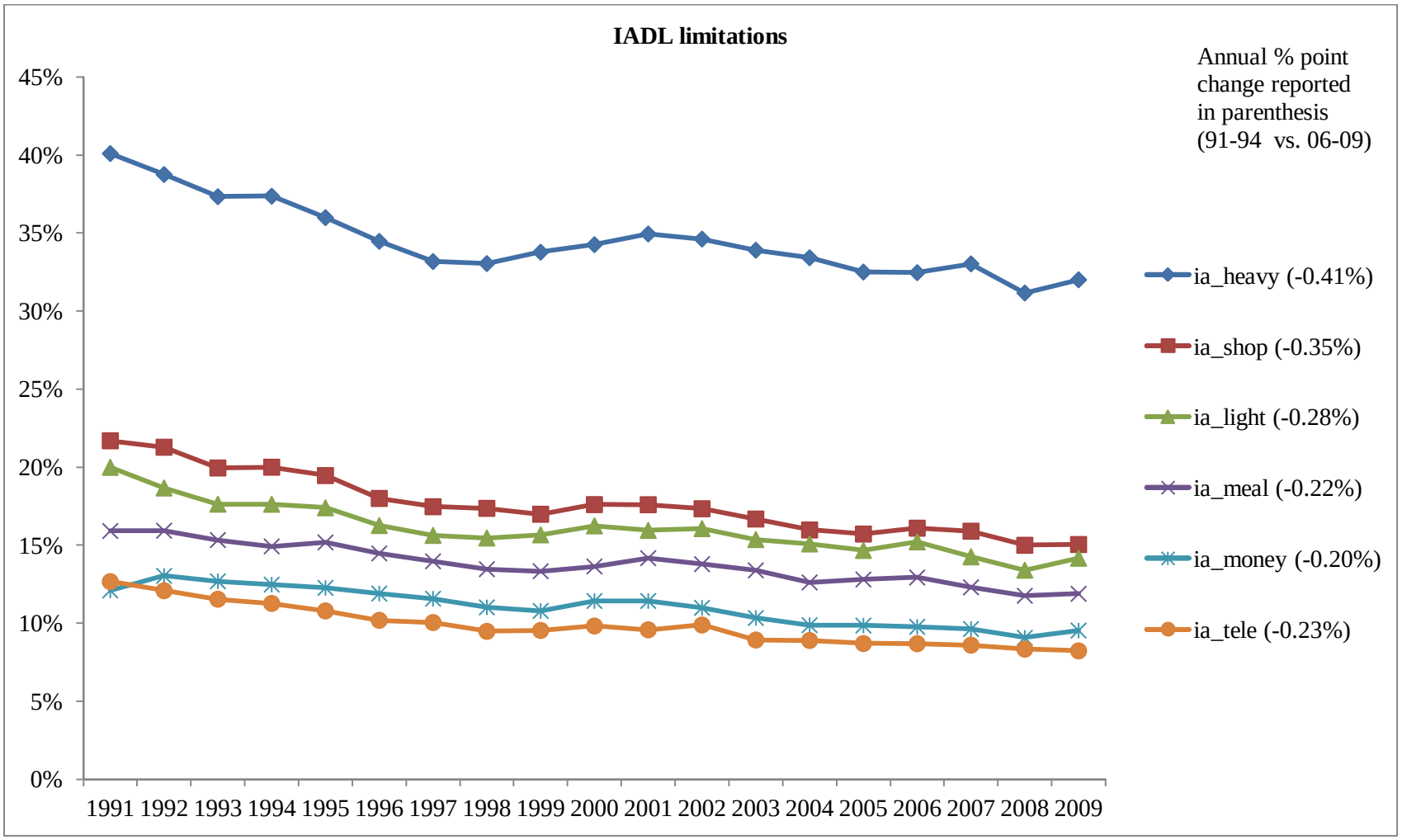
Note: Data are from the Medicare Current Beneficiary Survey and Medicare denominator files linked to MCBS, 1991-2009. Reported statistics is weighted to the population distribution by age, sex, and race in 2000.

Figure 4: ADL and IADL limitations in elderly Medicare beneficiaries



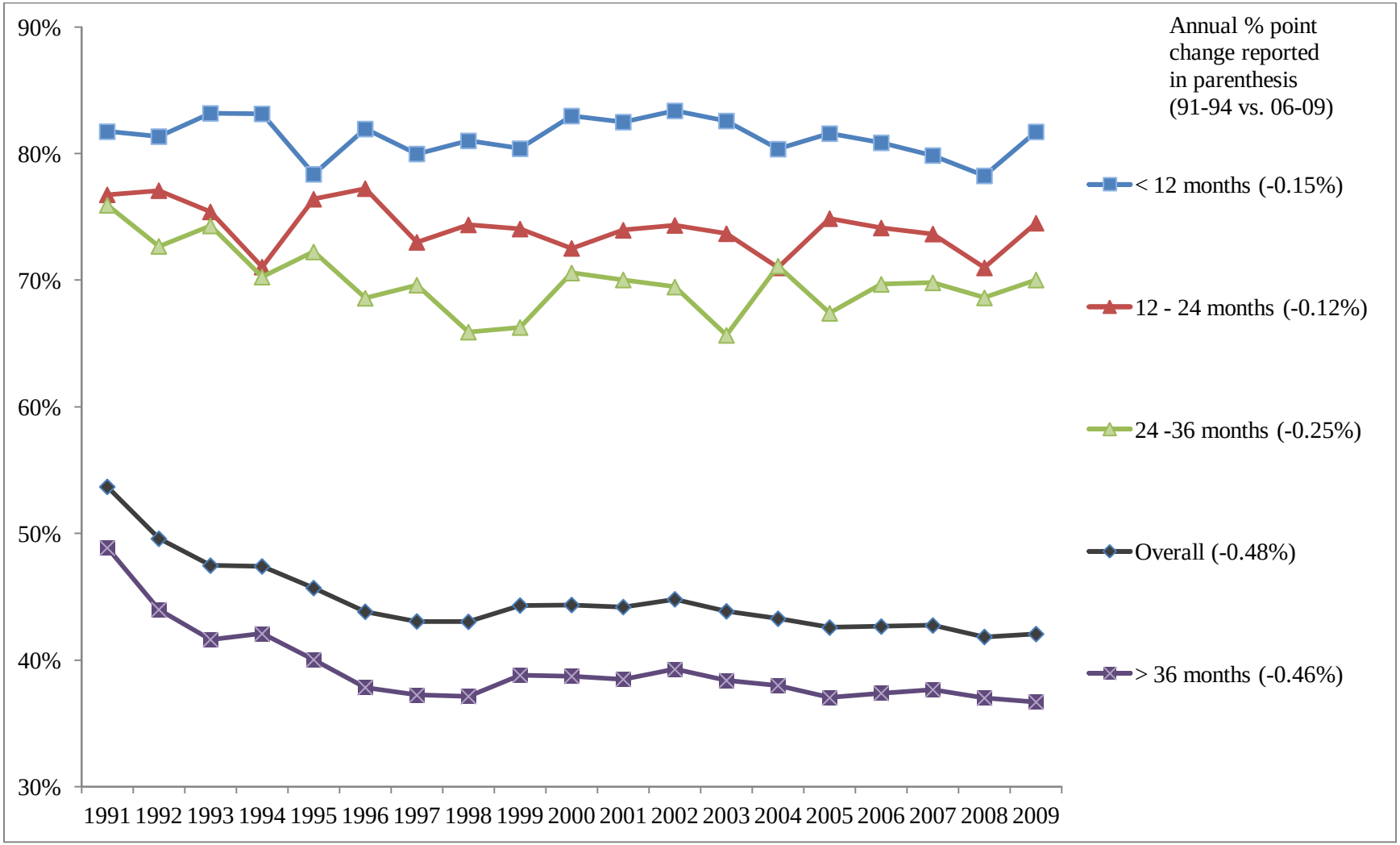
Note: Data are from the Medicare Current Beneficiary Survey, 1991-2009 and are weighted to the population distribution by age, sex, and race in 2000.

