Essays on the Value of Health and Longevity

by

Nirman Saha

A Dissertation Presented in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Approved April 2021 by the
Graduate Supervisory Committee:

Nicolai V. Kuminoff, Chair
Daniel Silverman
Jonathan Ketcham
Gregory Veramendi

ARIZONA STATE UNIVERSITY

May 2021

ABSTRACT

My dissertation investigates how individuals make tradeoffs between consumption and health risks, and the implications of their choices for the efficiency and equity of public policies. I study how these decisions can be used to infer their willingness to pay to reduce health risks, as well as the implications for dynamic complementarities between the quantity and the quality of life, and how decisions on health behavior affect educational attainment and workplace performance. Chapter 1 provides a brief preview of how I formalize these ideas, test them using micro data, and consider their policy implications in three interconnected essays.

In Chapter 2 I provide the first revealed preference evidence on the willingness to pay to reduce mortality risks by US senior citizens. I derive this evidence from the rates at which they consume medical services and the effects of their choices on survival probabilities. Instrumental variable estimates provide robust evidence that their Value of a Statistical Life (VSL) is well below $1 million and declines with age. Conditional on age, the VSL increases in health, income, education and is higher for women and for people who never smoked.

Chapter 3 develops a unified framework for valuing changes in health and longevity that explicitly allows for the complementarities between quantity and quality of life. I develop a dynamic life-cycle model of health investment, stochastic health and mortality, and use the model to characterize the VSL and the value of disease prevention for seniors. My results reinforce the qualitative variation in VSL with respect to age, health and income described in Chapter 2. I also document evidence of dynamic complementarity that implies there is increasing returns to health improvements. To demonstrate the policy implications of this complementarity, I use my calibrated model to calculate the benefits of mortality reductions in the 2000's that the US Environmental Protection Agency attributed to the Clean Air Act. I find that these

mortality reductions generated benefits, in part, by increasing the value of further health risk reductions due to increased life expectancy. The value of this improvement was equivalent to 6% of the total benefit of the Clean Air Act in 2010.

In Chapter 4 I investigate the causal effect of depression on illicit drug use among young adults. Using the 9/11 terrorist attack as an instrument for depression, I show that depression triggers illicit drug use among young adults (age 18-28) with heterogeneous impacts on different drugs. This finding suggests that treating depression can help to reduce illicit drug use. Dynamic complementarity in health improvements therefore, imply that there are likely to be positive externalities from the Affordable Care Act and other policies that increase insurance coverage for mental health.

DEDICATION

To my parents, my siblings and my wife, Joyeta

ACKNOWLEDGEMENTS

I would like to express my utmost gratitude to my advisor, Nicolai Kuminoff for his guidance, support and motivation throughout this dissertation process. His insightful feedback, continuous encouragement and persistent belief in my abilities have been instrumental in instilling a sense of confidence in me to excel in research and sharpen my thinking. I would also like to acknowledge the invaluable advice and guidance provided by Gregory Veramendi, Jonathan Ketcham and Daniel Silverman. This work has also benefited from the helpful suggestions and feedback from seminar participants at Arizona State University, Camp Resources XXV, ERG Winter Conference 2018 and WEAI Annual Conference 2019. Finally, I would also like to thank my colleagues and friends, Santiago Garcia-Couto, Jacob French, Tomás Sanguinetti, Spencer Perry, Nazim Tamkoç and Sophie Mathes for stimulating discussions and careful insights.

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Chapter 1

INTRODUCTION

My research investigates individual decision making under uncertainty about future survival and health. I observe people making trade-offs between their current consumption and future health risks and I assess these decisions for the distributional welfare effects of public policies. I focus primarily on how the dynamics of health investment decisions and the evolution of health can be used to infer people's willingness to pay for reductions in mortality risks, understand how this willingness to pay is affected by the dynamic complementarities between the quantity and quality of life, and consider how mental health shocks affects educational attainment and labor supply.

In Chapter 2, I develop and implement a novel approach to estimating the Value of a Statistical Life (VSL) for United States (US) seniors, specifically people over the age of 65. These seniors are the wealthiest and the fastest growing population by age-group in the US, in addition to being the largest beneficiaries of public policies targeting health improvements. However, there is a striking discrepancy in the way that VSL estimates are typically used to estimate mortality benefits for seniors in policy evaluation. Specifically, the VSL measures that are typically applied to seniors are estimated from hedonic wage regressions using data from younger and healthier workers. To resolve this inconsistency, I formulate a life cycle model of health and medical spending that allows me to estimate the VSL for seniors based on the rates at which they consume medical service and the effects of their decisions on medical expenditures on survival probabilities.

The VSL is estimated using individuals' responses to the Medicare Current Ben-

eficiary Survey linked to their Medicare records. To account for the endogeneity in econometric estimation of the survival function that may arise due to latent health, I leverage the supply side variation in medical expenditures by adopting the approach of Finkelstein *et al.* (2016). Instrumental variable estimates provide mean estimates for VSL below $1 million which is well below conventional estimates for prime-aged workers from hedonic wage models. The estimates are robust to controlling for spatial variation in health care quality, climate and pollution, state fixed effects, alternative parametric forms for survival function, alternative measures of medical expenditures and considerations of who makes health insurance decisions for the senior beneficiary and knowledge of the US healthcare system. Heterogeneity analysis reveals that VSL declines with age and increases with health, income and education and is higher for females and never-smokers. One of the conclusions of this study is that because the VSL increases with health, health augmenting policies might increase VSL due to dynamic complementarity between the quantity and quality of life. This may have important implications for the evaluation of public policies.

In Chapter 3 I formulate a unified framework for valuing changes in health and longevity that accounts for static and dynamic complementarities between the quantity and quality of life. Specifically, I develop a dynamic life-cycle model of health investment, stochastic health and mortality and use this to simultaneously characterize the VSL and the value of disease prevention for US seniors. The model embeds a Grossman style health production function, the parameters of which are identified using a dynamic latent factor model to account for measurement errors in any given proxy measure of health. By integrating health and survival, the model allows for static complementarity in risks in the sense that health shocks may simultaneously alter mortality and morbidity risks. The model also allows for dynamic complementarity in the willingness to pay (WTP) for health improvements. That is, a change

in current mortality risks may also change the WTP for future health improvements and vice versa.

After developing the model, I estimate the health production function using the same survey data linked to Medicare records that I use in Chapter 2. Then I calibrate the preference parameters in the life-cycle model using ancillary information on seniors' asset profiles from the Health and Retirement Study (HRS), in addition to moments on medical expenditure and health derived from the Medicare data. To calibrate VSL, I scale the shocks to survival such that the VSL at age 67 matches the estimates of VSL for workers aged 55-62 in Viscusi and Aldy (2007). Variation of the VSL with health, income and age qualitatively matches the results in Chapter 2. Additionally, I find that the WTP for disease prevention is increasing in severity of the disease. I also document evidence of dynamic complementarity; i.e. there is increasing returns to improvements in the quantity and quality of life. Finally, I use my model to revisit the mortality-reducing benefits of the Clean Air Act (CAA). I find that improvements in mortality due to CAA that occurred during the 2000's increased the overall benefits in 2010 by 6%. Overall, my results suggest WTP is endogenous to mortality and morbidity-reducing policies and that taking this endogeneity into account can improve efforts to evaluate these policies.

Chapter 4 investigates the effects of crisis-induced depression on illicit drug use among young adults aged 18-28. I use exogenous variation in depression created by an unique event – the 9/11 terrorist attack. Using data from in-home interviews of the National Longitudinal Survey of Adolescent to Adult Health (AddHealth), I find a significant jump in depressive symptoms by comparing individuals interviewed 30 days before and after the attack. The measure of depression that I use is a index following the Center for Epidemiological Depression Scale, which includes a set of questions measuring symptoms experienced in the past week relating to depression,

anxiety, loneliness and sadness.

