Preliminary

Understanding the Improvement in Disability Free Life Expectancy In the U.S. Elderly Population

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Understanding how healthy lifespans are changing over time is central to public policy. A good deal of medical spending is predicated on the idea that more intensive treatments improve quality-adjusted life expectancy. Measuring healthy life expectancy is thus a first start in understanding the value of medical advance. Further, 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.

Data on life expectancy is easy to obtain, but data on healthy life expectancy is 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, 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 impairment with any Activity of Daily Living or Instrumental Activity of Daily Living. We calculate the number a years a person turning 65 in different years can expect to live with and without a disability.

Our past research (Cutler et al., 2014) 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. 1997, 2001, 2009), though the data used in past studies were less complete, for reasons we explain briefly below and in more detail in our previous paper. 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 of time 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 high cholesterol. 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 observe trends in treatments and health, but we have little way to turn these trends into causal statements. To make a stab at the causal question, we use an indirect methodology. We combine trends in treatments with over time with clinical trial evidence on the impact of different treatments on mortality and disability. We use this to estimate the gain in

disability-free life expectancy that would be predicted to have resulted from medical advance, and then compare this to the actual improvement in health from these conditions.

In the case of cardiovascular disease, our results are extremely promising. Use of effective treatments has improved at a rate that the clinical literature suggests would have led to roughly the health improvements that we observe. The vision analysis is more paradoxical. Ever 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. However, the clinical literature does not suggest a meaningful impact of cataract surgery on disability. We thus find it difficult to draw firm conclusions about the role of cataract surgery in explaining the increase in disability-free life expectancy.

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

We measure health in two dimensions: life expectancy, and disability. 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 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 1** shows the change in life expectancy at 65 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 paper, 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 cause of death data.

Death codes change over time, and so the mix 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 15 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 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 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 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 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, the biggest reduction is in cancer of the trachea, bronchus, and lung – a cause strongly associated with tobacco use. However, other preventive efforts and medical treatments likely play a role in declining cancer mortality as well (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.

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 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.

The MCBS population becomes older and less white over time, as the elderly population changes demographically. We do not want to show trends that are influenced by these

demographic changes. We thus 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, dyed 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 about 5 percent on average. Reflecting the overall reduction in mortality, this share is declining over time (this is true of the population 1-2 years from death and 2-3 years from death 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 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 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 walking

declined the most, by 0.34 percentage points annually. The bulk of this decline happened in the first part of the time period, but this is not true about all ADL limitations. Other ADL difficulties also declined over the 18 years: bath or showering (0.35 percentage point 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. Crimmins (2004) reported similar trends in heavy house work during 1992-1998.

The disability metric we use is the share of the population that reports any ADL or IADL limitation. Using this definition, disability 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. Most studies in the 1980s and 1990s found that despite the increasing pattern in chronic diseases, functioning was improving (Crimmins et al., 1993, 1997, 2004; Manton et al., 1993, 1997, 2001, 2006). Other studies with similar conclusions include Freedman et al. (1998, 2002, 2004), Schoeni et al. (2001), and Cutler (2001).

A sharp decline in walking problem 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).

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 period 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 et al., 2001), 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 a decline of 0.46 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 et al., 2007, Cutler et al., 2014). In the next section, we combine the NCHS period life tables and 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 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:

$$LE(a) = \Sigma s \{Pr[Survive\ a+s \mid Alive\ a] + .5*Pr[Die\ at\ a+s \mid Alive\ a]\} \quad (1)$$

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 in two time periods: 1992 and 2008. The mortality data are from those exact years. The disability data are from 1991-94 and 2006-09. Although, disability data is available for individual years, we used the combined sample to provide more precise estimates.

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, we instead use regression analysis to smooth disability rates by age and time until death. Specifically, we estimate a regression model relating disability to medical conditions, 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, both the metric of 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, et al., 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, and disabled life expectancy fell 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 Global Burden of Disease study, (JAMA, 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. However, few results are reported by age group, and many of the top conditions are less relevant in elderly populations (for example, road injuries). The study found that leading risk factors related to disability-adjusted life-years (DALYs) were dietary risks, tobacco smoking, high body mass index, high blood pressure, high fasting plasma glucose, physical inactivity, and alcohol use. Other studies looking at the burden of diseases include Wang et al. (2010), Salomon et al. (2013), and Murray et al. (2013).

Cutler, Landrum, and Stewart (2007) 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. Cutler, Landrum and Stewart (2009), 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. 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 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 increased modestly (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 factor. Our equation is of the form:

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

where i denote individuals, and t denotes the period (1991-94 or 2006-09). Demographics include ten age-sex dummy variables and time to death dummy variables. Individual may show up multiple times in the regression, depending on how frequently they are sampled. For accurate standard errors, this should be accounted for. Our focus here is on the coefficients, however. We thus do not report 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. 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.

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 this decline are cardiovascular disease (2.5 percentage points) and vision problems (1.7 percentage points). 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 by medical conditions and demographics. This may include environmental factors (ramps, disability accessible buildings) or changes in living conditions (married, assisted living) that are getting better 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. 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 disability 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 will 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. Consistent with previous literature (Cutler, Landrum, and Stewart, 2009) cancer remains a major source of mortality but is relatively minor in its contribution to disability. The disability-free life expectancy gain from cancer is about 0.23 years. Vision problem shows 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.

Finally, there is a big "unexplained" part with no effect on mortality, but a significant impact on morbidity. The increase in disability-free life expectancy that is unexplained is about 0.65 years. This is the residual change in disability noted above, coming from reduced disability among people further away from death.

Overall, the most important gains in 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 and incidence rates 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). And deaths from

cardiovascular disease have greatly declined among the elderly in the United States over the past decades (Rosen, 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 the heart muscles to rupture. A stroke happens when poor blood flow to the brain or a hemorrhage in the brain results in cell death. Historically, heart attack and strokes 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 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%).

To better understand the impact of preventive technology (especially pharmaceuticals) for cardiovascular disease (Downs et al., 1998; Weisfeldt et al., 2007), we next examine the incidence of hospitalization from ischemic heart disease and stroke. Although, we have claims data in MCBS, the number of people being hospitalized for acute events is small. For this reason, we use Medicare claims data. We use a 5% random sample of Medicare data for beneficiaries 65 years and older. Because we have only data on the Traditional Medicare (fee-for-service) population, our trends are limited to that group. We restrict our sample to beneficiaries who are

enrolled in Traditional Medicare for all 12 months to avoid missing claims for switchers (FFS to HMO and vice-versa).

We use the CMS Medicare Provider Analysis and Review (MedPAR) to identify inpatient hospitalizations for ischemic heart disease or stroke as listed in the principal discharge diagnosis code. The ICD9-CM codes defined to include ischemic heart disease are 410.X (Acute myocardial infraction), 411.X (Other acute and subacute forms of ischemic heart disease), 412.X (Old myocardial infraction), 413.X (Angina pectris) and 414.X (Other forms of chronic ischemic heart disease). Stroke hospitalizations were identified by primary discharge diagnoses defined using International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) codes of 434.x and 436.x for ischemic stroke and 430.x and 431.x for hemorrhagic stroke.

Trends in incidence for ischemic heart disease and stroke are reported in **Figures 10 and 11**. The rates are per 100,000 people and are age and sex adjusted. For ischemic heart disease, there is almost no decline in incidence between 1992 and 2001, although after 2002 ischemic heart disease hospitalization declined significantly. To some extent, the lack of a reduction in incidence prior to 2002 may be a result of diagnostic changes. Over the 1990s, a new, very sensitive blood test to detect a heart attack, troponin, was widely used. The literature suggests that troponins might have increased heart attack incidence by as much as 15 to 30 percent (Alpert et al. 2003). The increase in ischemic heart disease incidence between 1995 and 2000 could correspond to increase testing with troponins. Even with this change, overall ischemic heart disease hospitalization declined 31 percent from 1992 through 2009.

The decline in stroke hospitalization is more continuous. The overall change is 35% over the time period, with a clear trend of declining incidence since 1997. Overall, both incidence and mortality for cardiovascular disease is declining.

TO COME, SOMETHING ON METHODOLOGY AND OTHER POSSIBLE APPROACHES.

To explain the reduction in incidence of heart disease and stroke, we examine trends in risk factors for cardiovascular disease. We use data from NHANES (National Health and Nutrition Examination Survey), a survey that directly measures cardiovascular risk factors like 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. The elderly population has become more obese. Even still, total cholesterol is falling in both men and women, and HDL cholesterol (good cholesterol) is increasing. 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 is increasing in both men and women, but people are doing better in managing their Hemoglobin A1C (HbA1c). overall, cardiovascular risk management is getting better over time.

Smoking and obesity are the two most significant risk factors for cardiovascular diseases. The rise in obesity clearly increases the chances of diabetes and other vascular diseases. (Goran et al., 2003, Weisfeldt, 2007). **Figure 12** shows the trends in smoking and obesity in the elderly Medicare population. The data show opposite trends in the two risk factors: rising obesity and falling smoking.

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, preventing clots in the arteries and thus reducing cardiovascular risk. Statins

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), diuretics. Aspirin use is also increasingly common. Literature suggests that low-dose aspirin helps reduce cardiovascular disease incidence and recurrence.

Figure 13 shows the trends in the use of Statins, beta-blockers, ACE inhibitors or angiotensin receptor blockers (ARBs), and diuretics in the elderly community population. The data on medication usage is from the Prescribed Medicine Events file that contains cost and utilization of prescribed medicines for the MCBS community population. Statins usage increased the most (2.1 percentage points annually), though the use of beta-blockers (1.4 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). The effectiveness of diuretics in preventing cardiovascular agents is similar to other antihypertensive agents and is now recommended more for the treatment of hypertensive patients (Chobanian et al., 2004). Aspirin is available over the counter (OTC). We get usage in the earler 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. We show the plots for aspirin use in dotted lines.

Clinical trial data shows significant effectiveness of statins and different antihypertensive medications in preventing major cardiovascular events. Table 5 shows a summary of the findings. Most of the results are reported as relative risks – the risk of cardivoascular events for a person taking the medication relative to one not taking the medicaiton. For example the

relative risk of a cardiovascular disease event is 27 percent lower for a person taking a statin drug than for an equivalent person not taking a statin.

We use these relative risks to simulate the change in cardiovascular disease incidence that might be expected from the increase in medication use over time. We treat the relative risks as independent and evaluate the different medications in turn. The net effect is shown in the last column of table 5. As a result of increased use of cardiovascular medication over time, cardiovascular disease hospitalizations are predicted to have declined by 21% between 1992-94 and 2007-09. The largest predicted contributor to this trend is increased use of statin drugs, though greater use of ACE/ARBs is important as well. In addition to this 21% implied reduction, there is an additional 2% reduction in hospitalization predicted by the reduction in smoking over time.

While the overall change in incidence predicted by the medication use and reduced smoking is close to the 35% overall reduction shown in figure 10, the timing is not particularly consistent. **Figure 14** shows the simulated yearly incidence rates along with the actual change in heart disease hospitalizations. In the period between 1996 and 2001, the simulated ischemic heart disease incidence rates are much lower than the actual incidence rates. This could well be a result of greater diagnosis of heart attacks, as noted above. IHD hospitalization rates started declining in early 2000's, and the declines in this period are much greater than can be explained by medication use. Thus, we find it difficult to explain hospitalization trends with this model.