Figure 4: ADL and IADL limitations in elderly Medicare beneficiaries (Contd.)



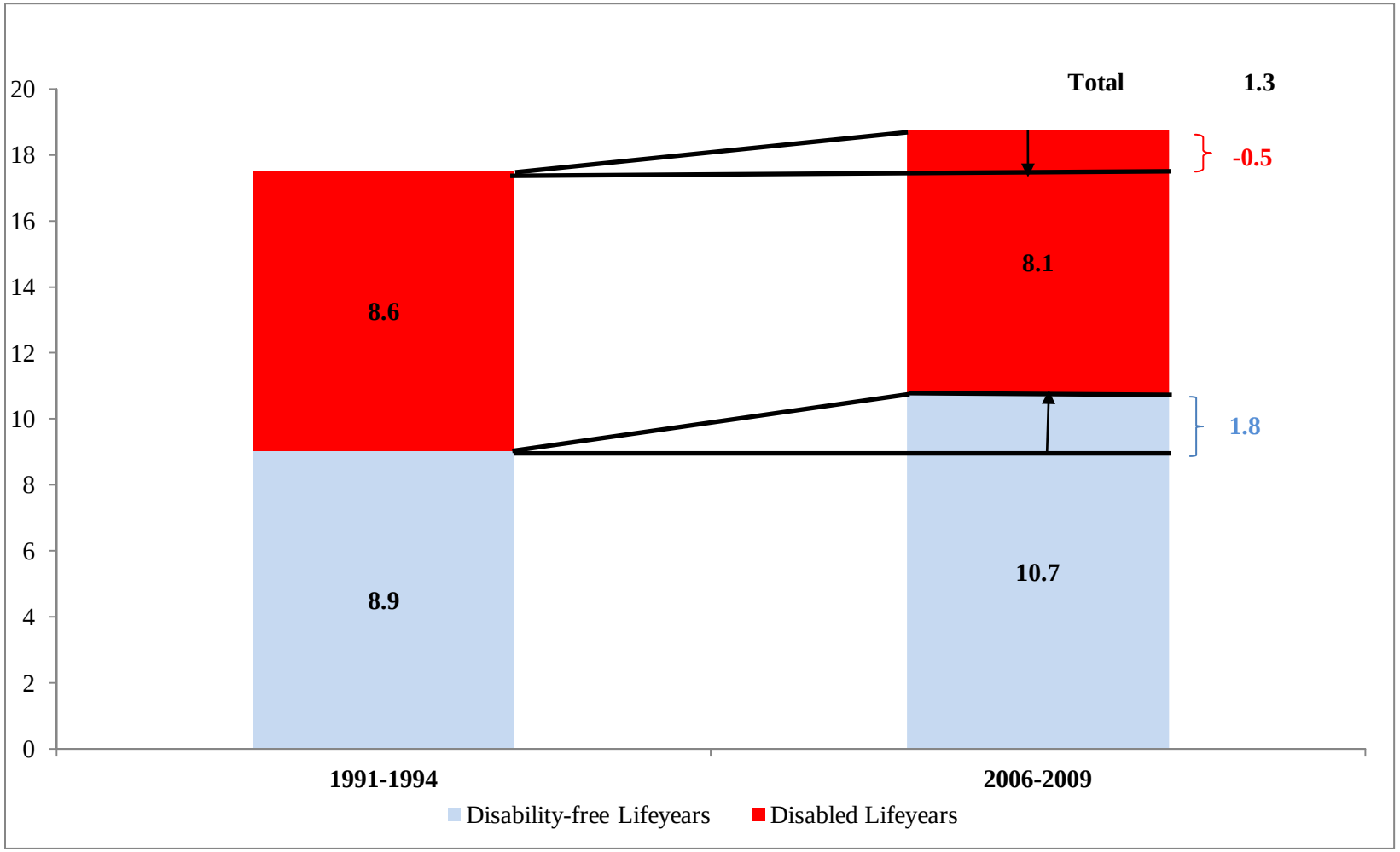
Note: Data are from the Medicare Current Beneficiary Survey, 1991-2009 and are weighted to the population distribution by age, sex, and race in 2000.

Figure 5: ADL/IADL disability by time until death in elderly Medicare beneficiaries



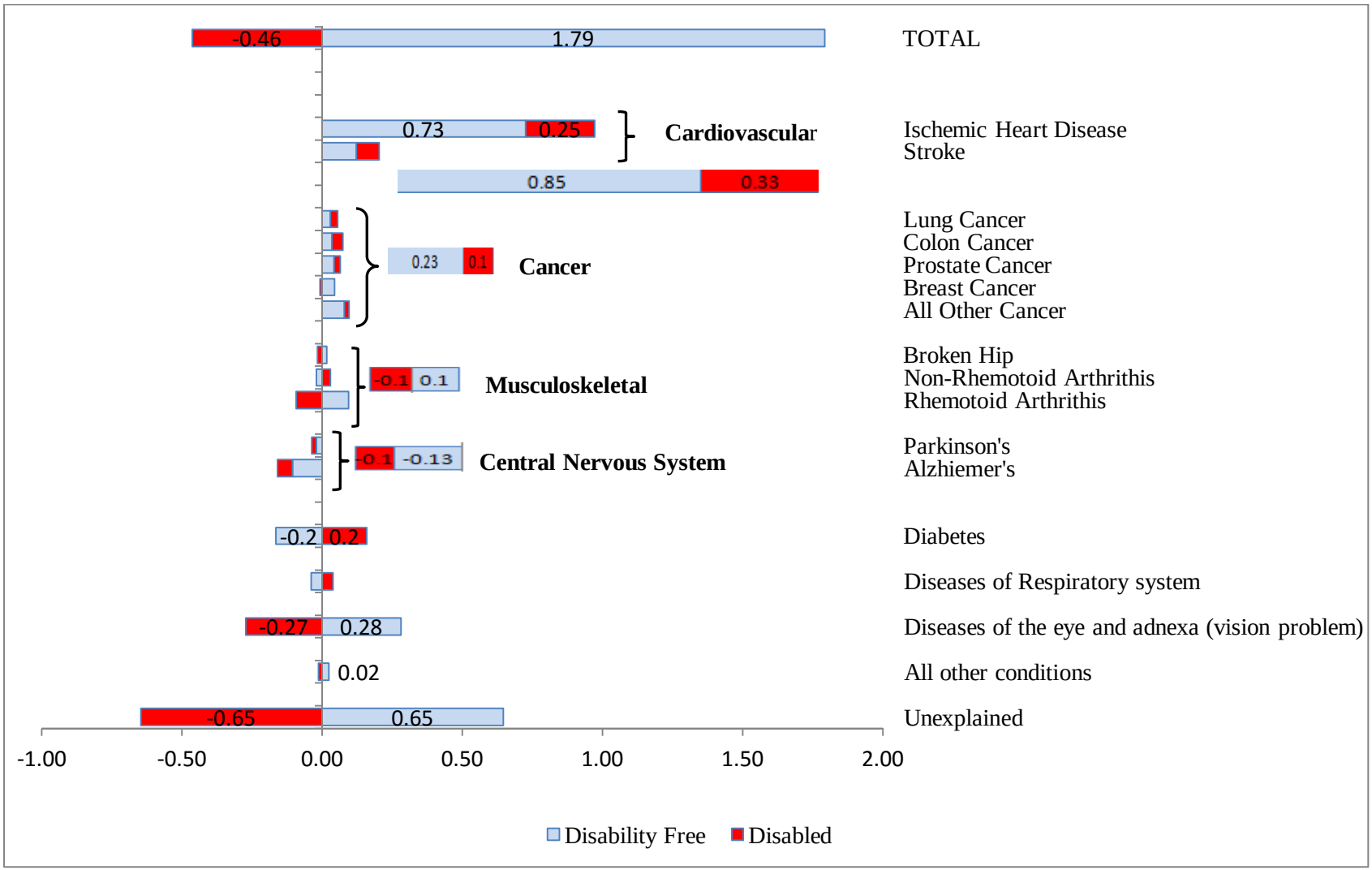
Note: Data are from the Medicare Current Beneficiary Survey and Medicare denominator files linked to MCBS, 1991-2009 and are weighted to the population distribution by age, sex, and race in 2000.