I find that individuals who experience an increase in depression, are more likely to engage in illicit drug use and also binge drinking. The instrumental variable results are an order of magnitude larger than the OLS results and suggest that respondents use illicit drugs as a coping mechanism to alleviate the effects of depression. The effects are also heterogeneous across different types of drugs with the largest effects observed for marijuana. I also document behavioral change of substance use, which suggests that young adults are more likely to substitute alcohol with illicit drugs to cope with the effects of depression as they age. I also investigate how illicit drug use might affect educational attainment or labor supply. My partial identification strategy suggests that both depression and illicit drug use can cause sleep disorders and reduce both productive time in school or work and labor supply due to absenteeism. Overall, my estimates suggest that treatment of depression has significant potential to reduce the problem of substance abuse.Combining this finding with the evidence of dynamic complementarity in Chapter 3 suggest that policies that increase access to mental health care, like the Affordable Care Act, might create positive externalities in the form of complementary benefits of curbing drug abuse.

Chapter 2

VALUING STATISTICAL LIFE USING SENIORS' MEDICAL SPENDING (WITH NICOLAI KUMINOFF AND JONATHAN KETCHAM)

2.1 Introduction

Mortality rates are affected by many government activities such as regulating air pollution, setting speed limits, and funding health programs. Evaluating the equity and efficiency of these activities requires policymakers to weigh their benefits, including mortality reductions, against their costs. US federal agencies are required to evaluate many of these activities by reporting the expected monetary costs and benefits of every major regulation they propose. The standard approach to monetizing changes in mortality rates due to a regulation is to multiply the expected change in the number of premature deaths by a constant value per statistical life (VSL). The monetized mortality effects often dominate cost–benefit analysis. In fact, Lee and Taylor (2019a) report that survival gains represent up to 70% of all monetary benefits calculated for all federal regulations.

VSL measures are typically derived from econometric estimates of the compensating differentials paid to workers to induce them to perform jobs with higher risks of accidental death (Cropper *et al.* (2011a)). Workers whose choices generate this evidence are almost entirely under age 65, but people over 65 often account for a large share of policies' survival benefits. For example, senior citizens represent 75% of annual premature deaths avoided by regulating air pollution (U.S. Environmental Protection Agency (2011)). This discrepancy between the age group used to calculate VSL and the age group to whom VSL is applied may yield substantial mismeasure-

ment of the benefits of mortality reductions. Economic theory predicts that the VSL will evolve over the life cycle with changes in health, wealth, risk aversion and remaining life expectancy (e.g. Arthur (1981), Rosen (1988a), Evans and Smith (2006), Murphy and Topel (2006), Bauser *et al.* (2018)). However, the net effect of how these changes evolve with age is largely an empirical question, and researchers have not provided any revealed preference evidence on this evolution beyond age 65. [1] As a result, the sign and magnitude of mismeasurement under the status quo are unknown.

This paper is the first study to develop revealed preference evidence on how much Americans over age 65 are willing to pay to reduce their own mortality risks. The evidence comes from reconciled Medicare records linked to survey data on the rates at which people choose to consume medical care relative to other private goods. I view their choices through the lens of a life–cycle model in which people on fixed incomes make repeated decisions about how much to spend on medical care while facing uncertainty about their future health and survival. This setup is similar to the representative agent model in Hall and Jones (2007a) but focuses on individual decisions made by people with heterogeneous health, wealth, and preferences. Their optimal choices will equate their marginal cost of medical care (conditional on insurance coverage) with its marginal benefits as determined by the discounted expected utility of future life, where this utility depends on medical care's expected effects on the quantity and quality of remaining life. This equality yields a key insight: the marginal effect of medical expenditures on the probability of survival can reveal how much people are willing to pay for marginal increases in their survival probability. Measures of individual willingness to pay can then be aggregated to calculate the

[1] Prior research on age—related variation in the VSL has either employed stated-preference designs (e.g. Krupnick (2007), Blomquist *et al.* (2011a)) or stratified hedonic wage regressions by age bins (e.g. Smith *et al.* (2004), Evans and Smith (2006), Viscusi and Aldy (2007), Aldy and Viscusi (2008a), Evans and Schaur (2010)). Both approaches have yielded mixed results with no consensus for predicting how the VSL evolves beyond age 65.

VSL for groups of people who differ by age, health, income, and other characteristics.

I estimate VSL measures for a nationally representative random sample of about 22,000 people aged 67 to 97 who participated in the Medicare Current Beneficiary Survey (MCBS) from 2005–2011. The MCBS provides up to three years of reconciled Medicare records on each person's total and out–of–pocket (OOP) medical spending. These are the most comprehensive and accurate data on OOP spending for US seniors. They track all medical expenditures processed by Medicare, Medicaid, Medigap, employer–sponsored plans and other insurance plans, as well as any expenditures paid entirely OOP. The MCBS also reports each participant's insurance coverage, smoking history, income, education, employment status, knowledge of Medicare programs, utilization of assistance in making medical decisions, and their self–reported health and limitations in activities of daily living. I further link the MCBS to Medicare administrative records for the surveyed individuals, allowing me to additionally observe each person's demographics, residential location, medically diagnosed illnesses, and death date. Then I use the linked data to estimate survival functions.

The survival functions measure how an individual's medical spending in the current year affects her probability of surviving through the next year. A key identification challenge is that medical spending is likely to be positively correlated with latent morbidity. This may bias the estimated return to spending toward zero if people who are sicker in unobserved ways tend to spend more on health care and die sooner. I overcome this challenge by adapting the approach of Finkelstein *et al.* (2016) to derive an instrument for medical spending from geographic variation in the supply of medical care. Intuitively, some of the variation in individuals' medical expenditures arises from similar individuals facing sets of treatment options that differ in costs due to differences in health care supply across markets. The instrument is constructed for

individuals' medical spending using this supply–side variation by using a separate, larger dataset describing within–person changes in annual Medicare expenditures for just under half a million people who moved between Dartmouth Atlas hospital referral regions. When I use this index to instrument for medical expenditures, my main specification of the survival function implies that an additional $1,000 in spending reduces mortality in the following year by about 0.4 percentage points on average. This average marginal effect varies from about 0.2 to 2 percentage points across groups of people who differ in their health, demographic, and socioeconomic characteristics. This range is consistent with the range of local average treatment effects found in prior studies, as is that finding that the returns to spending increase with illness and age.

Combining my main estimates for the return to medical spending with each person's observed coinsurance rate yields a mean VSL of about $402,000 (year 2010 dollars) at age 67. Adjusting this value to account for estimated life expectancy and assuming a 7% discount rate implies a value per statistical life year (VSLY) of about $39,000. My estimates for age–specific VSL and VSLY measures decline as near–monotonic functions of age. I take a systematic and comprehensive approach to evaluating the sensitivity of these main findings to my research design, following Leamer (1983), Banzhaf and Smith (2007), and Greenstone *et al.* (2013). First, I define alternative analytic decisions along five dimensions: (i) sample criteria, (ii) source of data on medical expenditures, (iii) choice of instrument for medical expenditures, (iv) parametric form of the survival function, and (v) choice of covariates and spatial fixed effects. Then I estimate VSL measures for every possible combination of these decisions, yielding 200 sets of estimates. All of them produce mean VSLs below $1 million, and for all of them the VSL declines with age. Further, they all produce mean VSLYs below $100,000 at each age from 67 to 97 whether I assume a 3% or 7%

discount rate.