It is possible that other factors could be involved in explaining these trends. The key issue is why people are hospitalized for heart disease. In the earlier period, many hospitalizations were associated with elective PCIs in an inpatient setting. Increasingly, though, PCI can be performed on an outpatient basis (Olivier et al. 2007, Laarman et al.). Further, there

may be increased use of observational units in the emergency department instead of admission for chest pain (Graff et al., 1995).

TO COME: ANALYSIS OF HEART ATTACK HOSPITALIZATION TREND

These observations suggest that the model may be better at explaining mortality than hospitalization. To consider this, we start by showing trends in medication usage among patients with pre-existing ischemic heart disease (**figure 15**). As with the population as a whole, the use of cardiovascular disease medications increased greatly in this group. In addition to medications, figure 15 also shows a large increase in the use of primary PCI (stenting to restore blood flow after a heart attack).

Table 6 shows that the impact of medications and primary PCI on relative risk of death from heart disease. As before, the first columns show the relative risk of death for those using the particular therapy. The latter columns show the predicted change in mortality rate that results from the estimate relative risk and the change in use of the therapy. Based on the literature, we estimate that increased usage of cardiovascular medication would reduce IHD mortality by about 36%. Medication usage accounts for 29 percentage points of this decline (statins =8%, Beta-blockers=9%, ACE or ARB =6%, Aspirin =4% , Diuretics=2%), and primary PCI accounts for 5%. The remaining 2% is due to declining in smoking.

Figure 16 shows the annual prediction and the alignment of the annual prediction with the observed mortality trend. The fit in this case is much better. The actual and simulated mortality declines match very closely, with the exception that the actual decline is somewhat greater than the predicted decline in the later years of the sample. It is thus plausible that the improvement in health-related quality of life from cardiovascular disease is attributable to

medical advanced, most importantly greater use of medication but also increased surgical care for people having an acute event.

VI. Vision impairment in the elderly population

We now conduct an analysis of possible factors that may explain the change in disability-ajusted life expectancy associated with vision impairment.

The MCBS asks people if they have a current vision problem. The trend in this variable is shown in **figure 17.** Current vision problems have declined from about 40% of the elderly population to about 25%. This decline has been noted in other studies (Freedman and Martin 1998, Cutler 2001, Freedman et al. 2007, Cutler et al. 2014).

There are several reasons why people may have vision problems. The most prevalent in the elderly is cataracts. Cataracts is a medical condition in which the lens of the eye becomes progressively opaque, resulting in poor vision. Cataracts are a leading cause of vision impairment. Most cataracts happen as 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 procedure performed in the U.S to remove cataracts. **Figure 17** also shows the percentage of people who have had cataract surgery in the elderly Medicare population. Self-reported cataract surgery increased from 20% to 33%. The decline is 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 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. This is also increasing over time, though the rates are much lower.

We proceed with vision impairment in the same way as for cardiovascular disease, in particular by examining the litearture on the impact of cataract surgery on disability. **Table 8** contains a brief literature review of studies documenting improved vision problem after cataract surgery. The results indicate 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. The improvements in vision after cataract surgery is well documented in the literature.

Despite these improvements in vision, however, studies of QOL using measures such as EQ-5D, SF-12 and SF-36 showed no significant change in periods after cataract surgery.

Our conclusion about vision impairment is thus uncertain. On the one hand, greater use of cataract surgery seems like a plausible explanation for reductions in vision problems, and vision problems are associated with high rates of disaiblity. On the other hand, there is no clinical evidence that cataract surgery leads to reductions in disability. We do not know how to reconcile these two sets of results.

TO COME: LOOK AT TRENDS WITHIN INDIVIDUALS.

VII. Conclusion

Our results show clearly that over the 1991-2009 period disability-free life expectancy rose, and disabled life expectancy declined. The diseases that contributed to these gains are cardiovascular disease and improvements in vision problem. Some of these gains can also be due to other social and environmental factor.

Our research shows that statins and antihypertensive medications have a significant effect in reducing major cardiovascular events and mortality. The increased used with medications and

procedures like primary PCI predicts 83% of mortality declines. The improvements in vision problem can be attributed to more people having cataract surgery and better treatment of macular degeneration and glaucoma.

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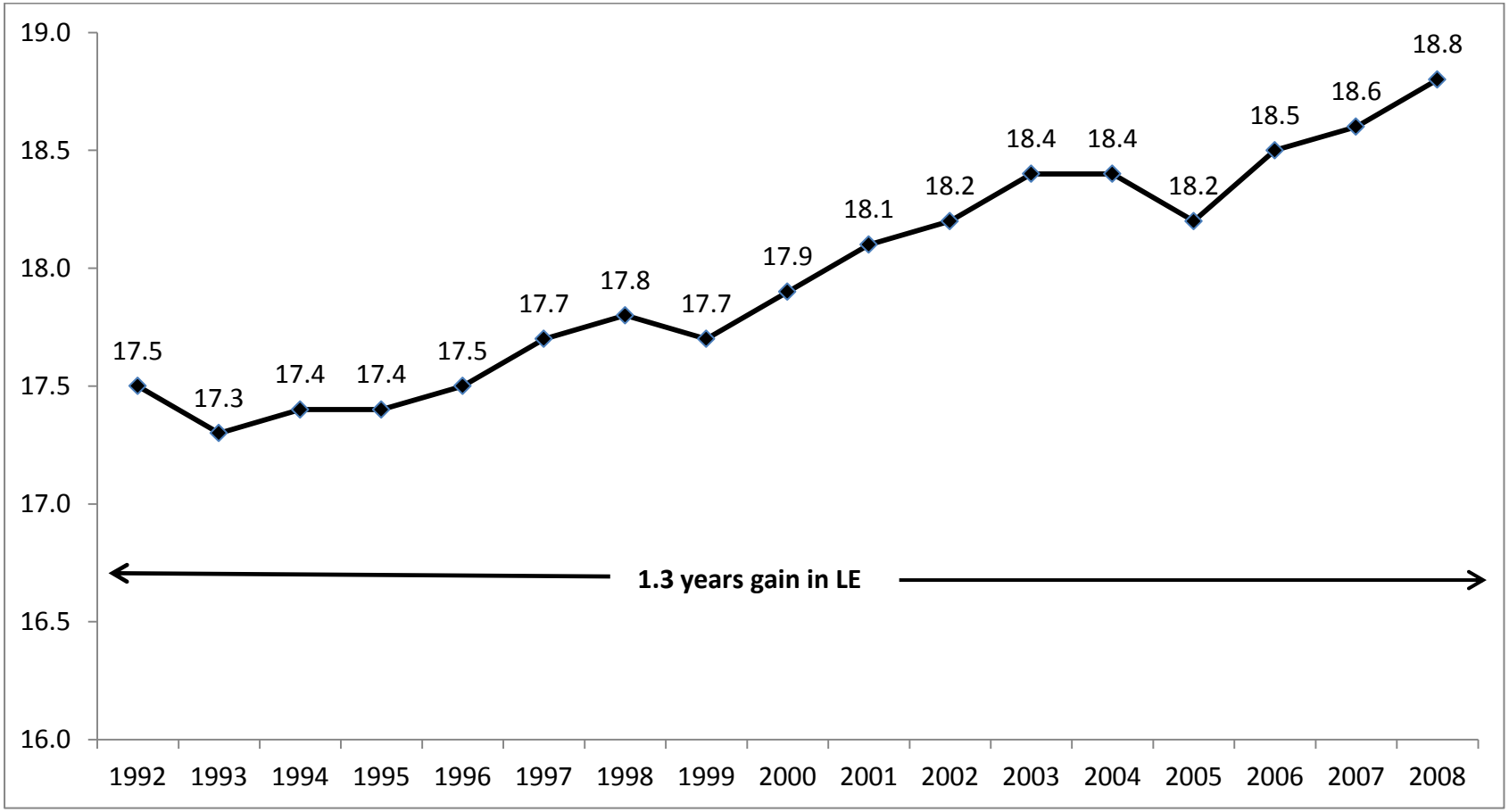
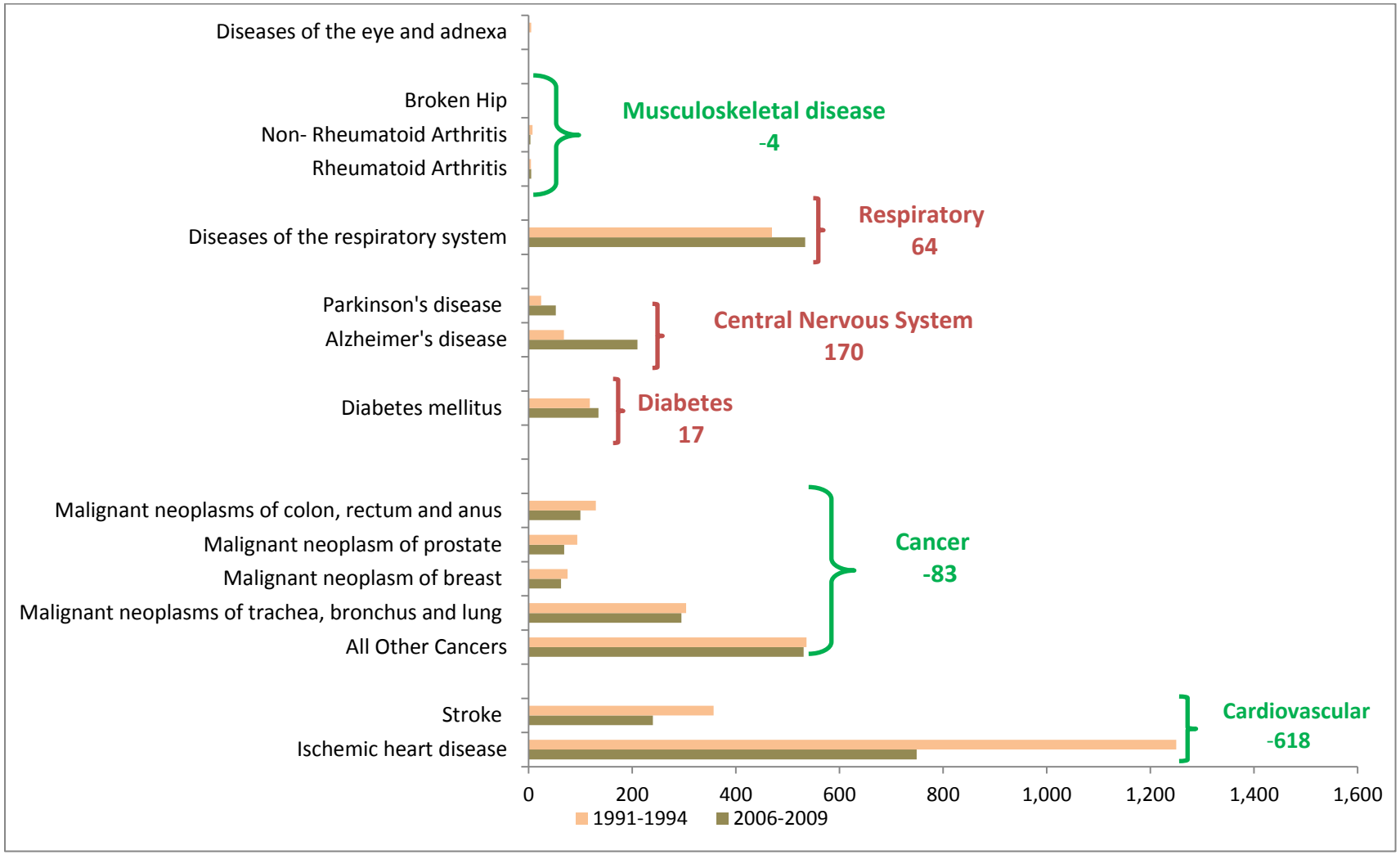
Figure 1: Life expectancy at age 65 (Total population)