Figure 6: Trend in disabled and disability-free life expectancy at age 65



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.

Figure 7: Change in disabled and disability-free life expectancy at age 65 by disease (1991-94 vs 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.

Figure 8: IHD mortality rates per 100,000 (age-sex adjusted) in elderly Medicare beneficiaries

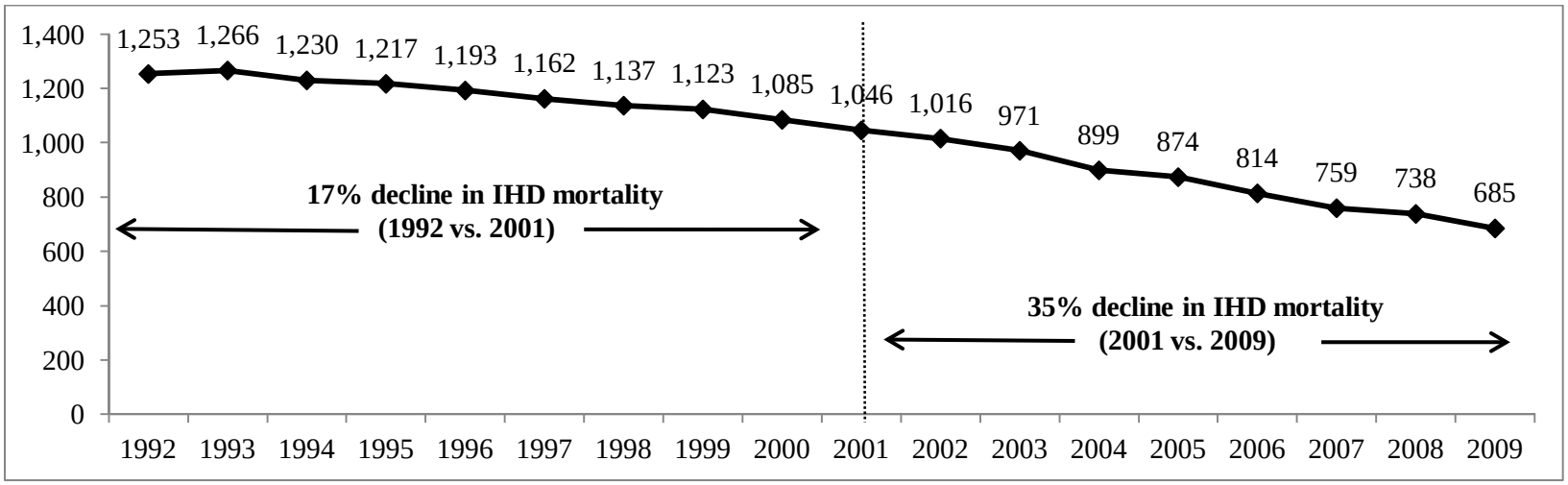
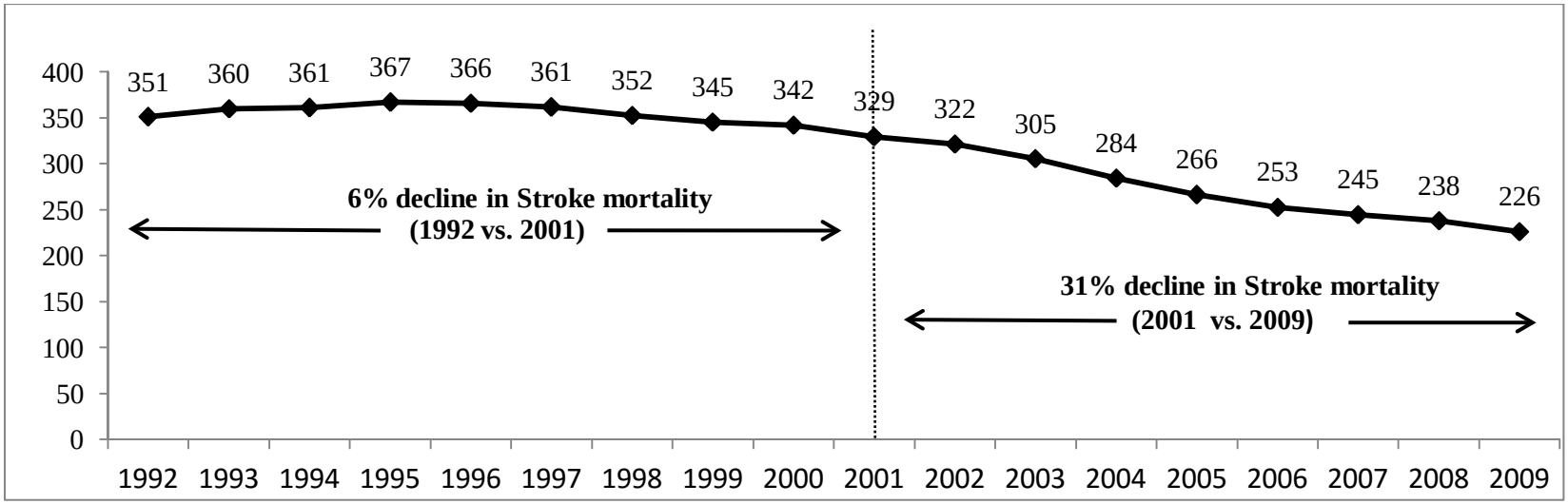
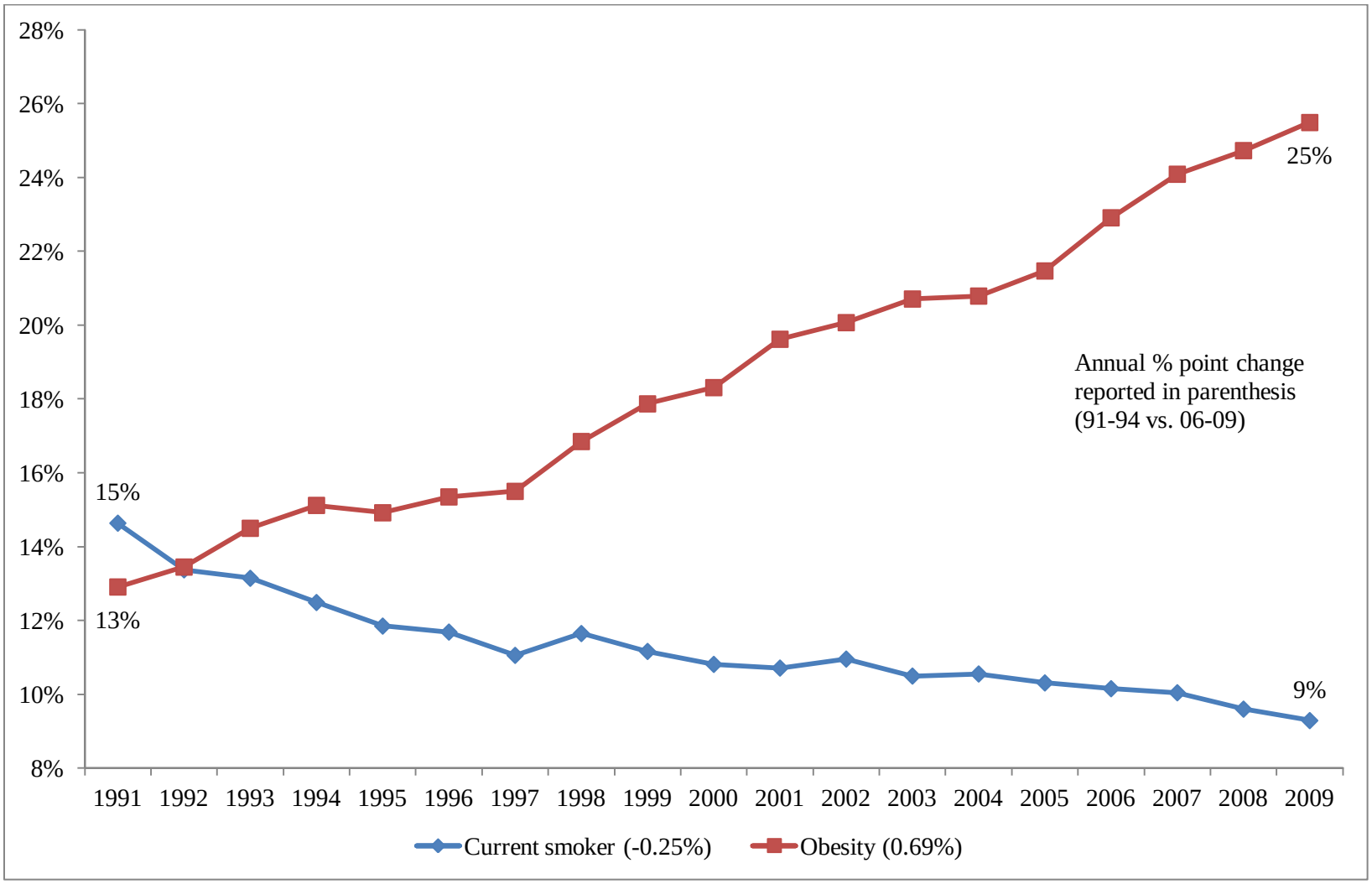