Next, I investigate heterogeneity. At age 67 the VSL is higher for women compared to men, people who never smoked compared to those who have, and for people with more income and education. In each of these comparisons, people with higher VSL tend to be healthier. These patterns are also evident when I stratify the VSL by subjective measures of health, objective measures of health, and limitations in activities of daily living. For example, 67 year old people who describe their health as "excellent" for their age on a Likert scale have an average VSL of $843,000, which is more than double the average VSL among all 67 year olds ($402,000), and more than twenty times the average for 67 year olds who describe their health as "poor" ($36,000). These group–wise differences appear to be due to differences in both quantity and quality of remaining life. As age increases, the VSL ranking across groups persists but the differences between their levels decline, consistent with declining differences in remaining life expectancy conditional on survival.

While the patterns of conditional heterogeneity in my VSL estimates can be rationalized by a life–cycle model, the levels of my estimates fall an order of magnitude below the range commonly used to monetize mortality reductions for seniors. Federal agencies typically assume a constant VSL between $6 and $10 million (year 2010 dollars) for every avoided death, regardless of age and health. [2] The $6 to $10 million range is consistent with evidence on average VSL from hedonic wage regressions of workers aged 18 to 65 (e.g. Costa and Kahn (2004), Cropper *et al.* (2011a), Deleire

[2]The one–size–fits–all approach to monetizing life extension in federal cost–benefit analyses is based on the US Office of Management and Budget's (U.S. Office of Management and Budget (2003)) judgment that there is insufficient evidence to guide age-based adjustments to VSL. They state that: "The age of the affected population has also been identified as an important factor in the theoretical literature. However, the empirical evidence on age and VSL is mixed. In light of the continuing questions over the effect of age on VSL estimates, you should not use an age adjustment factor in an analysis using VSL estimates." Political sensitivity to age adjustments came to light after the EPA proposed to reduce the VSL for seniors by 37% when calculating benefits of The Clear Skies Act, which became known as the "senior death discount" and was ultimately abandoned following controversy and opposition from interest groups (Viscusi and Aldy (2007)).

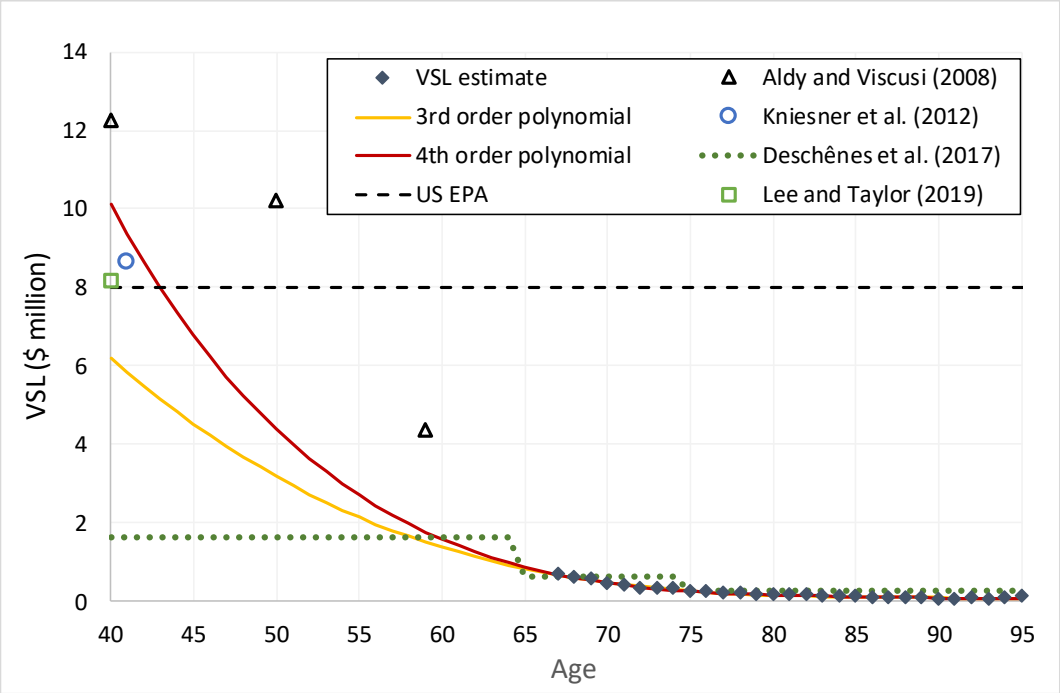
et al. (2013), Kneisner *et al.* (2012), and Lee and Taylor (2019a)).

Figure 2.1 summarizes how my estimates compare to the most closely related prior studies. The diamonds show my age–specific estimates for the mean VSL among relatively healthy people who report no major illnesses or functional limitations. My findings are closest to a VSL–age function that Deschenes *et al.* (2017) derived by calibrating the life—cycle model from Murphy and Topel (2006) to match a VSL measure constructed from evidence on speed limit changes (Ashenfelter and Greenstone (2004)). For example, Figure 1 shows that their calibrated value of $640,000 at age 67 is similar to my revealed preference estimate of $700,000; their calibrated value of $280,000 at age 75 is near my estimate of $240,000.

The solid lines in Figure 2.1 show projections made by regressing my age–specific estimates on third– and fourth–order polynomial functions of age and then projecting the fitted values back to age 40. This backward extrapolation of my estimates, while speculative, yields a predicted VSL range from $6 to $10 million for people in their early 40s. This range is consistent with evidence from well-identified hedonic wage studies (Kneisner *et al.* (2012), and Lee and Taylor (2019a)). Further, the decline from age 40 to age 62 tracks with the findings of Aldy and Viscusi (2008a). While additional research on medical consumption decisions among younger workers would be particularly insightful, these results suggest that the differences between my VSL estimates for US seniors and labor market evidence on younger healthier workers may be due to declines in health, functionality, and life expectancy that occur as people age rather than by methodological differences.

My approach shares methodological features with the wage–hedonic method while differing in some important ways. Both approaches assume that people make informed choices for how to continuously trade consumption for mortality risk. The estimates may diverge if the conventional revealed preference assumptions common to both

Figure 2.1: Comparing My Estimates to Prior Literature



Notes: The figure reports measures for the VSL in year 2010 dollars by age. The dashed line at $8 million is a benchmark one–size–fits–all VSL used by the US EPA and other agencies based on a review of academic studies comprised mainly of wage–hedonic regressions of workers age 18 to 65. The circle and square mark point estimates from wage–hedonic studies by Kneisner *et al.* (2012) and Lee and Taylor (2019a) that take steps to mitigate threats to identification. The dotted line shows calibrated values from Deschenes *et al.* (2017). The triangles show estimates from Aldy and Viscusi (2008a) for workers aged 35-44, 45-54, and 55-62. They note that fitting a third–order polynomial to their estimates implies a VSL close to $2 million at age 62. Finally, the diamonds show how my study fills the gap in knowledge about the VSL's evolution beyond age 65. Each diamond is an age–specific mean VSL calculated from the main IV survival function for people who report no functional limitations or chronic illnesses. I focus on this relatively healthy subsample to enable comparability with younger workers.

settings are less applicable to either labor market decisions or medical care choices. On one hand, my medical care setting provides stronger incentives for people to make careful choices and greater means to do so. Mortality risks are higher than in labor markets, information about risk is more accessible, and medical professionals are tasked with helping patients make informed decisions. Further, in contrast with a worker's job opportunities, seniors face essentially a continuum of options for the intensity and cost of medical care. In particular, patients can choose their medical care providers, including which physicians, specialties and hospitals as well as their treatment plans such as intensity and frequency of testing, use of newer versus older technologies, and medical versus surgical interventions.