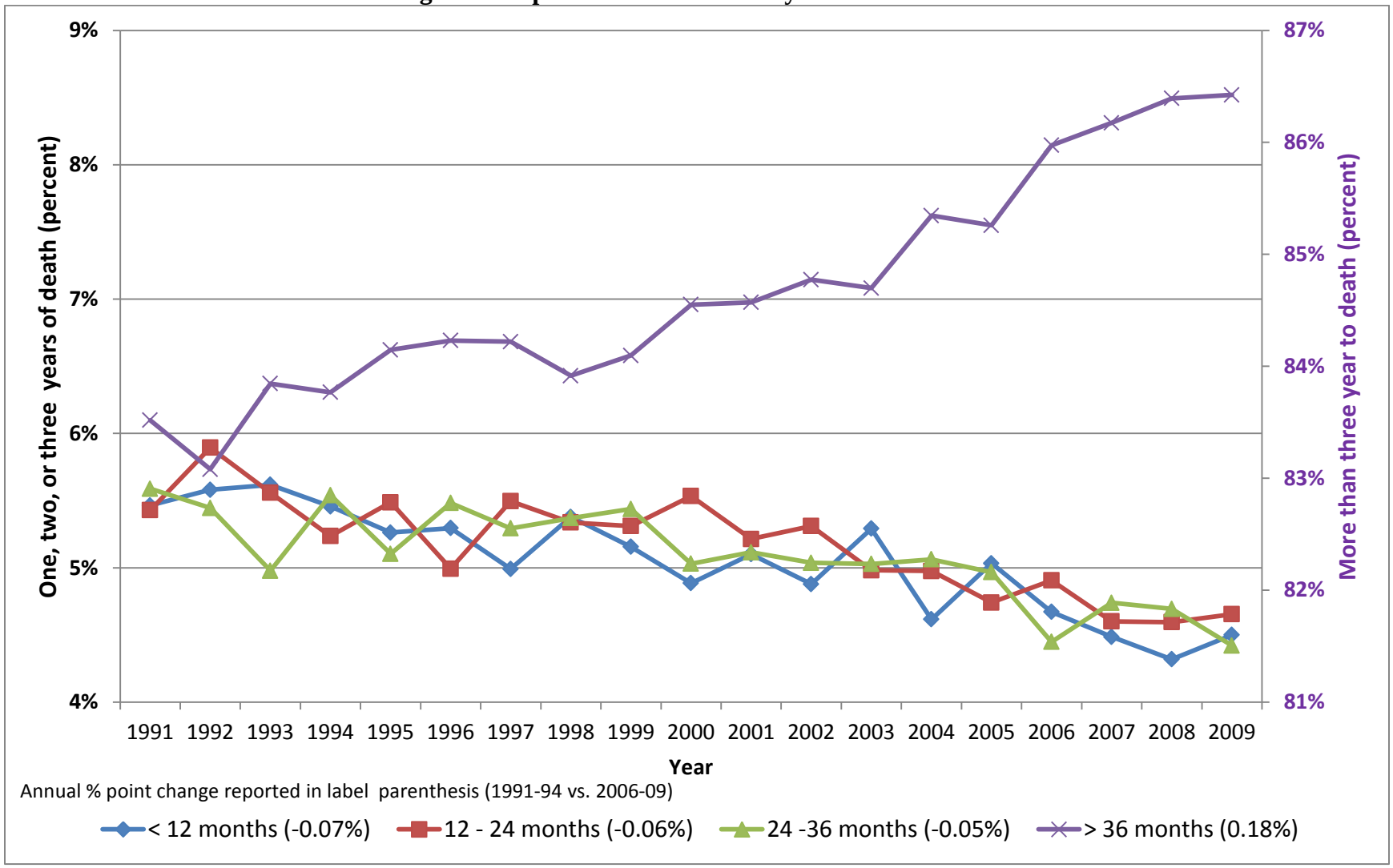
Note: Data are from the Vital Statistics of the United States from the Centers for Disease Control and Prevention/National Center for Health Statistics.

Figure 2: NCHS Causes of Death for 65+: Mortality rates per 100,000 (age-sex adjusted)



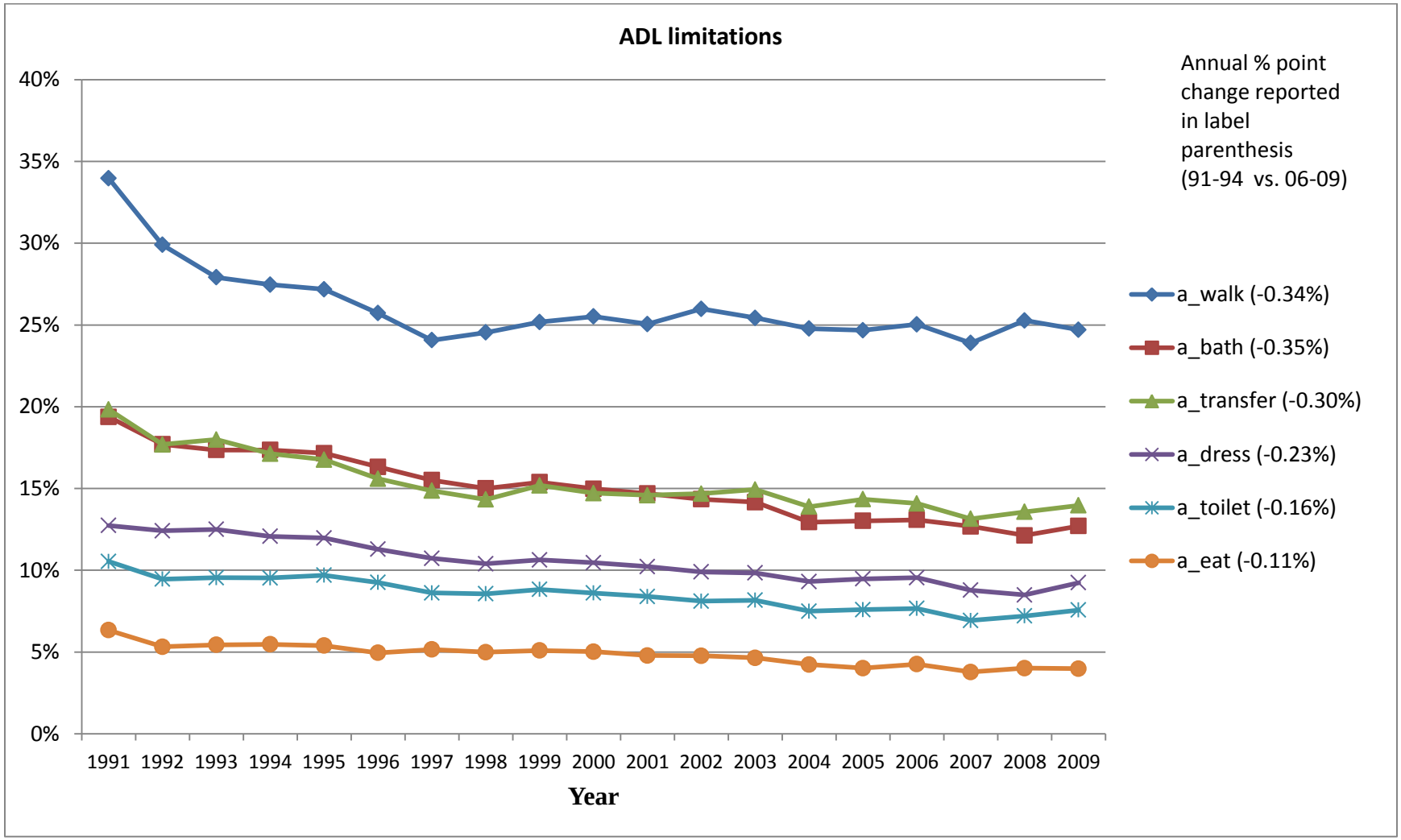
Note: Data are from the Centers for Disease Control and Prevention/National Center for Health Statistics on Causes of Death. The change in death rate is for two time periods 1991-1994 and 2006-2009.

Figure 3: Population Distribution by Time until Death



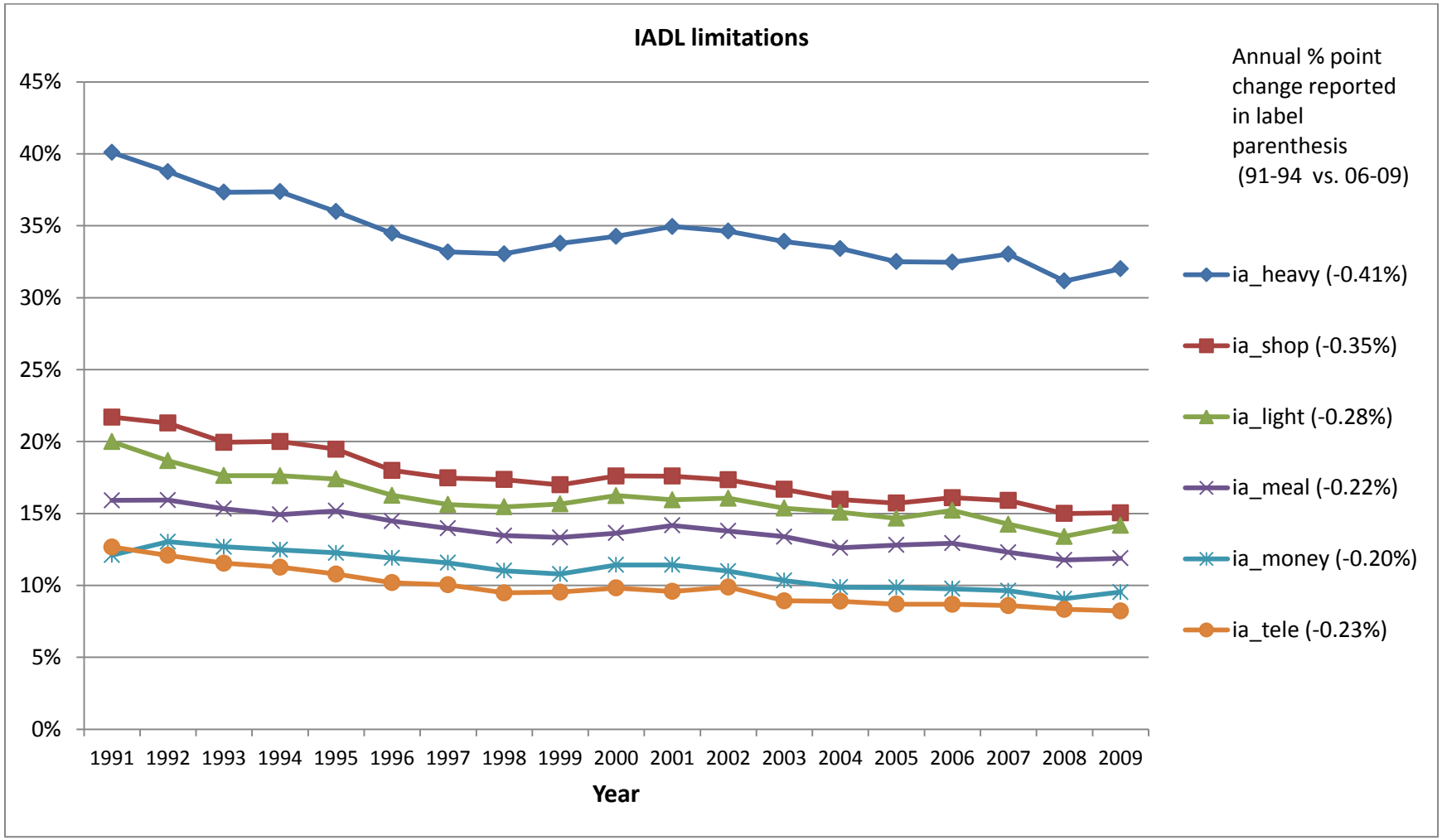
Note: Data are from the Medicare Current Beneficiary Survey and Medicare denominator files linked to MCBS, 1991-2009. Reported statistics is weighted to the population distribution in 2000 by age, sex, and race.