Figure 9: Stroke mortality rates per 100,000 (age-sex adjusted) in the elderly Medicare beneficiaries



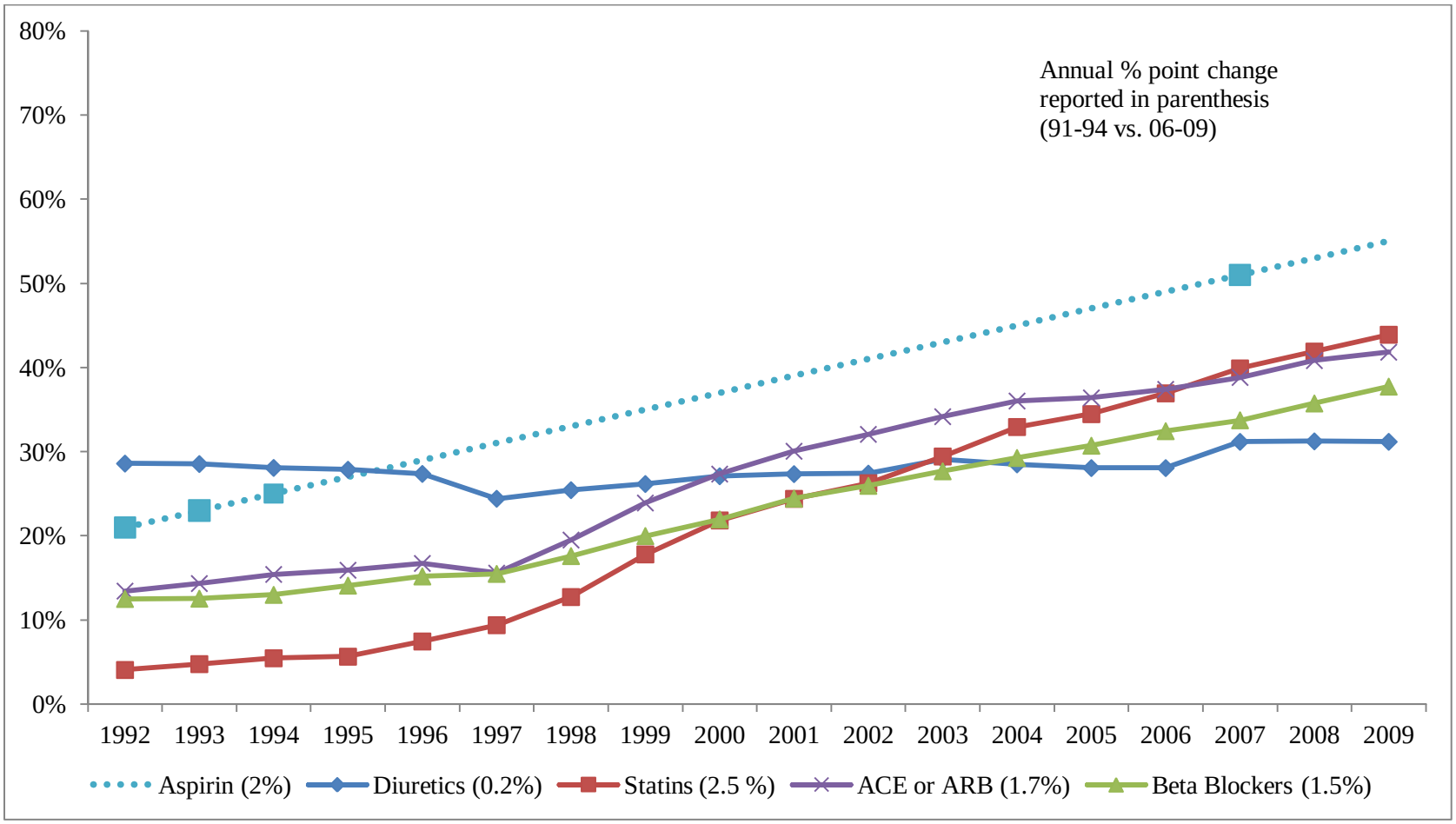
Note: Data are from the Centers for Disease Control and Prevention/National Center for Health Statistics on Causes of Death and micro data on mortality available at the National Bureau of Economic Research.