On the other hand, health insurance is complex and everyone may not fully understand their treatment options and billing procedures. To explore the sensitivity of my estimates to maintaining revealed preference assumptions for everyone in my sample, I investigate how VSL estimates vary with seniors' medical decision–making processes and knowledge. I find that conditional on age there is virtually no difference in VSL measures between people who usually make their own health insurance decisions, people who get help making decisions from family, and people who rely on others to make decisions for them. In addition, I implement the strategy suggested in Bernheim and Rangel (2009) by using ancillary data on each person's cognitive functioning, decision–making, and knowledge of Medicare institutions to divide people into groups for whom revealed preference assumptions are more or less likely to hold. VSL measures are lower on average among the group for whom ancillary data provide reasons to suspect that choices may not reveal preferences, but the differences are too small to reconcile my estimates with those from the wage–hedonic literature. Further analysis indicates that these differences cannot be reconciled by physicians overtreating patients, or by patients ignoring the fact that insurance covers substantial shares

of their medical care costs.

The results in this study have two broad implications for evaluating the efficiency and equity of a wide range of activities that reduce mortality including environmental regulations, safety regulations, health insurance programs such as Medicare and Medicaid, and medical technology. First, because the VSL and VSLY estimates for seniors are far below the wage–hedonic estimates for younger healthier workers that have traditionally been used to monetize mortality reductions among seniors, using my estimates would reduce the monetized benefits of policies to varying degrees depending on the age and health of the beneficiaries. Second, the estimates imply that activities that improve health will increase the VSL and VSLY due to dynamic complementarity between the quantity and quality of life. The net effect of these two countervailing implications for estimated cost–benefit ratios likely varies across applications.

2.2 Data

I link panel data on Medicare Current Beneficiary Survey (MCBS) participants to administrative records on the same individuals from the US Centers for Medicare and Medicaid Services (CMS). The MCBS is a nationally representative rotating panel survey that is administered to approximately 16,000 randomly chosen Medicare beneficiaries each year. Each respondent is interviewed for up to four consecutive years even if they change addresses or move to long–term care facilities, and if they become cognitively impaired then someone else responds as their proxy. The linked data provide a nationally representative sample of the 65+ population because all Americans become eligible for Medicare benefits at age 65. [3]

[3]The linked data do not allow me to obtain a nationally representative sample of people under 65 because their Medicare eligibility stems from illness or poverty rather than age.

Importantly, the MCBS provides comprehensive measures of each respondent's total and out–of–pocket medical spending. CMS develops these measures by combining federal administrative records on the respondent's Medicare claims with the respondent's financial records on expenditures that were not processed through Medicare. However, due to the time needed to collect and reconcile these measures, they are only available for the second year of the survey onward. The MCBS also provides detailed information on each person's socioeconomic characteristics, household composition, labor market participation and self–assessed health. This complements the information available in CMS administrative records on each person's demographics, diagnoses of medical conditions, residential address and timing of moves, and death dates.

2.2.1 Sample Construction

The MCBS interview data from 2005 to 2011 for respondents over age 65 is linked to data extracted from each person's CMS administrative files from 2005 through 2012. The linked data contain 51,191 person–years with annual spending data for people who survived to the end of the calendar year. The minimum age is 67–the youngest age at which I observe MCBS respondents in their second full calendar year of survey participation. Then I make two sample cuts. First, I drop 730 person–years in which respondents declined to answer questions about their socioeconomic status or health, or their reported medical spending was zero, or their reported medical spending exceeded \$100,000. [4] Second, I drop 5,764 person–years where the respondent was employed at the time of their MCBS interview. Dropping workers simplifies my

[4]These data cuts retain 99% of my study population. Dropping the extreme tails of the expenditure distribution reduces the scope for outliers to affect my estimates. It also parallels labor market studies of the VSL such as Kneisner *et al.* (2012) which drops workers with real hourly wages below \$2 per hour or above \$100 per hour.

analysis by allowing me to avoid modeling how current medical spending may affect future income through intermediate health shocks that could, in principle, affect labor productivity and the timing of retirement (Grossman (1972)). However, section 2.6.1 shows that adding these workers to the estimation sample does not meaningfully change the magnitude of my VSL estimates relative to the status quo estimates.

The main sample is comprised of 22,206 people who are observed for 44,697 person–years. Individuals are observed for one, two or three years. It is not possible to observe three years of spending for everyone because some people die while enrolled in the MCBS and others' MCBS enrollment cycles extend beyond the endpoints of my study period. Finally, I use administrative data on death dates to observe one–year mortality for everyone in the sample, including those who exit the MCBS during my study period. Table 2.1 reports summary statistics. The average person is 78 years old and 5% die during the year after I observe their medical spending. The distribution of people by sex, race and educational attainment matches 2010 Census data on the US population age 65+. [5] I also see that about half are married and 93% have living children.

2.2.2 Medical Expenditures

The US Medicare program provides universal health insurance for Americans over age 65. Enrollees can choose between traditional "fee–for–service" Medicare that pays medical care providers a fixed fee for each service they perform and Medicare Advantage plans that charge a monthly premium in exchange for lower out–of–pocket (OOP) costs for certain services. Some people have additional health insurance provided by their former employers or spouses' employers, and some people purchase

[5] American Community Survey data for 2010 identify 85% of the US population age 65+ as white, 57% as female, and 21% as having a bachelor's degree or higher.

Table 2.1: Summary Statistics

Measures	Mean	Data Source
1-year mortality (%)	5	Admin
Age	78	Admin
Female (%)	58	Admin
White Non-Hispanic (%)	85	Admin
Education: College Degree(%)	21	MCBS
Married(%)	52	MCBS
Has Living Children (%)	93	MCBS
Gross Medical Spending (2010 $)	11,489	MCBS-Admin
Out-of-pocket Medical Spending (2010 $)	1,817	MCBS-Admin
Ever-Smoked(%)	58	MCBS
Underweight BMI (%)	4	MCBS
Number of Chronic Conditions (out of 61)	7	Admin
Mean Log of HCC Scores	-0.27	Admin
Self-reported Health =“Poor/Fair” (%)	21	MCBS
Self-reported Health =“Good/Very Good” (%)	64	MCBS
Self-reported Health =“Excellent” (%)	15	MCBS
1+ limitations of Instrumental Activities of Daily Living (%)	28	MCBS
1+ limitations of Activities of Daily Living (%)	30	MCBS
Total Number of People	22,206	
Total Number of Person Years	44.691	

Notes: Spending measures are adjusted to year 2010 US dollars using the CPI. Variables with the “MCBS” label are based on survey responses. Variables with the “admin” label are drawn from CMS administrative files. The spending variables are labeled “MCBS–admin” because they combine information from administrative files and MCBS–based tracking of respondents’ medical and financial records.

private Medigap insurance plans to supplement their public Medicare coverage. The MCBS spending measure includes all of these public and private forms of coverage as well as expenditures paid entirely OOP.