Figure 4: IADL and 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.

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



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.

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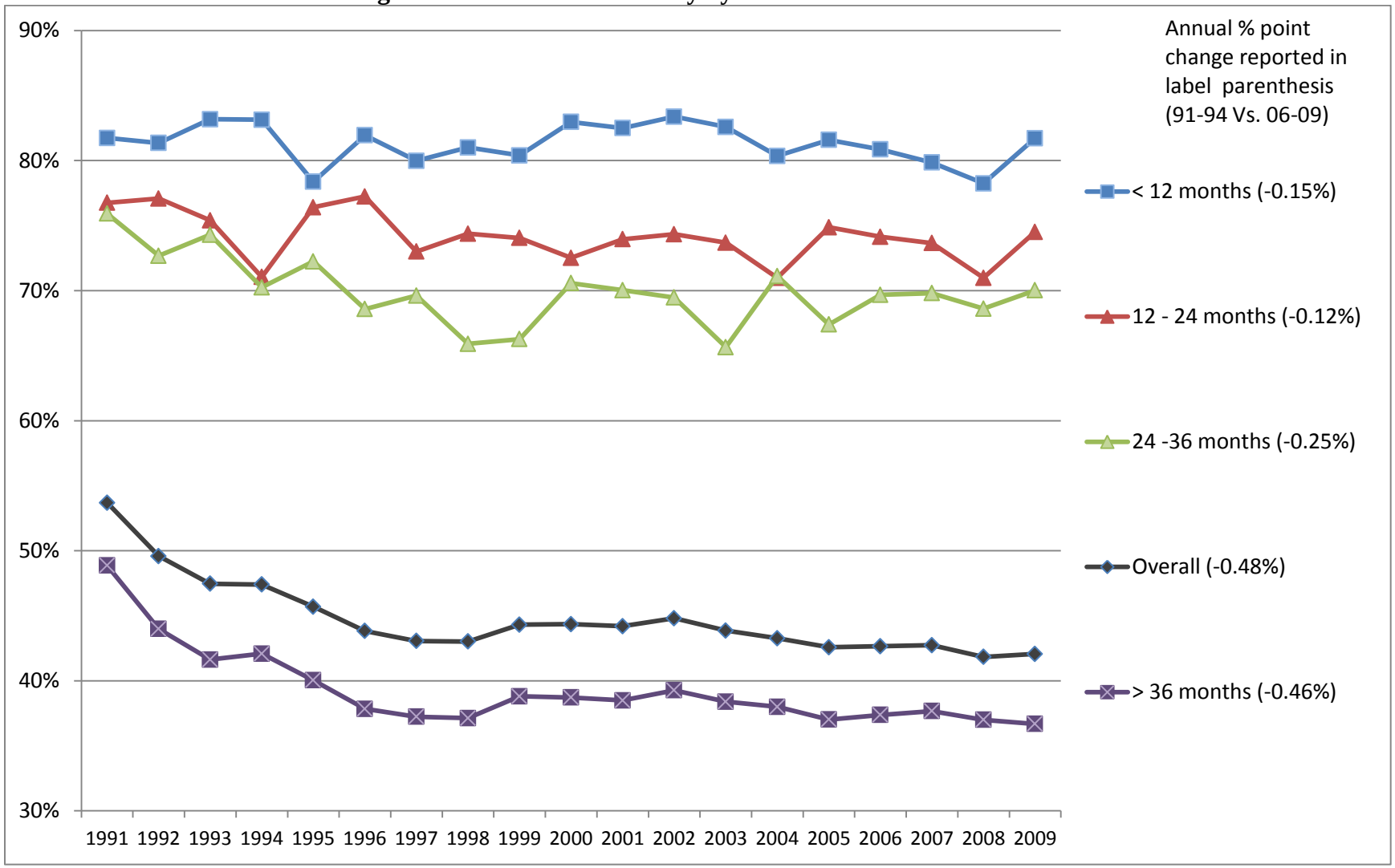
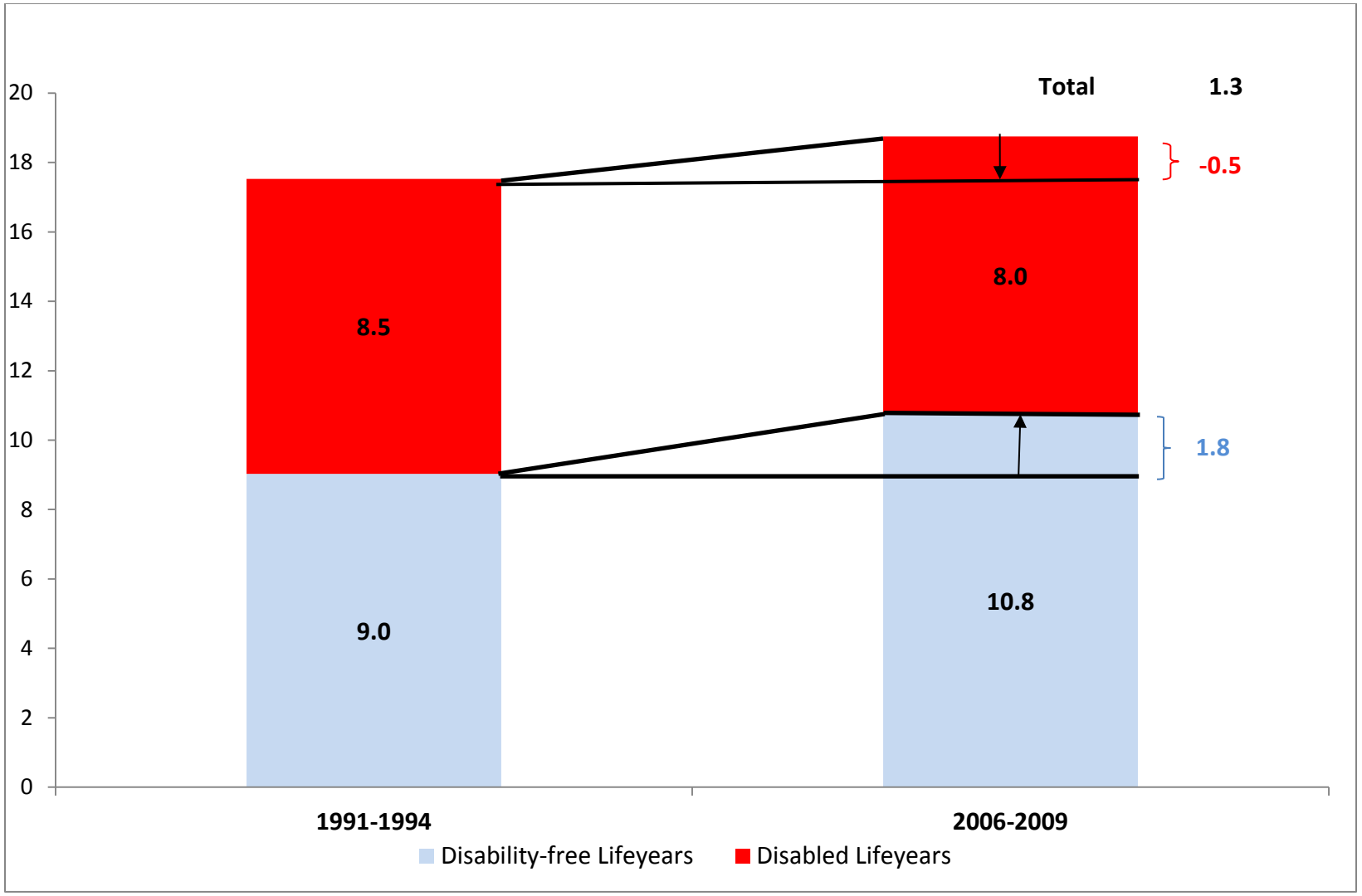
Figure 5: ADL/IADL Disability by Time until Death



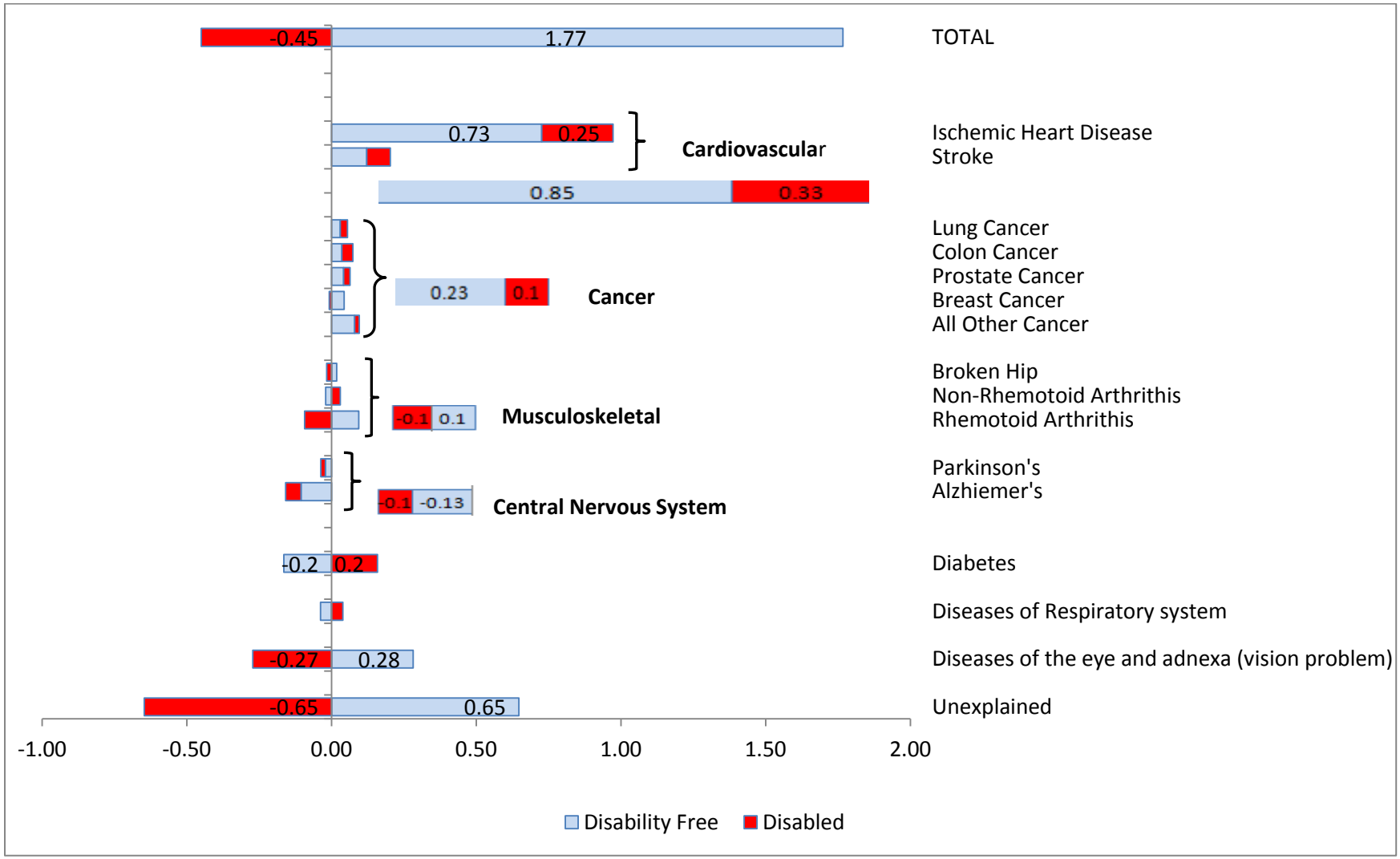
Note: Data are from the Medicare Current Beneficiary Survey and Medicare denominator files linked to MCBS, 1991-2009 and are weighted to the population distribution in 2000 by age, sex, and race.