Figure 10: Smoking and obesity prevalence in elderly Medicare beneficiaries



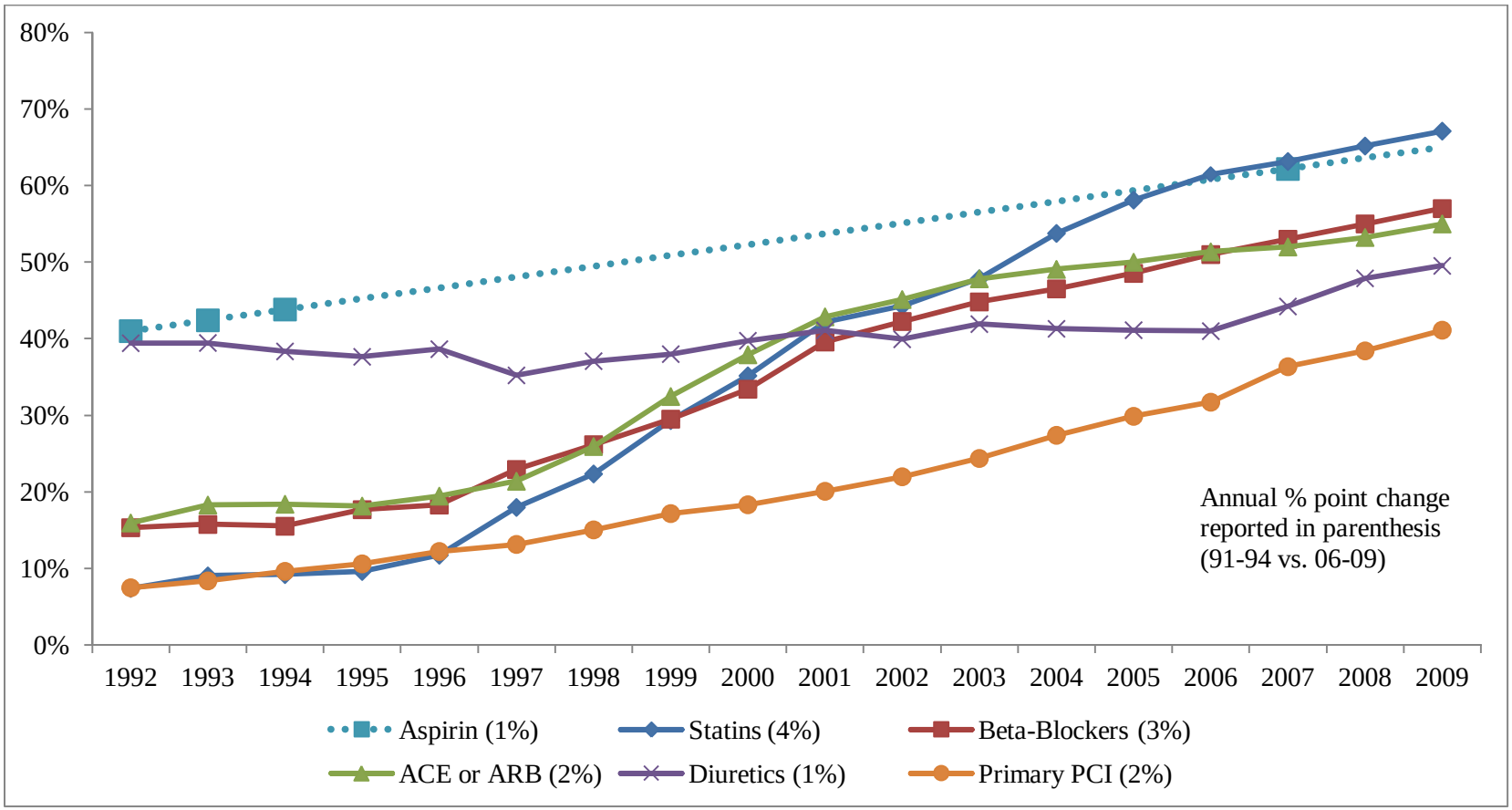
Note: Data are from the Medicare Current Beneficiary Survey and Medicare denominator files linked to MCBS, 1991-2009 and are weighted to the population distribution by age, sex, and race in 2000.

Figure 11: Trends in cardiovascular medication usage in elderly Medicare beneficiaries



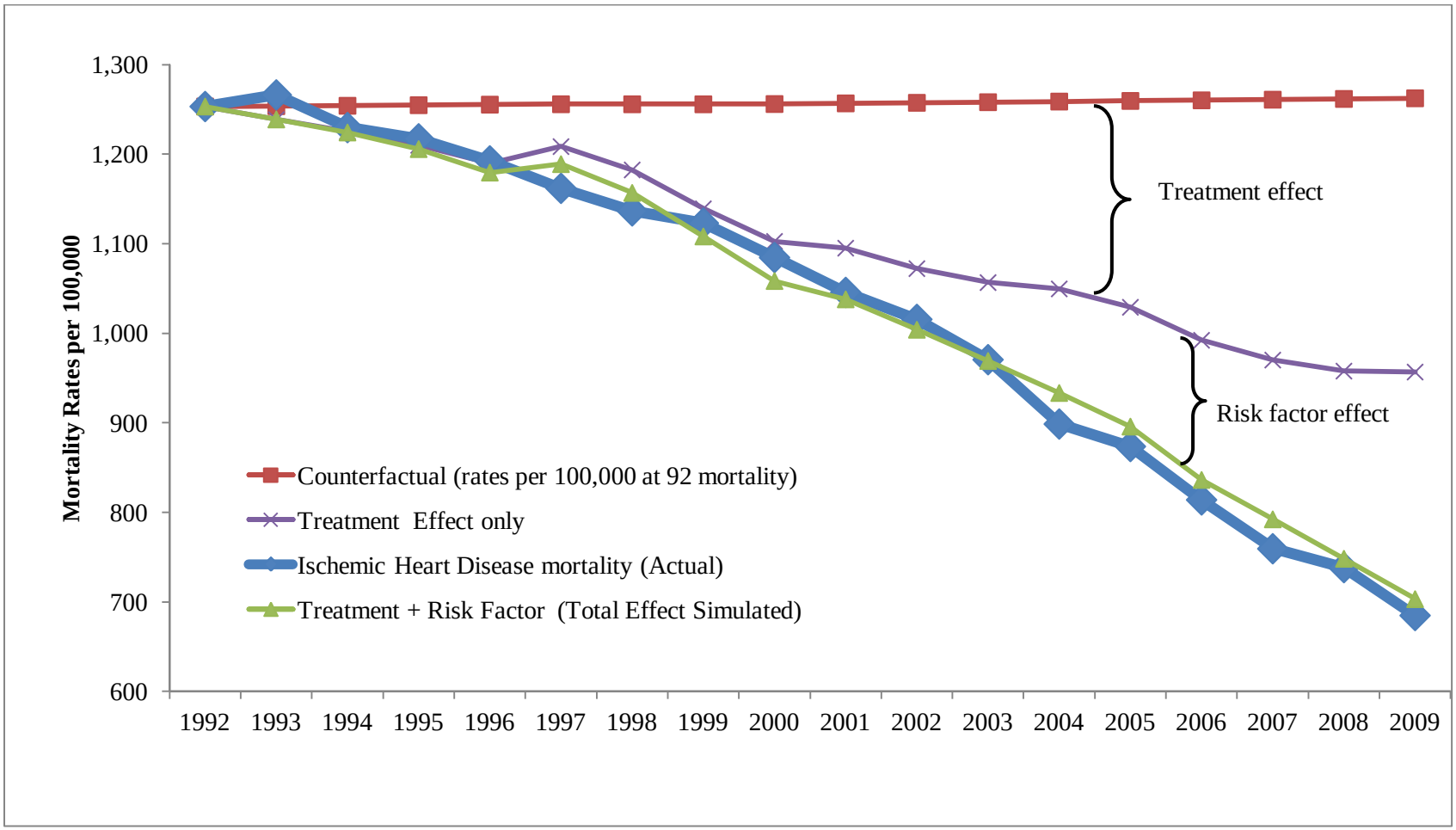
Note: Data on medication usage (statins, ACE or ARB, Beta-blockers, and diuretics) is from Prescribed Medicine Events in the MCBS data. Rates are adjusted to population by age, sex, and race in 2000. The aspirin usage from the 1992-94 period is from NHANES III and the later period is from literature based on the 2007 MEPS. The intermediate years are linear interpolations.