The MCBS reports comprehensive measures of each respondent's total and OOP medical spending during their second, third and fourth years of survey participation. These data are considered the best available for measures of OOP spending among the US Medicare population and they include costs for services not covered by Medicare. They account for all payments by third–party payers, including Medicaid, Medigap, or employer–sponsored insurance, which may cover some or all of the typical patient cost–sharing under Medicare. The data are collected from respondents who record medical events in calendars and keep documentation and receipts, e.g. from insurers, pharmacies, and Medicare explanations of benefits. CMS then reconciles these records with its administrative data on insurance claims. The resulting spending measures are more comprehensive than Medicare claims because they also include expenditures that were not processed through the Medicare system or not retained in CMS's administrative files during my study period. Examples include prescription drug expenditures made before Medicare started subsidizing drugs in 2006, spending in Medicare Advantage and Medigap plans, and expenditures paid entirely OOP with no claim submitted, e.g. some generic drugs. Equally important is the fact that the reconciled spending measures provide a detailed accounting of how expenditures were divided across payees including the federal government, employer–sponsored plans, private insurers, and the beneficiary. This accounting allows me to observe the fraction of each MCBS respondent's total annual medical expenditures that were paid OOP, i.e. their effective annual coinsurance rate. [6]

[6]CMS's official description of these files states: "The MCBS Cost and Use files link Medicare claims to survey–reported events and provides complete expenditure and source of payment data on all medical care services, including those not covered by Medicare. Expenditure data were

Table 2.1 shows that the average person spent $11,489 on medical care annually. [7] OOP expenditures on medical services were on average $1,817, which is equivalent to 7% of per capita income for the over–65 population in 2010 (US Current Population Survey, 2011).

2.2.3 Health

The lower part of Table 2.1 reports means for several measures of health. First, I track whether people face a statistically higher mortality risk because they have a history of smoking (58%) or were underweight based on their body mass index at the time of the survey (4%). Second, I use CMS Chronic Conditions Warehouse files to identify whether and when each person was first diagnosed with chronic illnesses based on insurance claims. [8] The average person is diagnosed with 7 illnesses (out of 61). Third, I use data on CMS's hierarchical conditions categories (HCC) risk–adjustment score. HCC scores synthesize data on diagnosed illnesses, age, gender,

developed through a reconciliation process that combines information from survey respondents and Medicare administrative files. The process produces a comprehensive picture of health services received, amounts paid, and sources of payment. The file can support a broader range of research and policy analyses on the Medicare population than would be possible using either survey data or administrative claims data alone. Survey-reported data include information on the use and cost of all types of medical services, as well as information on supplementary health insurance, living arrangements, income, health status, and physical functioning. Medicare claims data includes use and cost information on inpatient hospitalizations, outpatient hospital care, physician services, home medical care, durable medical equipment, skilled nursing home services, hospice care, and other medical services."

[7] This statistic is for 12 months of spending. To measure per capita expenditures consistently I exclude the calendar years in which people die. The median death occurs in early July.

[8] The set of chronic conditions includes: acute myocardial infarction, ADHD and other conduct disorders, anemia, anxiety, asthma, atrial fibrillation, bipolar disorder, brain injury, cancer (breast, colorectal, prostate, lung, endometrial), cataract, cerebral palsy, chronic kidney disease, chronic obstructive pulmonary disease, congestive heart failure, dementia, depression, diabetes, epilepsy, fibromyalgia, glaucoma, hearing impairment, hip fracture, HIV, hyperlipidemia, hypertension, hypothyroidism, heart disease, intellectual disabilities, learning disabilities, leukemia, liver disease, mild cognitive impairment, migraine, mobility impairment, multiple sclerosis, muscular dystrophy, other development delays, personality disorders, post-traumatic stress disorder, obesity, osteoporosis, peripheral vascular disease, rheumatoid arthritis, schizophrenia, spina bifida and other congenital anomalies of the nervous system, spinal cord injury, stroke, tobacco disorder, ulcers, visual impairment, viral hepatitis.

and initial reason for Medicare eligibility into a normalized index of health risk that CMS uses to made capitation payments to Medicare Advantage plans. [9]

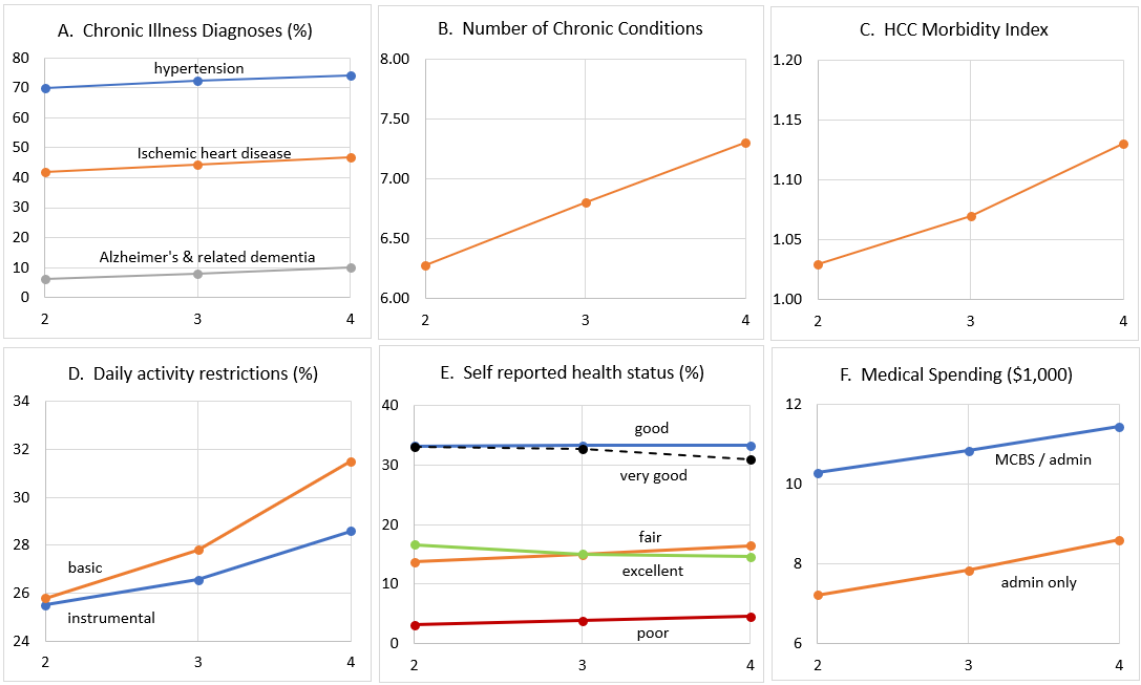
The objective measures of health are augmented with subjective measures recorded in the MCBS. Respondents are asked, "In general, *compared to other people your age,* would you say that your health is ... excellent, very good, good, fair, or poor?" (emphasis added). Table 2.1 shows that the distribution of self–reported health is slightly left–skewed with 79% of people reporting that their health is good, very good, or excellent. I also track whether morbidity interferes with respondents' daily lives. The MCBS reports whether people say they are capable of performing various activities of daily living (ADL). Approximately 28% of respondents have difficulty performing at least one "instrumental" ADL, which includes activities that affect the ability to live independently such as managing money, doing household work, using the telephone and preparing meals. Approximately 30% of respondents report difficulty in performing one or more "basic" ADLs such as bathing, dressing, eating, walking, and using the bathroom. These subjective variables may help to capture latent heterogeneity in health not captured by the objective measures. For instance, people who have difficulty performing tasks of daily living because of mobility limitations may also be more likely to suffer from more severe and debilitating symptoms of heart disease than other people with heart disease.

2.2.4 The Evolution of Health and Medical Spending

Figure 2.2 illustrates how health declines and medical spending increases with age. The figure documents the evolution of health and spending over MCBS years 2 through 4 for the subset of people in Table 2.1 whom I observe for all three years. As

[9]Background information on CMS's HCC model can be found at `http://www.nber.org/data/cms-risk-adjustment.html`. I follow Finkelstein *et al.* (2016) in adjusting raw HCC scores for spatial and temporal trends. This adjustment is described in Appendix A.1.