Figure 6: Trend in Disabled and Disability Free Life Expectancy at 65

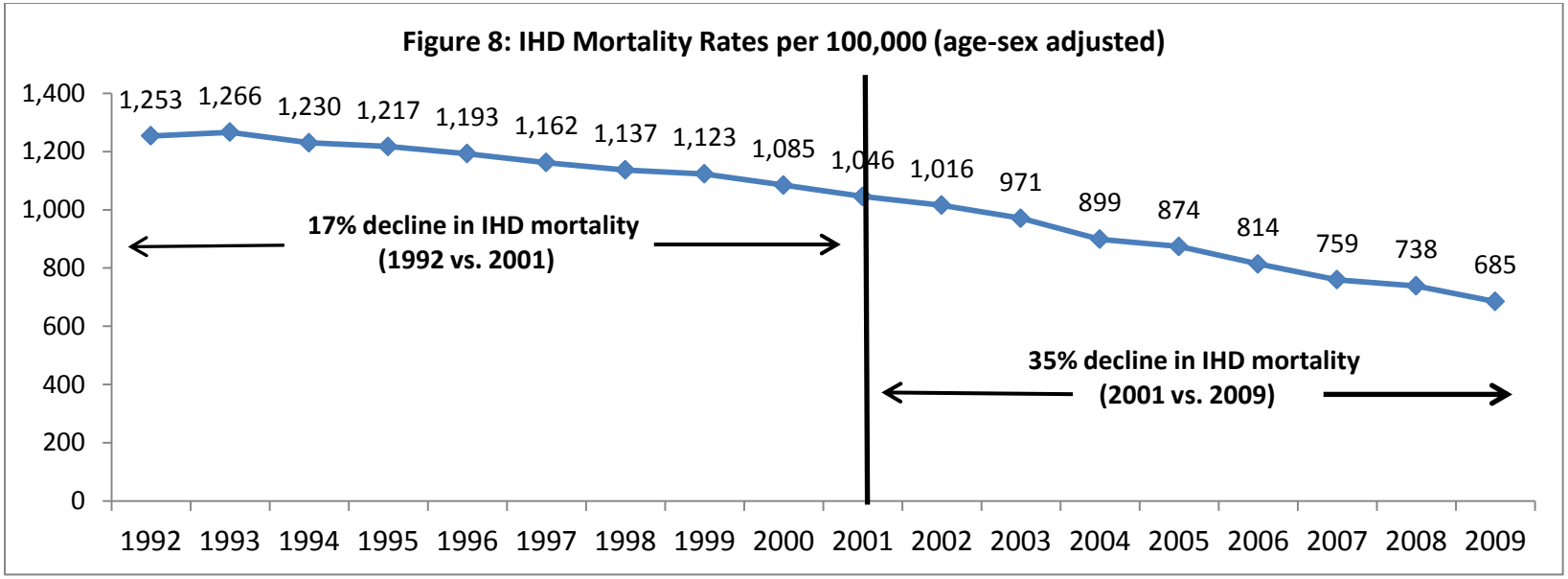


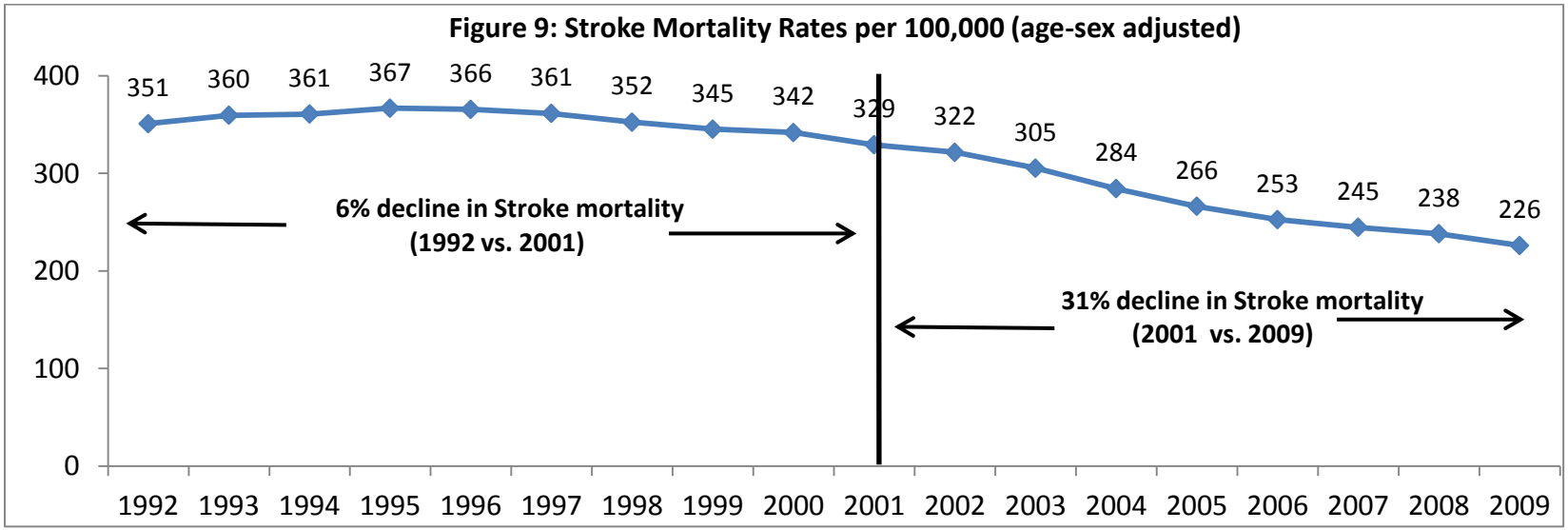
Note: The figure combines life expectancy data from the NCHS with imputed disability rates by age and time until death from MCBS data linked to Medicare.

Figure 7: Change in Disabled and Disability Free Life Expectancy at age 65 by disease (1991-94 Vs 2006-2009)

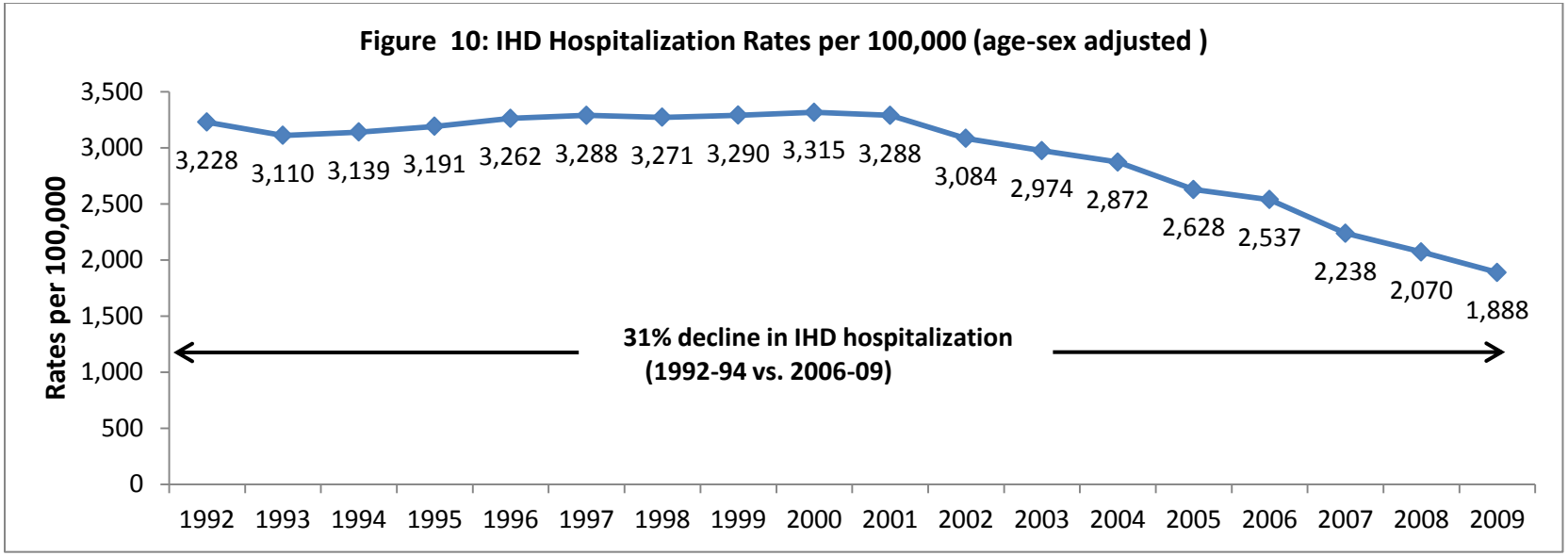


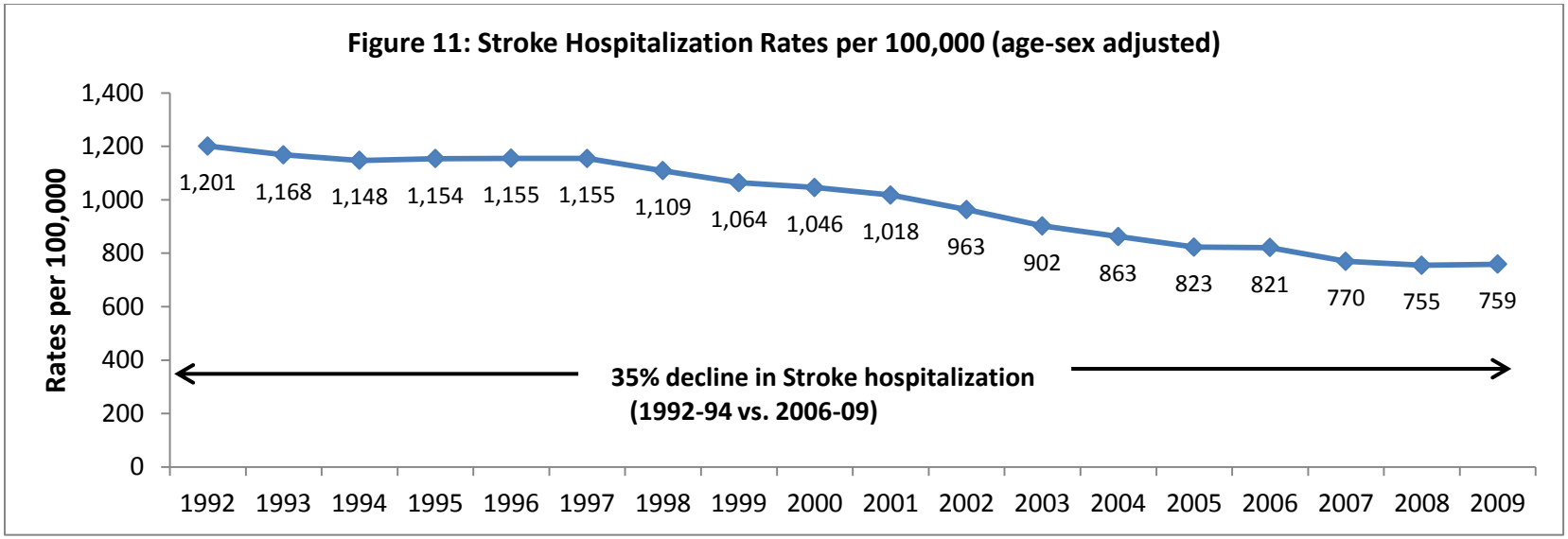
Note: The figure combines life expectancy data from the NCHS combined with Causes of Death data and imputed disability rates by age and time until death from MCBS data linked to Medicare.