Figure 12: Trends in medication usage in ischemic heart disease patients in elderly Medicare beneficiaries



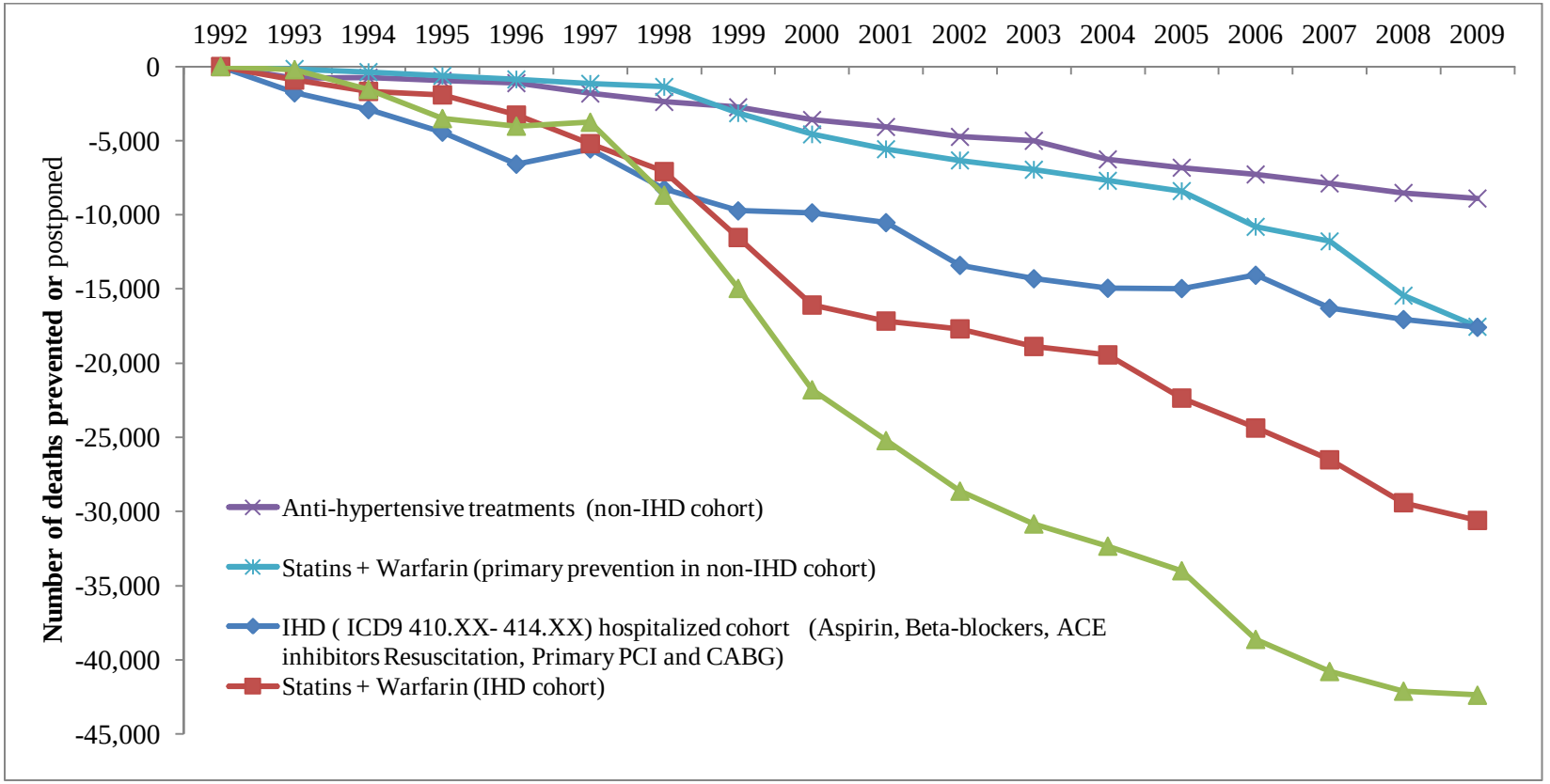
Note: Data on medication usage (statins, Beta-blockers, ACE or ARB, diuretics) is from Prescribed Medicine Events in the MCBS data. Rates are adjusted to population by age, sex, and race in 2000. Aspirin usage for the earlier period is from NHANES III and the later year is from MEPS. The intermediate years are linear interpolations. Primary PCI usage is from 5% 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.

Figure 13: Ischemic heart disease mortality rates per 100,000: Actual vs. simulated in elderly Medicare beneficiaries