Figure 2.2: Evolution of Health and Medical Spending Over MCBS Years 2 to 4



Notes: The figure summarizes the evolution of health and medical spending during years two through four of the Medicare Current Beneficiary Survey, the period for which comprehensive spending measures are observed. The figure is constructed from data on the subset of respondents who are observed in all three survey years.

the average respondent ages from 77 to 79, they are more likely to be diagnosed with chronic conditions. For example, panel A shows that the share of people diagnosed with hypertension increases from 70% to 74%, the share diagnosed with ischemic heart disease increases from 42% to 47%, and the share diagnosed with Alzheimer's disease and related dementia increases from 6% to 10%. Panel B shows that the average person is diagnosed with a total of 6.3 chronic illnesses in year 2 and that this increases to 7.3 by year 4. Panel C shows that the average HCC morbidity score increases with the average number of chronic illnesses.

As people get older and sicker, Figure 2.2 shows that they are more likely to

experience restrictions on instrumental and basic activities of daily living (panel D). Yet self–reported health status is relatively stable (panel E). This is consistent with the fact that the question is asked relative to others of the same age. Finally, panel F shows that per capita medical spending increases by 5% to 6% per year. While the reconciled MCBS measures of total medical spending that I rely on are larger than spending measures constructed from Medicare claims alone, their trends are nearly parallel.

2.3 A Dynamic Model of Medical Expenditures, Health, and Survival

I use a dynamic model to explain how retirees choose to adjust their medical spending as they experience health shocks that affect their expected future quantity and quality of life. People enter the model at age 65 with endowments of health and wealth. [10] Each year they determine how much to spend on non–medical consumption and medical services which, in turn, affect their future health and wealth.

People face two sources of uncertainty when they make decisions. First, their health evolves through a partially stochastic process. The stock of health declines with age, on average, but the decline can be slowed or temporarily reversed by investing in medical care. Second, people can die at any time. Survival to the next period is modeled as a probabilistic function of age, health, and medical expenditures. Hence, people can increase the expected quantity and the quality of their lives by purchasing medical services to slow the degradation of their health stock and reduce their short–term probability of death. When people decide how much to invest in health they face an intertemporal tradeoff. Increasing medical expenditures decreases their current

[10] The median retirement age in the United States is 62. Individuals born before 1955 received full retirement benefits from the Social Security Administration if they retired at age 66. Among all individuals age 66 and over in the Medicare Current Beneficiary Survey, approximately 13 percent were working in 2010.

quality of life by reducing non–medical consumption, but it increases their expected future quantity and quality of life through the health stock and survival probability. Under standard assumptions, the way that people respond to this tradeoff reveals their willingness to pay for marginal changes in probabilistic life extension.

Many decisions about individual medical care may be made at the household level, or even without the individual's input in the case of people suffering from dementia and other cognitive impairments. Here I abstract from the complications of within–household bargaining and make no distinction between the decision–maker and the individual receiving care. My model also embeds versions of the "continuous choice" and "full information" assumptions that are ubiquitous in the revealed preference literature on VSL estimation. Specifically, I assume that people are free to purchase medical services in continuous quantities and that they do so knowing how those purchases will affect their probability of surviving through the end of the following period. Section 2.8 takes a step toward relaxing these standard but strong revealed preference assumptions by stratifying the VSL measures based on whether people make their own medical care decisions and consider themselves to be well informed.

2.3.1 Preferences and Health

In each time period, t, retiree i's utility depends on her amount of non–medical consumption, c_{it}, and health, h_{it}:

$$U_{it} = u(c_{it}, h_{it}). \tag{2.1}$$

Health evolves over time as a function of medical expenditures, m_{it}. The retiree's stock of health in period $t+1$ depends on her period t health stock, medical expenditures, age, and a random shock denoted by ϵ_{it}. In a slight abuse of notation I use t to index both age and time period so that evolution of the health stock can be

represented as

$$h_{it+1} = f(h_{it}, m_{it}, t, \epsilon_{it}). \tag{2.2}$$

Equation (2.2) captures the essence of a Grossman (1972) style health production function. Each retiree inherits a stock of health upon entering the model at age 65. Their initial stock may reflect genetic endowments and the cumulative effects of past medical consumption, lifestyle choices, pollution exposures, and health shocks. Health depreciates following negative shocks, but such declines can be partially offset by medical expenditures. For instance, health shocks may be caused by the arrival or worsening of conditions such as diabetes, heart disease, and cancer. Their negative effects on future health may be moderated by seeing doctors to obtain medications, surgeries, preventative care, or guidance on lifestyle choices such as diet and exercise. However, the return on medical care investments may decline with age.

I model death using a distinct probabilistic function. Let s_{it} represent person i's probability of survival to period $t + 1$. Survival is assumed to be a deterministic function of period t medical expenditures, health, age, and a random shock, μ_{it}. Integrating over the shock yields the survival probability.

$$s_{it} = g(m_{it}, h_{it}, t). \tag{2.3}$$

Together, equations (2.2) and (2.3) illustrate how medical expenditures may increase both the quantity and quality of life in future periods. Investing in medical care may increase the future quality of life through the health stock, and increasing the health stock may lower the short–term probability of death. Medical care investments may also increase the survival probability directly without affecting the health stock. Examples include medications and surgeries that reduce the chances of a fatal heart attack or stroke. Overall, the dynamic, stochastic nature of the return to investment in medical care presents budget–constrained retirees with a tradeoff

between increasing current utility through non–medical consumption and investing in future utility through medical consumption.

2.3.2 Intertemporal Budget Constraint

For simplicity, I abstract from credit markets and require people to maintain non–negative assets each period. Equation (2.4) shows the intertemporal budget constraint.

$$a_{it+1} = (1+r)a_{it} + y_i - c_{it} - \gamma_{it} m_{it} \ \geq \ 0 \ \forall t. \tag{2.4}$$

The retiree's total assets in period $t+1$ are equal to the assets retained from the prior period, a_{it}, which are assumed to grow at a risk–free interest rate of r, plus non–asset income from all other sources, y_i, less expenditures on medical and non–medical consumption. Medical expenditures are subsidized by the government so that person i's out–of–pocket costs are equal to $\gamma_{it} m_{it}$. The subsidy rate, γ_{it}, varies across people depending on their mix of medical services. Finally, retirees' incomes are assumed to be fixed.

2.3.3 The Dynamic Optimization Problem

The retiree's dynamic optimization problem can be expressed as the following Bellman equation:

$$V_t(a_{it}, y_i, h_{it}) = \max_{\{c_{it}, m_{it}\}} u(c_{it}, h_{it}) + \alpha_i \ s_{it}(m_{it}, h_{it}, t) \ E[V_{t+1}(a_{it+1}, y_i, h_{it+1})]. \tag{2.5}$$

Each period, the agent allocates assets toward medical and non–medical consumption to maximize expected utility over the remaining lifetime, with a discount factor of α_i. The expectation operator is taken with respect to the following period's health stock. The maximization problem is subject to the budget constraint in (2.4), the survival

function in (2.3) and the health production function in (2.2). [11]

The agent maximizes utility by choosing levels of medical and non–medical consumption such that their marginal utilities are equalized at each age. Solving the optimization problem in period t and combining the first–order conditions yields the following expression:

$$\frac{1}{\gamma_{it}}\left[\alpha_i EV_{t+1}(a_{it+1}, y_i, h_{it+1})\frac{\partial s_{it}}{\partial m_{it}} + \alpha_i s_{it}\frac{\partial E[V_{t+1}(a_{it+1}, y_i, h_{it+1})}{\partial m_{it}}\right] = u_c(c_{it}, h_{it})]. \tag{2.6}$$