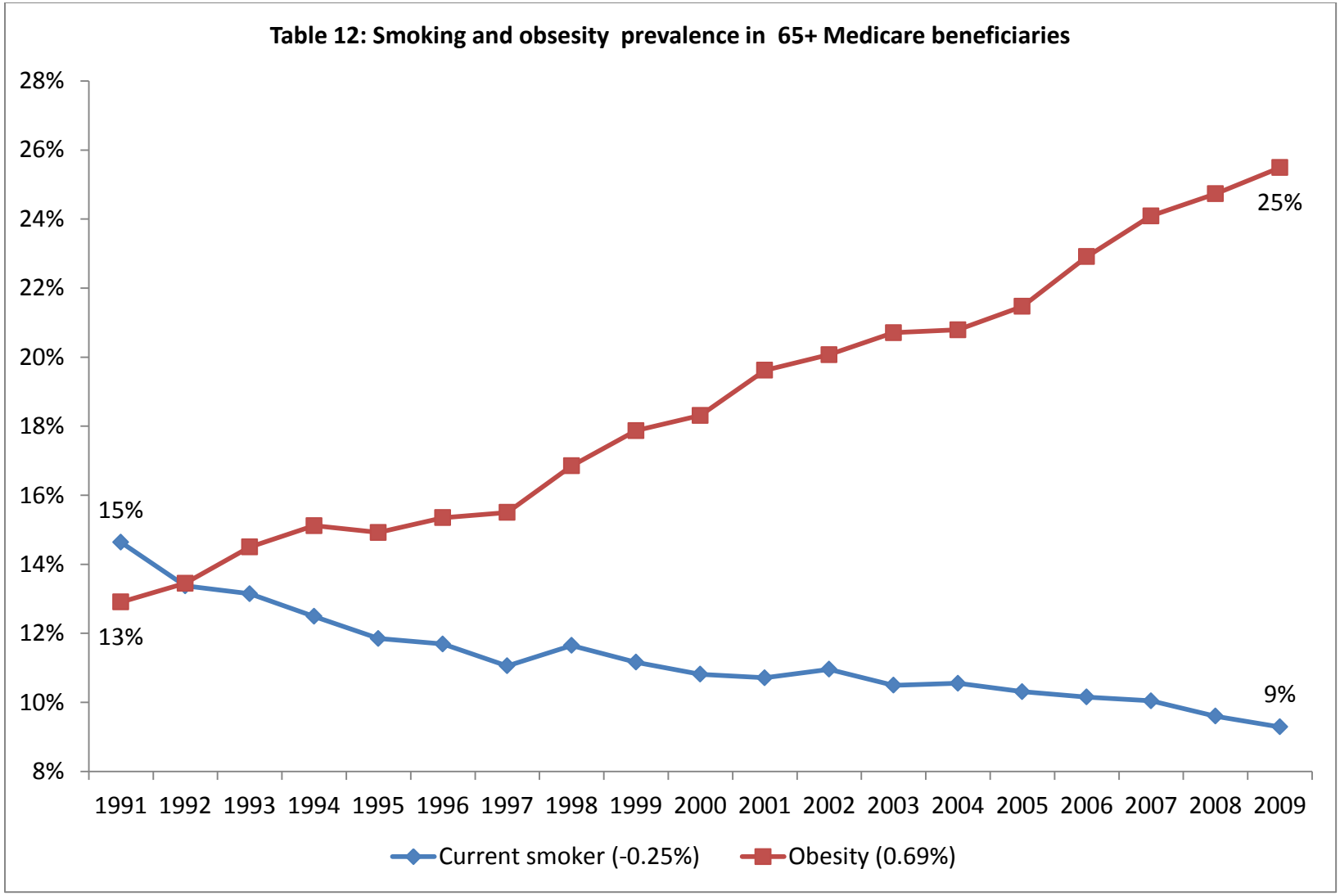


Note: Data are from the Centers for Disease Control and Prevention/National Center for Health Statistics on Causes of Death and micro data on mortality available at the National Bureau of Economic Research

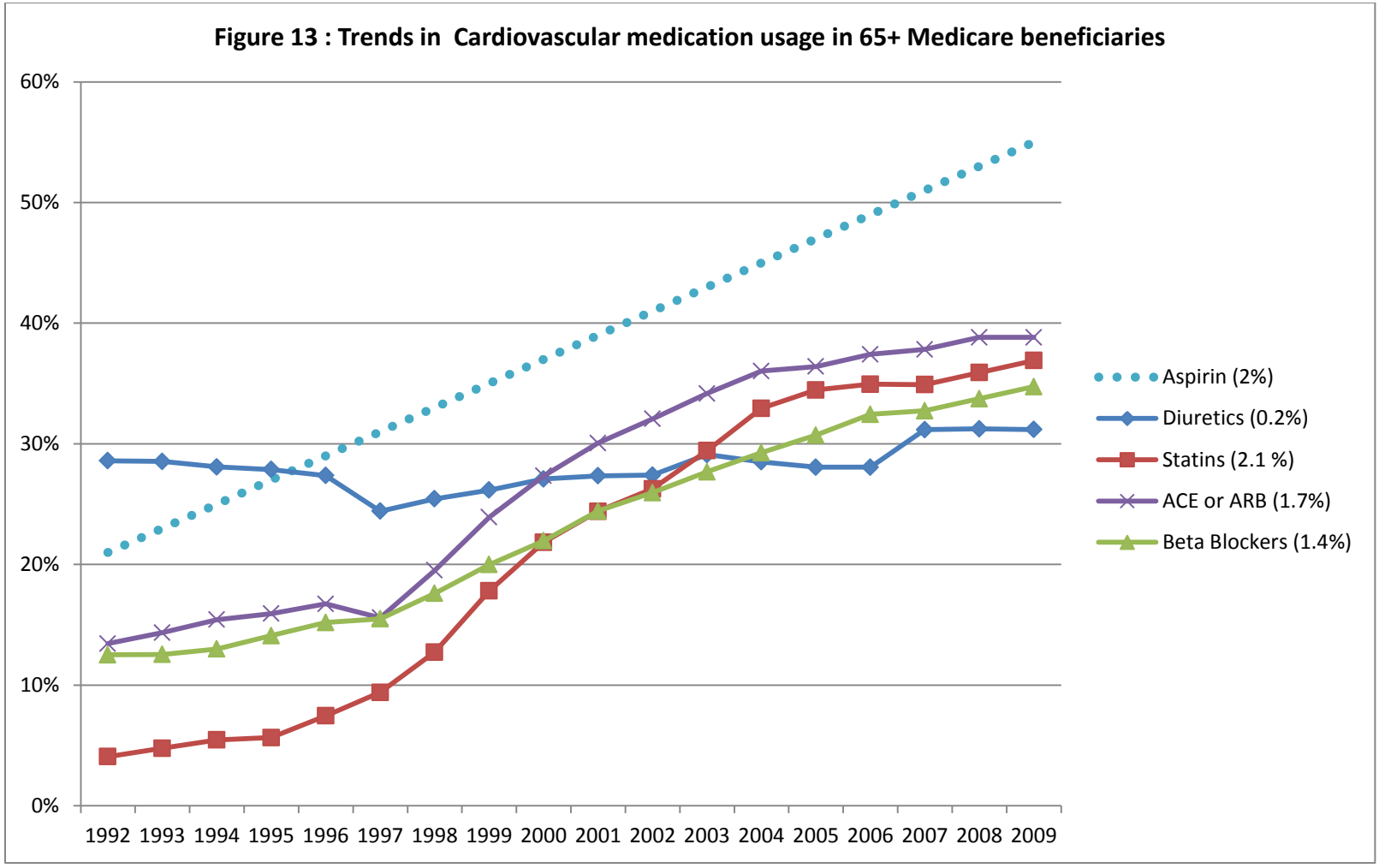




Note: We use 5% Medicare Sample for beneficiaries 65 years and older and Fee-for-Service enrolled. The rates per 100,000 are age-sex adjusted .Inpatient hospitalization of Ischemic Heart Disease or Stroke as principal discharge diagnosis



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



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 2000 population by age, sex, and race. The aspirin usage from the 1992-94 period is from NHANES III and the later period is from MEPS, 2007. We did a linear interpolation for the intermediate years.