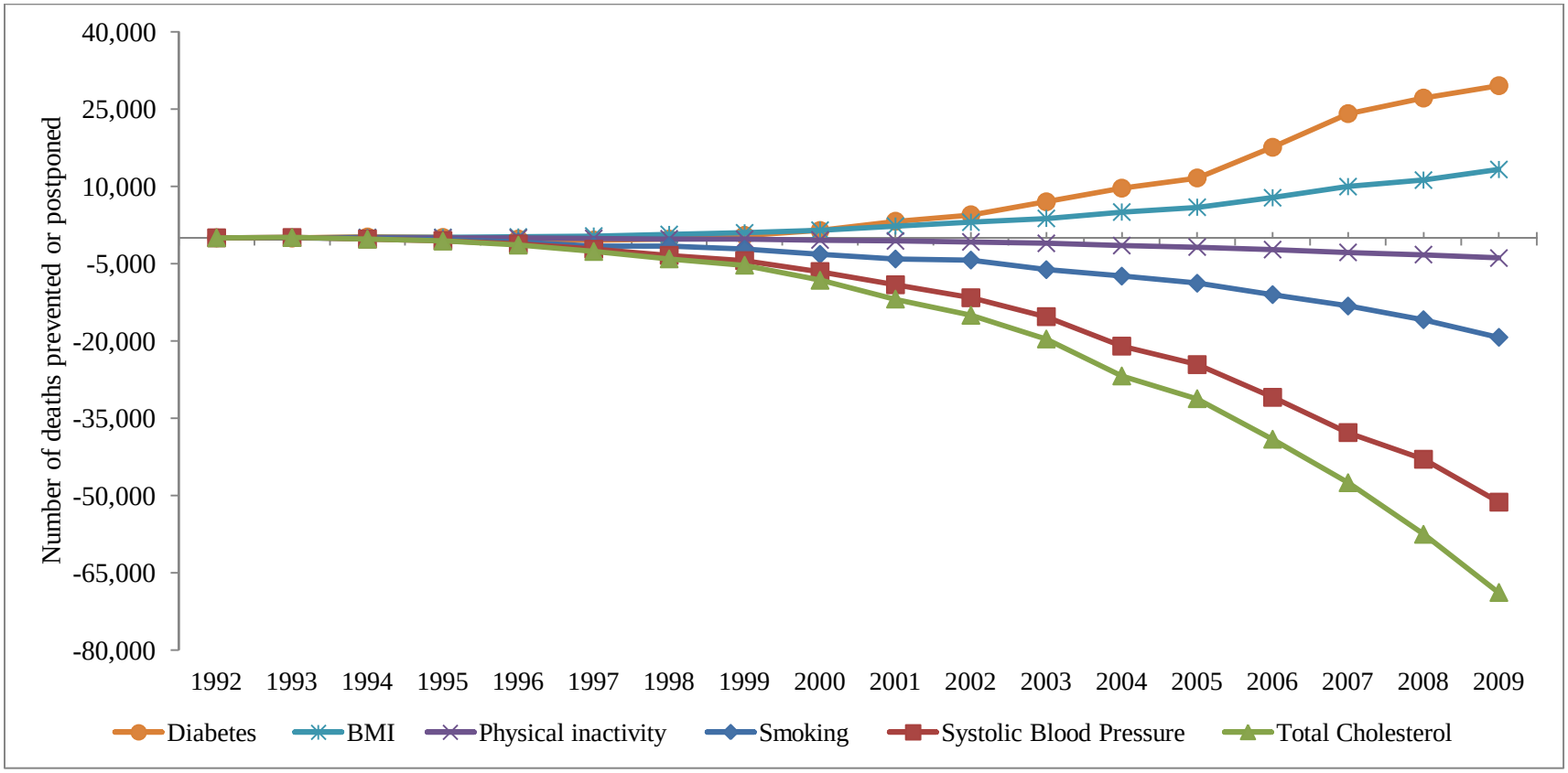
Note: Data are from the Centers for Disease Control and Prevention/National Center for Health Statistics on Causes of Death and the US Census of population. The line with squares shows the counterfactual mortality rate per 100,000 if the mortality rate by age remained at 1992 level and only the population has changed. The line with diamonds shows the actual mortality rate. The line with triangles shows the simulated effect of treatment and improvements in risk factors combined on mortality. The line with X's shows the impact of treatment only on mortality.

Figure 14: Estimated deaths prevented or postponed in the elderly United States population: Treatment effect in elderly Medicare beneficiaries



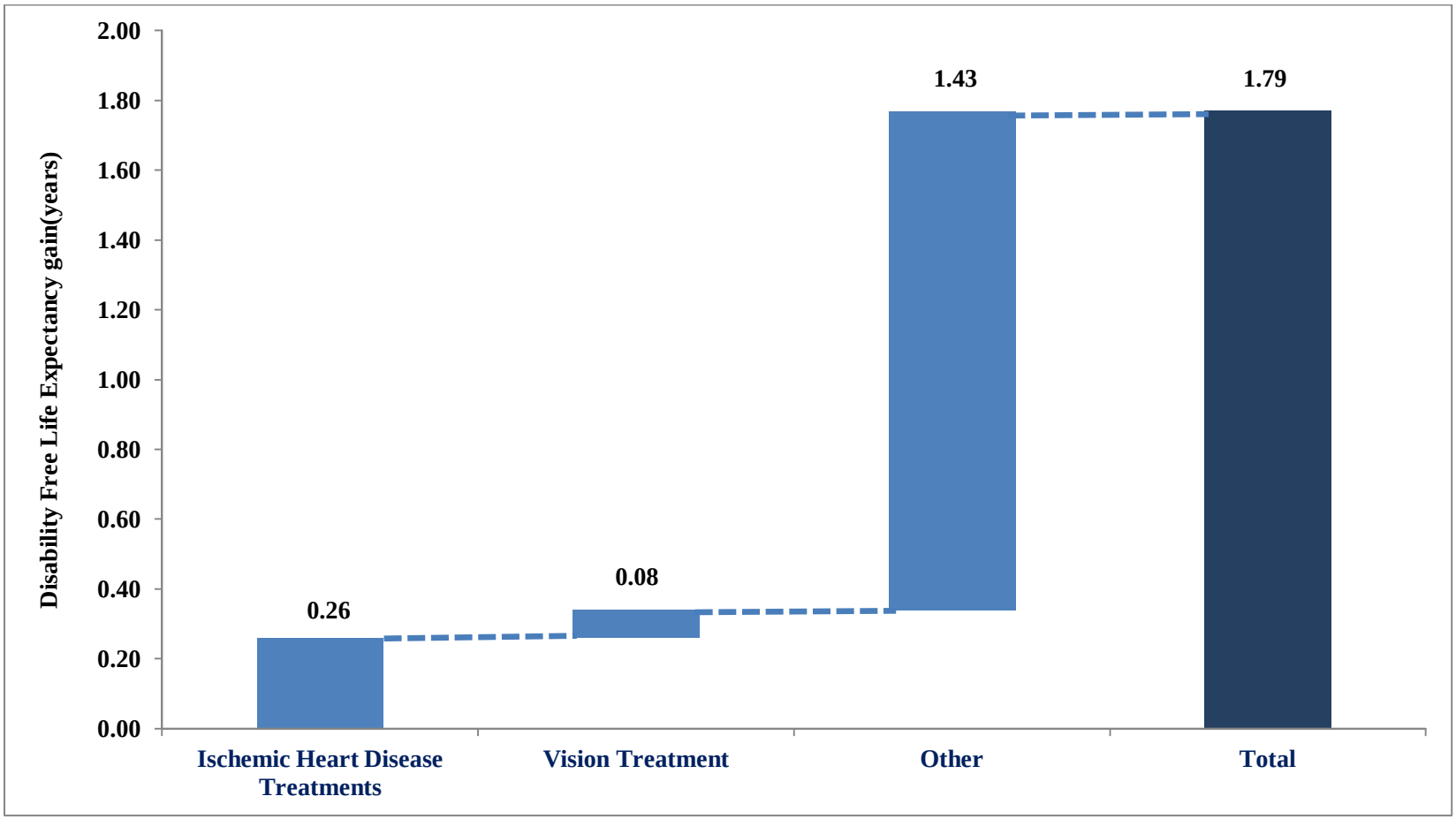
Note: The figure shows the effect of individual medication on mortality. The treatment effects are calculated using data from NHDS, MCBS, Medicare and other sources cited in Ford et al. (2007). For example, between 1992 and 2009, the increased use of statins for primary and secondary prevention saved roughly 48,000 lives. Increased use of Ace inhibitors and beta-blockers in ischemic heart disease patients saved roughly 43,000 lives.