The first term inside brackets reflects the discounted stream of benefits from increasing the survival probability by investing an additional dollar in medical expenditures. The second term captures the associated return in terms of improved future health. Dividing by the marginal utility of income and rearranging terms yields an equilibrium condition equating the marginal benefits and costs of investing in medical care for probabilistic life extension:

$$\frac{\alpha_i EV_{t+1}(a_{i,t+1}, y_i, h_{it+1})}{u_c(c_{i,t}, h_{i,t})} + \alpha_i \frac{s_{i,t}}{u_c(c_{it}, h_{it})} E\left[\frac{\partial V_{t+1}(a_{it+1}, y_i, h_{it+1})(f_m/g_m)}{\partial h_{it+1}}\right] = \gamma_{it}\frac{\partial m_{it}}{\partial s_{it}}. \tag{2.7}$$

The expression to the left of the equality in (2.7) defines the expected marginal private benefit of increasing the survival probability via medical expenditures. The first term is the benefit of surviving to the next period conditional on the health stock. The second term captures the co–benefit of increasing the future health stock via the same investment in medical consumption that increases the survival probability. The ratio $f_m/g_m = \frac{\partial h_{it+1}}{\partial m_{it}}\frac{\partial m_{it}}{\partial s_{it}}$ tracks how the increase in medical expenditures that is used to marginally increase the survival probability affects the future health stock which, in turn, influences both the quality of life and the survival probability in future periods.

[11] Because I do not explicitly model bequests, the utility value of transferring wealth to others is implicitly included as a form of non-medical consumption.

The expression to the right of the equality in (2.7) defines the marginal private cost of increasing the survival probability. This statistic is proportional to the cost of saving a statistical life. For instance, if increasing medical expenditures by \$10,000 increases the survival probability by 0.001, then the total cost of avoiding the death of one type i individual at age t is \$10 million. If $\gamma_{it} = 0.5$ then the private out–of–pocket cost of avoiding that death for type i individuals is \$5 million.

The equilibrium condition in (2.7) relates two important statistics for evaluating public policies that may affect people's health and survival: the marginal return to medical spending and the VSL. The relationship between them suggests a simple "sufficient statistics" approach to estimating VSL. First estimate the survival function, then differentiate with respect to medical spending to calculate $\frac{\partial s_{it}}{\partial m_{it}}$, which can be rescaled by the coinsurance rates to calculate private VSL for a type i individual at age t, as in (2.8):

$$VSL_{it} \propto \frac{\gamma_{it}}{\partial s_{it}/\partial m_{it}}. \tag{2.8}$$

This VSL measure recognizes that an agent's willingness to pay for a marginal change in statistical life extension may depend on their expected future health. Assuming that flow utility is strictly increasing in health and that expected future health is weakly increasing in medical spending, equation (2.7) implies that the VSL revealed by medical spending will exceed a hypothetical health-neutral VSL. [12] This feature is also common to the market environments used to estimate VSL for younger people. For example, improving workplace safety is likely to reduce the risk of a variety of non-fatal injuries as a co-benefit to reducing the risk of death on the job.

Under additional assumptions, the model can be used to formalize hypotheses about sources of heterogeneity in the VSL. For instance, under mild restrictions on

[12] Alternatively, a health-neutral VSL could exceed the measure in (2.8) if medical spending reduces future health, for example through undesirable side effects of prescription drugs.

similar life–cycle models, (Murphy and Topel (2006) and Hall and Jones (2007a)) predict that the VSL will increase in wealth and decline in age among retirees on fixed incomes because the health stock and survival probability both tend to decline beyond age 65. Similarly, Dow *et al.* (1999) and Murphy and Topel (2006) predict that complementarity between different types of health investments will cause the VSL to decline as people experience negative health shocks. For example, a diagnosis of heart failure may reduce the VSL by accelerating the expected decline of health. I test these and other hypotheses about heterogeneity in the VSL in Section 2.7.

2.4 Econometric Model of Survival

As the theoretical model demonstrates, a key empirical object for my approach to measuring VSL is the individual–specific returns to medical spending in terms of reduced mortality. Prior work on estimating the return to medical spending used long–term aggregate measures (Hall and Jones (2007a)) or estimated local average treatment effects for specialized cohorts of patients or specific types of medical spending (Huh and Reif (2017a), Clayton (2018)), Doyle *et al.* (2015a), Romley and Sood (2013)) such as hospital spending for patients who were hospitalized through the emergency department for heart attacks while visiting Florida (Doyle (2011a)). Because prior literature does not provide the information needed for my approach, one contribution of this study is to provide the full set of estimates of the marginal returns to medical spending across the full range of age, health and other characteristics.

I model survival as a discrete–time process over the annual intervals at which individuals' medical expenditures are observed. Each year, death is predicted by lagged values for medical expenditures and health. Formally, let s_{it}^* be a latent variable that determines survival, scaled so that person i lives through period $t+1$ if and only if $s_{it}^* > 0$. Survival depends on medical expenditures, health, age, and a random

shock:

$$s_{it}^{*} = \beta + \beta_m m_{it} + \beta_h h_{it} + \beta_t t - \mu_{it}. \tag{2.9}$$

The probability of survival, s_{it}, can be represented as

$$s_{it} = Pr(s_{it}^{*} > 0) = Pr(\beta + \beta_m m_{it} + \beta_h h_{it} + \beta_t t > \mu_{it}). \tag{2.10}$$

Under the assumption that survival shocks are *iid* draws from a Type I extreme value distribution, the survival probability takes the complementary log–log form,

$$s_{it} = \exp(-\exp(\beta + \beta_m m_{it} + \beta_h h_{it} + \beta_t t)). \tag{2.11}$$

This parametric form is an intuitive choice for modeling death among older adults because the model's asymmetry allows the probability to approach 1 (survival) slowly relative to the rate at which it approaches 0 (death). [13] I measure the explanatory variables at the end of calendar year t so that I am modeling how survival during a particular year depends on health and age at the start of that same year, along with total medical expenditures for the prior year.

Latent health presents a key challenge to identifying the survival model parameters. While the data contain a rich set of measures of each person's health (Table 2.1), any function of those variables is still likely to contain some error in measuring the true stock of health that determines survival. This problem is magnified by the potential for the latent component of health to be correlated with both medical expenditures and survival. That is, people who are sicker in unobserved ways are likely to have higher medical spending and lower survival rates. [14] All else constant, this

[13] If I rescale the dependent variable to be 1 in the case of death the resulting mortality function is commonly known as the Gompit model because of its similarity to the Gompertz model of human mortality. Section 2.6.1 shows that my main findings are robust to estimating a Gompertz mortality function.

[14] For example, consider the severity of disease. CMS records allow me to observe if and when each individual is first diagnosed with chronic kidney disease, but I am unable to directly observe whether the individual's kidneys are mildly damaged (stage 1) or have already failed so that the individual requires costly dialysis treatments or a kidney transplant to survive (stage 5).

simultaneity will lead to a downward bias in the estimator for the marginal effect of medical spending on survival and upward bias in the estimator for the VSL. I use several different instrumental variables approaches to mitigate this threat.

2.4.1 Instrument for Medical Expenditures

Economists have often used geographic variation in medical treatment style to construct instruments for measuring how medical care affects survival (e.g. McClellan *et al.* (1994), Stukel *et al.* (2007), Currie and Slusky (2020)). My featured instrument adapts the method developed in Finkelstein *et al.* (2016) to decompose geographic variation in medical expenditures across the 306 US Hospital Referral Regions (HRRs) into demand–side factors and place–specific supply factors. [15] Making this decomposition enables me to identify the survival model parameters from variation in medical expenditures that is unrelated to patient health. Intuitively, people with identical morbidities who live in different geographic areas face menus of treatment options with different costs, which leads to variation in their medical expenditures and survival probabilities. I develop an index of this supply–side variation to use as an instrument for individuals' medical expenditures.