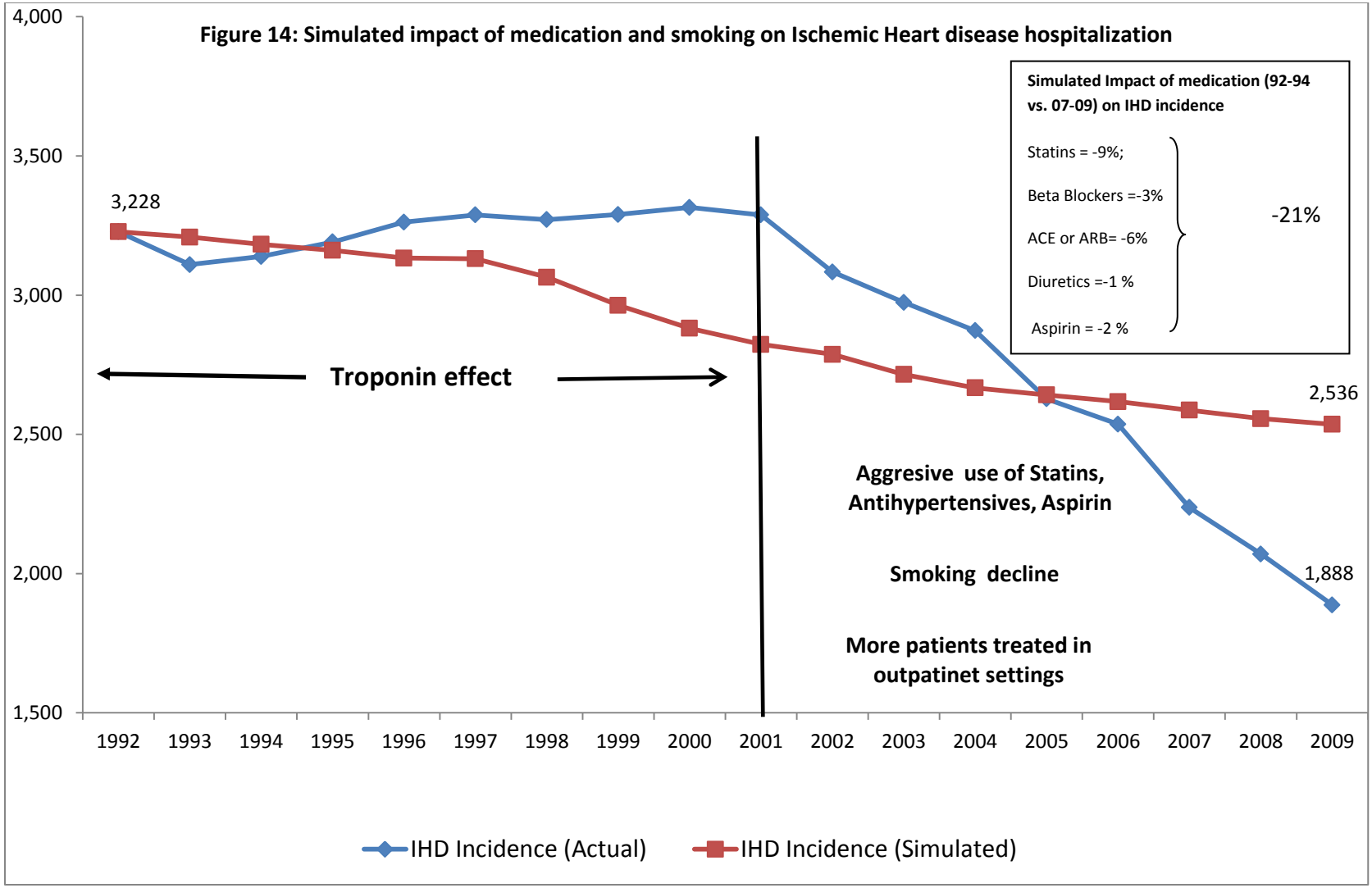
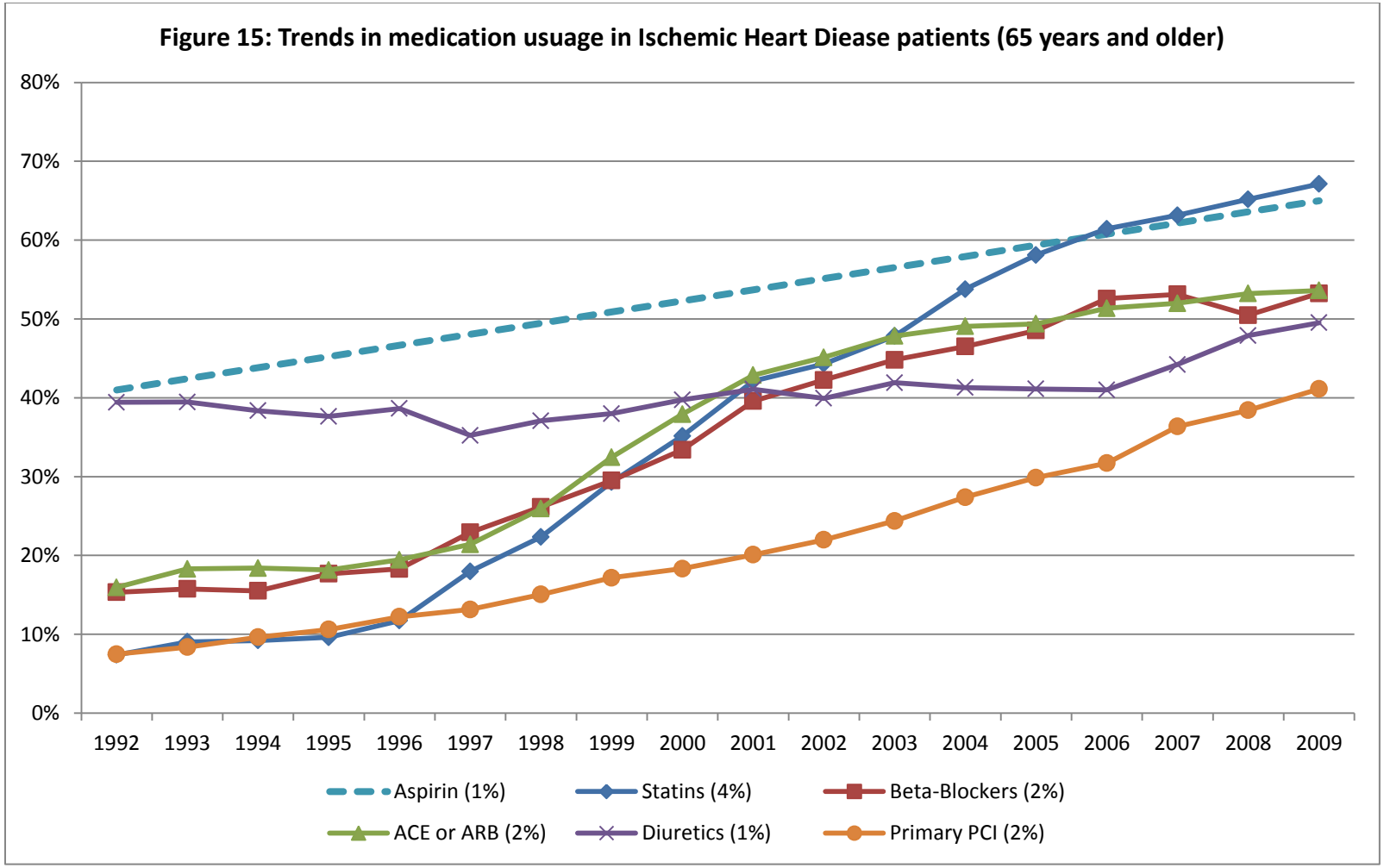


Figure 14: Simulated impact of medication and smoking on Ischemic Heart disease hospitalization
Simulated Impact of medication (92-94 vs. 07-09) on IHD incidence
Statins = -9%;
Beta Blockers =-3%
ACE or ARB= -6%
Diuretics =-1 %
Aspirin = -2 %
-21%
4,000
3,500
3,000
2,500
2,000
1,500
3,228
2,536
1,888
Troponin effect
Aggresive use of Statins, Antihypertensives, Aspirin
Smoking decline
More patients treated in outpatinet settings
1992
1993
1994
1995
1996
1997
1998
1999
2000
2001
2002
2003
2004
2005
2006
2007
2008
2009
IHD Incidence (Actual)
IHD Incidence (Simulated)

Note :



Note: 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% Medicare sample for people hospitalized for Ischemic Heart Disease (410.X – 414.X).