Figure 15: Estimated deaths prevented or postponed by risk factor changes in elderly Medicare beneficiaries



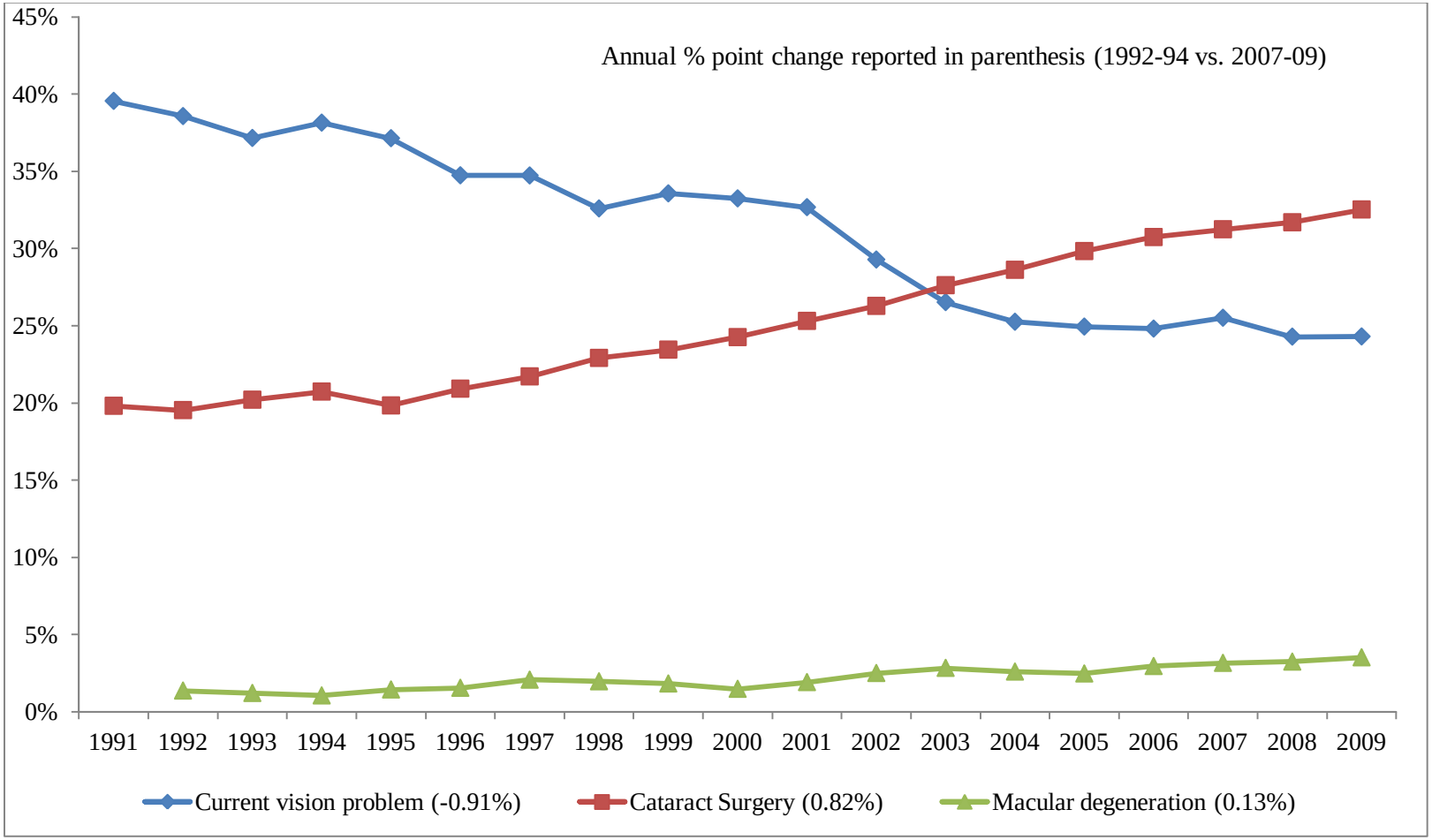
Note: Figure 15 shows the effect of individual risk factors on mortality. The risk factors are calculated using data from NHANES and MCBS and Ford et al, (2007). For example, between 1992 and 2009, roughly, 58,000 deaths were prevented due to lower total cholesterol, and 51,000 deaths prevented due lower systolic blood pressure. On the other hand, increased incidence of diabetes and BMI caused roughly 43,000 additional deaths.

Figure 16: Impact of treatments on changes in disability-free life expectancy at age 65



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.

Figure 17: Cataract surgery and vision problems in the elderly Medicare beneficiaries



Note: 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.

Table 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%

Note: Tabulations are from the MCBS Access to Care sample for 1991-2009 and restricted to elderly Medicare beneficiaries**.** We use sample weights adjusted to a constant year 2000 population by age, gender, and race. This table is based on 251,872 person-year observations.

Table 2: Self-Reported Medical Event Questions in the MCBS

Num	Ever told have	Prevalence	Annual % point change (1991-94 to 2006-09)
	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%

Note: Tabulations are from the MCBS Access to Care sample for 1991-2009 and restricted to the elderly Medicare beneficiaries. We use sample weights adjusted to the year 2000 population by age, gender, and race. This table is based on 251,872 person-year observations.

Table 3: Regressions Explaining Disability in elderly Medicare beneficiaries

					Oaxaca Decomposition		
	Prev. 91-94	Coeffs. (Robust Std. Err.) 91-94	Prev. 06-09	Coeffs. (Robust Std. Err.) 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%**
ALZHEIMERS	4.7%	0.25 (0.01)	5.8%	0.28 (0.01)	0.1%	0.2%	0.4%
PARKINSONS	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 ARTH	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%
BREAST 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%**

Table 3 (continued)

	Prev. 91-94	Coeffs. (Robust Std. Err.) 91-94	Prev. 06-09	Coeffs. (Robust Std. Err.) 06-09	Oaxaca Decomposition: Effect of Change in Beta X*DBETA	Oaxaca Decomposition: Effect of Change in X BETA*DX	Oaxaca Decomposition: Net Effect
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 2006-09. 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 β's, decomposed into those for conditions, those for demographics, and the constant term. Robust standard errors are reported in parenthesis.

Table 4: Prevalence of Cardiovascular Risk Factors in NHANES Subjects Aged ≥ 65 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 2001-02 and 2003-04; NHANES 2005-06 and 2007-08; and NHANES 2009-10 and 2011-12.

Table 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. Risk factor estimates are from NHANES. The treatment data comes from several sources including NHDS, Medicare data, MCBS and some other studies.

Table 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)

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 51-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 was developed for the Medical Outcomes Study (MOS).
SF-36 is a 36-item, patient-reported survey of patient health commonly used to determine the cost-effectiveness of medical treatments.

Table 7: Vision problem acuity and disability states in the elderly
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
Non-White	-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	

Note: 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.