Figure 16: Simulated impact of cardiovascular medication on IHD mortality

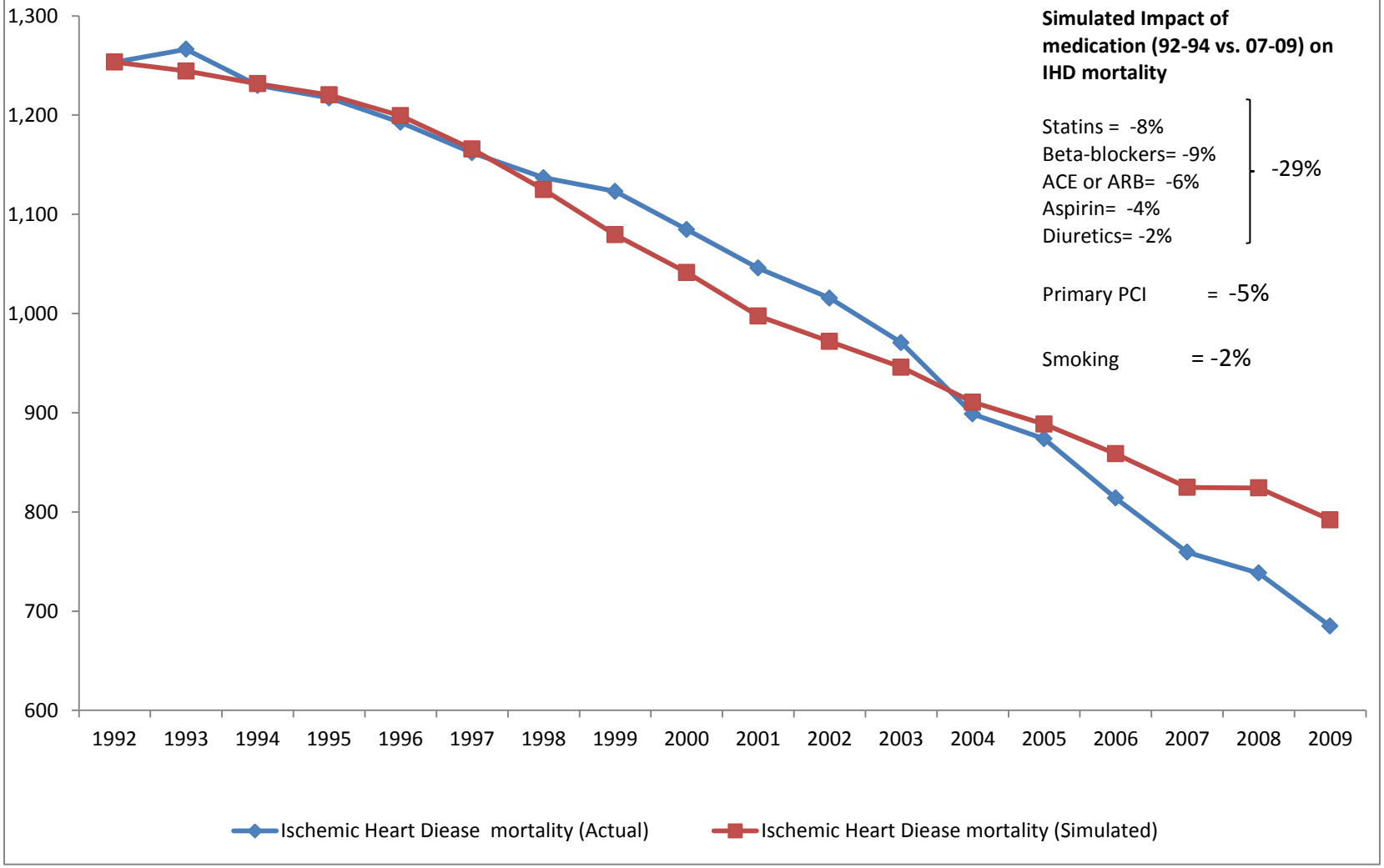


Figure 17: Cataract Surgery and vision problem

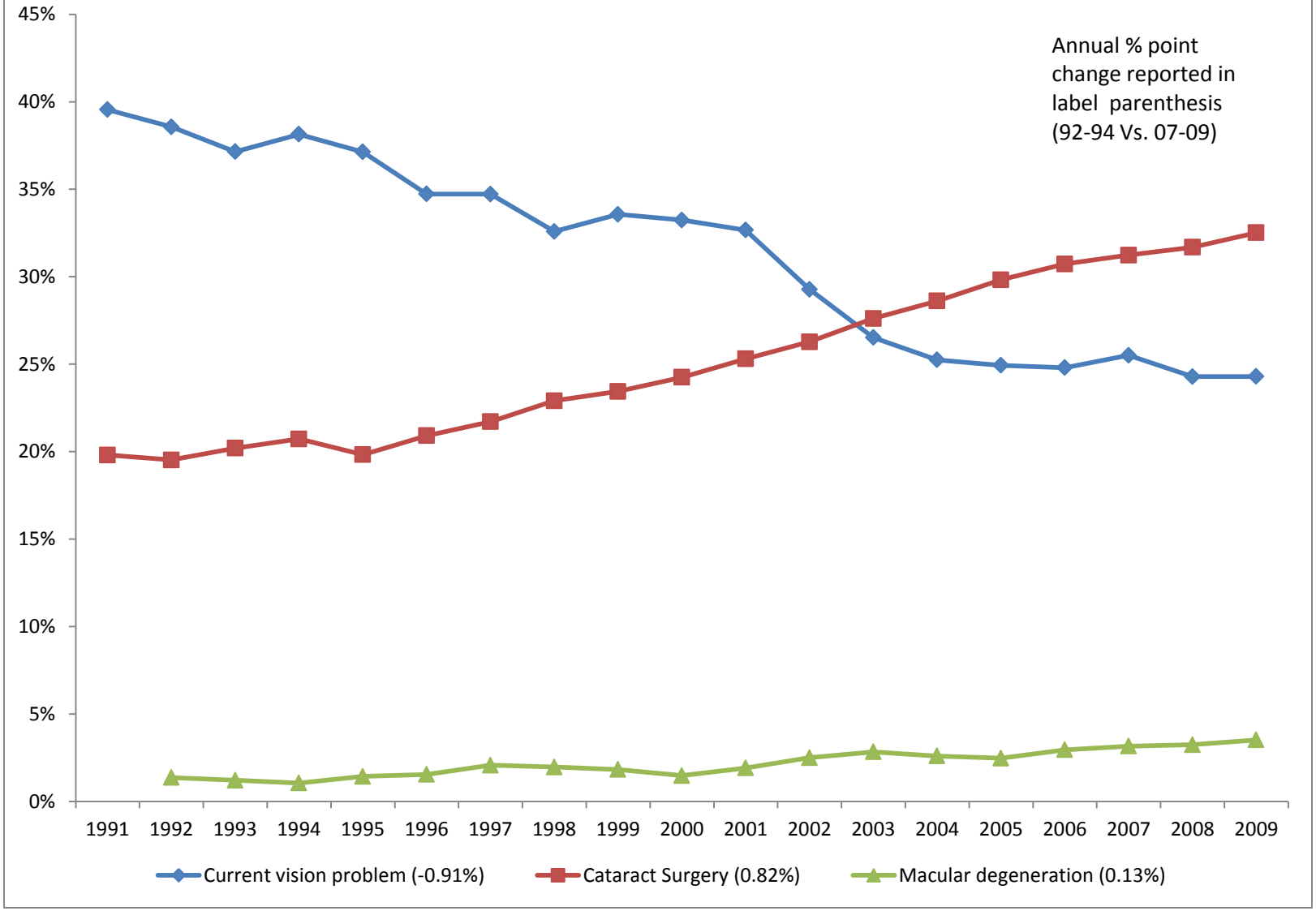


Table 1: Health Status Questions in the MCBS, 1991-2009

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 use sample weights adjusted to a constant year 2000 population by age, gender, and race.

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 use sample weights and use sample weights adjusted to a constant year 2000 population by age, gender, and race.

Table 3: Regressions Explaining Disability

					OAXACA DECOMPOSITION		
	Prevalence 91-94	Coefficients 91-94	Prevalence 06-09	Coefficients 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	5.8%	0.28	0.1%	0.2%	0.4%
PARKINSONS	1.8%	0.18	1.6%	0.24	0.1%	0.0%	0.1%
Cardiovascular disease					**-1.7%**	**-0.8%**	**-2.5%**
ISCHEMIC HEART DISEASE	29.5%	0.11	22.9%	0.06	-1.5%	-0.7%	-2.2%
STROKE	11.3%	0.16	10.9%	0.14	-0.2%	-0.1%	-0.3%
Pulmonary disease							
PULMONARY	13.3%	0.14	15.9%	0.13	**-0.02%**	**0.4%**	**0.3%**
Diabetes	16.2%	0.11	23.8%	0.12	**0.1%**	**0.9%**	**0.9%**
Musculoskeletal disease					**-0.3%**	**-0.2%**	**-0.5%**
RHEUMATOID ARTHRITIS	12.3%	0.22	10.7%	0.20	-0.3%	-0.4%	-0.6%
NONRHEUMATOID ARTH	43.5%	0.13	46.2%	0.12	-0.2%	0.4%	0.2%
BROKEN HIP	5.2%	0.13	3.5%	0.16	0.2%	-0.2%	-0.1%
Cancer					**-0.3%**	**-0.03%**	**-0.3%**
LUNG CANCER	0.7%	0.09	1.1%	0.08	0.0%	0.0%	0.0%
BREAST CANCER	4.2%	0.04	4.8%	0.00	-0.2%	0.0%	-0.1%
PROSTATE CANCER	2.2%	0.02	4.1%	-0.01	-0.1%	0.0%	0.0%
COLORECTAL CANCER	2.9%	0.03	2.3%	0.03	0.0%	0.0%	0.0%
OTHER CANCER	8.4%	0.05	6.5%	0.05	-0.1%	-0.1%	-0.2%
Vision Problem							
VISION PROBLEM	38.4%	0.13	24.7%	0.13	**0.1%**	**-1.7%**	**-1.7%**

Time to death					**-4.5%**	**-0.4%**	**-4.9%**
12 - 24 months	5.5%	-0.04	4.8%	-0.04	0.0%	0.0%	0.0%
24 -36 months	5.4%	-0.04	4.6%	-0.07	-0.1%	0.0%	-0.1%
> 36 months	83.6%	-0.19	86.2%	-0.24	**-4.4%**	**-0.5%**	**-4.9%**
Other demographics					**0.9%**	**0.0%**	**0.9%**
Male 70 to 74 years	11.9%	0.01	11.9%	0.00	-0.1%	0.0%	-0.1%
Male 75 to 79 years	9.5%	0.06	9.5%	0.04	-0.2%	0.0%	-0.2%
Male 80 to 84 years	5.6%	0.14	5.6%	0.13	-0.1%	0.0%	-0.1%
Male 85 years and older	3.8%	0.28	3.8%	0.22	-0.2%	0.0%	-0.2%
Female 65 to 69	12.7%	0.10	12.7%	0.08	-0.3%	0.0%	-0.3%
Female 70 to 74	14.5%	0.12	14.5%	0.09	-0.4%	0.0%	-0.4%
Female 75 to 79	13.1%	0.19	13.1%	0.15	-0.5%	0.0%	-0.5%
Female 80 to 84	9.2%	0.28	9.2%	0.22	-0.5%	0.0%	-0.5%
Female 85 years and older	9.1%	0.37	9.1%	0.35	-0.2%	0.0%	-0.2%
_cons	100.0%	0.27	100.0%	0.30	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.

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

	1988-1994	1999-2000	2001-2004	2005-2008	2009-2012	1988-1994	1999-2000	2001-2004	2005-2008	2009-2012
	Men	Men	Men	Men	Men	Women	Women	Women	Women	Women
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

Table 5: Effectiveness and Use Of Medicine and Smoking on Heart Disease Events in 65+ Medicare population

Therapy	Relative Risk of Cardiovascular Event	Source	1992 -1994 Use (%)	2007 - 2009 Use (%)	Change in Usage (%)	Impact on Cardiovascular Event
Statins	0.73 (0 .67 - 0.80)	Taylor et al. , 2011	5%	36%	31%	-9%
Beta-blockers	0.88 (0.79–0.97)	Bradley et al. , 2006	13%	34%	21%	-3%
ACE or ARB	0.78 (0.70–0.86)	Yusuf et al., 2000	14%	38%	24%	-6%
Diuretics	0.79 (0.69 -0.92)	Yusuf et al.	28%	31%	3%	-1%
Aspirin	0.94 (0.77-1.15)	Yasuo Ikeda et al.,2014	21%	55%	34%	-2%
Medicines						**-21%**
Behavioral Factors						
Smoking	1.5	Gary D. Friedman, 1997	13%	10%	-3%	**-2%**
Total Impact						**-23%**

Table 6: Effectiveness And Use Of Medicine on Heart Disease Cohort

Therapy	Relative Risk of mortality	Source	1992 -1994 Use (%)	2007 - 2009 Use (%)	Change in Usage (%)	Impact on Mortality
Statins	0.85 (0.73–0.99)	Wilt et al., 2004	9%	65%	57%	-8%
Beta-blockers	0.77 (0.69–0.85)	Freemantle et al., 1999	16%	52%	37%	-9%
ACE or ARB	0.83 (0.71–0.97)	Domanski et al., 1999	18%	53%	35%	-6%
Aspirin	0.82 (0.70–0.99)	Weisman et al., 2002	42%	64%	21%	-4%
Diuretics	0.74 (0.59 -0.93)	Domanski M, 2003	39%	47%	8%	-2%
Medicine						**-29%**
Revascularization						
Primary PCI	0.73 (0.60-0.89)	Keeley et al., 2003	9%	39%	30%	**-5%**
Behavioral Factors						
Smoking	1.85	Gary D. Friedman, 1997	13%	10%	-3%	-2%
Total						**-36%**

Table 7: Effectiveness and Use of Cataract Surgery on vision problem and disability

Findings	Source
Cataract Surgery in both eyes reported greater improvement in subjective visual function than did those who underwent surgery in one eye	Javitt et al. , 1993
The percentage of patients whose vision impairment improved at 4 months after cataract surgery varied by the outcome measure used: Snellen visual acuity (96%); VF-14 score (89%); satisfaction with vision (85%); self-reported trouble with vision (80%); and Sickness Impact Profile score (67%).	Steinberg et al. , 1994
At 12 months after surgery, 95% of patients had improved Snellen visual acuity, 80% had improved ADVS scores, but only 36% had improved SF-36 physical functioning. Average scores on seven of eight SF-36 subscales worsened at 12 months.	Mangione et al. , 1994
Patients who went for cataract surgery had half the rate of crash involvement during the follow-up period compared with cataract patients who did not undergo surgery.	Owsley et al. , 2002
For nursing home residents, visual acuity for near and distance and contrast sensitivity improved following cataract surgery ($p<0.001$). After controlling for age-sex, the cataract surgery group showed significant score improvement in the general vision ($p = 0.005$), reading ($p = 0.001$), psychological distress ($p = 0.015$), and social interaction ($p = 0.033$) subscales of the Nursing Home Vision-targeted Health-Related Quality of Life Questionnaire and the VF-14 ($p = 0.004$). But, the study didn't find any differences in the SF-36, Geriatric Depression Scale or the Cataract Symptom Score.	Owsley et al. , 2007
The NEI-VFQ25 total score and all 11 subscales showed significant improvements during the first interval (baseline and one month). During the second interval (1-6 months post-surgery), significant improvements were observed for the total score and 5 of 11 NEI-VFQ25 subscales. There were significant increases in HRQOL during the first interval on	Gross et al, 2013

some preference-based generic HRQOL measures though changes during the second interval were mostly non-significant. None of **the SF-36v2or SF6D** scales changed significantly between any of the assessment periods.	
Contrast sensitivity and stereopsis, but not visual acuity, were significant factors affecting improvement in vision-related quality of life or depressive symptoms after first eye cataract surgery.	Fraser et al. , 2013