The logic for the instrument starts from the observation that per capita annual medical expenditures vary greatly across the United States among the Medicare population. [16] Some of this variation may reflect patient preferences and health, whereas

[15] "Hospital Referral Regions" represent regional medical care markets for tertiary medical care as determined by the Dartmouth Atlas. Each HRR contains at least one hospital that performs major cardiovascular procedures and neurosurgery. HRRs were defined by assigning Hospital Service Areas to the region where the greatest proportion of major cardiovascular procedures were performed, with minor modifications to achieve geographic contiguity, a minimum population size of 120,000, and a high localization index. The Dartmouth Atlas defines a Hospital Service Area as a collection of ZIP Codes whose residents receive most of their hospitalizations from hospitals in the area. For further details see: `http://www.dartmouthatlas.org/downloads/methods/geogappdx.pdf`.

[16] This fact is well documented. For evidence, see Finkelstein *et al.* (2016), Cutler *et al.* (2019) and references therein.

some of the variation reflects differences in the supply of medical care. For example, Cutler *et al.* (2019) points to the importance of differences in physician practice style, highlighting that aggressive treatment practices increase spending. Chandra and Staiger (2007) highlights the importance of productivity spillovers and physician migration. Other supply factors that may contribute to spatial variation in expenditures include peer effects among physicians, differences in physical capital, and institutional features of local medical care markets. Against this background, Finkelstein *et al.* (2016) use Medicare administrative records on patients who move between HRRs to implement a regression–based procedure to decompose the spatial variation in patients' expenditures into supply and demand factors, finding that each source accounts for about half of the total variation. I follow their approach to estimation and use the resulting measure of regional supply–side variation in expenditures to instrument for individuals' total expenditures. In Section 2.6.1 I describe other ways of constructing this instrument and alternative instruments altogether and show that the VSL estimates are robust across these analytic decisions.

I construct the instrument from 3.2 million person–years of claims-based data on expenditures for 484,000 people over age 65 who were enrolled in traditional Medicare and changed their residential address from one HRR to another exactly once between 1999 and 2013. These data were extracted from a 10% random sample of Medicare beneficiaries. I use CMS administrative records for this random sample of movers to estimate the supply–side component of their Medicare expenditures that varies across HRRs,

$$m_{ijt} = \sigma_i + \phi_j + \chi_t + \psi X_{it} + o_{ijt}. \tag{2.12}$$

The dependent variable in the regression is total medical expenditures in year t for a person living in HRR j. Covariates include an individual fixed effect, σ_i, an HRR fixed effect, ϕ_j, a year fixed effect, χ_t, a vector of time–varying person–specific co-

variates, X_{it}, and an orthogonal error term, o_{ijt}. I follow Finkelstein *et al.* (2016) in defining X_{it} to include dummies for 5–year age bins and dummies for the current year relative to the year in which an individual is observed moving between HRRs. These relative–year dummies allow migration decisions to coincide with unobserved health shocks that simultaneously affect the demand for medical care and the desire to live close to caregivers such as children. The resulting vector of HRR fixed effects, $\hat{\phi_1}, ..., \hat{\phi_{306}}$, provides an index for spatial variation in medical expenditures driven by supply factors.

The validity of using $\hat{\phi}$ to instrument for medical expenditures rests on the assumption that it is uncorrelated with latent health. While this assumption cannot be tested directly, the specification in (2.12) is designed to reduce the scope for such correlation. Finkelstein *et al.* (2016) provide a detailed discussion of how the HRR fixed effects in equation (2.12) are identified by supply–side variation, holding health fixed. In summary, the index $\hat{\phi}$ is identified by the ways in which changes in movers' medical expenditures differ between their origin and destination locations. To see this, first notice that if people never moved then $\hat{\phi}$ could not be identified because the individual fixed effects would absorb all of the spatial variation in average per/capita expenditures. Second, the year–relative–to–move fixed effects included in X_{it} absorb average trends in medical expenditures around the move. This forces the identification to come from differential changes in expenditures across people undertaking different migration patterns. While equation (2.12) allows expenditures to differ arbitrarily between movers (via σ_i) and to differ systematically around their moves (via X_{it}), Finkelstein *et al.* (2016) point out that it maintains the assumption that health shocks leading to expenditure changes do not precisely coincide with the timing of moves. I relax this assumption by the way I constuct the estimation sample. Specifically, my estimation sample excludes movers who were newly diagnosed with any

chronic conditions during their move year. [17] Thus, the instrument is identified by differential changes in medical expenditures among people who move between HRRs and do not experience observed health shocks during their move years.

2.4.2 Main Econometric Model

I instrument for total medical expenditures in a linear first–stage regression,

$$m_{it} = \pi + \pi_h h_{it} + \pi_t t + \pi_z z_{it} + \omega_{it}, \tag{2.13}$$

where the supply side expenditure index for person i living in HRR j in year t is defined as $z_{it} = \hat{\phi}_j - \hat{\phi}_k$, and k is used to index an arbitrary reference location. I then estimate the survival function as the second stage control function that includes the first–stage residuals:

$$s_{it} = \exp(-\exp(\beta + \beta_m m_{it} + \beta_h h_{it} + \beta_t t + \hat{\omega}_{it})), \tag{2.14}$$

where h_{it} includes measures of demographics, socioeconomic status, and health from Table 2.1 as proxy measures for the health stock. Finally, I use the parameter estimates to predict the marginal effect of medical expenditures on survival and rescale it by the coinsurance rate to calculate the VSL measure from equation (2.8).

In summary, the estimation approach proceeds as follows. First I use Medicare claims data to construct the expenditure instrument from equation (2.12). Then I estimate equations (2.13) and (2.14) using the linked MCBS–administrative data. The two–stage control function approach yields a consistent estimator for model parameters under the assumption that the instrument is valid and the survival function is correctly specified (Terza *et al.* (2008), Wooldridge (2015)). The standard errors and

[17] I also follow Finkelstein *et al.* (2016) in dropping the very small fraction of people who moved multiple times because such individuals complicate the definition of the year–relative–to–move fixed effects.

confidence intervals are calculated using a nonparametric bootstrap over sequential estimation of (2.13) and (2.14) with the errors clustered by HRR to coincide with the identifying source of variation in the instrument (Cameron *et al.* (2008), Abadie *et al.* (2017)).

2.5 Main Results

2.5.1 Evidence on Supply–Side Variation in Medical Expenditures

I use equation (2.12) to estimate HRR fixed effects from claims–based data on movers. Then I normalize the estimates relative to Birmingham, AL. The normalized estimates range from +$2,500 for Miami, FL to -$1,150 for Greensboro, NC. Moving from the 10th percentile to the 90th percentile in the distribution of HRR effects is equivalent to increasing annual expenditures by $1,870, or approximately 22% of the mean expenditure that is observed in claims–based data for traditional Medicare enrollees in MCBS data. [18] Likewise, the between–HRR standard deviation is $661 (7% of the mean expenditures). These results are consistent with Finkelstein *et al.* (2016) in suggesting that supply–side factors explain a substantial share of the between-HRR variation in Medicare expenditures per person. [19]

Next I collapse the normalized fixed effects into an index of supply–side variation in average expenditures, assign index values to MCBS respondents based on their residential locations, and use this variable to instrument for their annual medical expenditures. To test the hypothesized supply–side mechanisms underlying the instrument I regress it on measures of physician treatment style constructed by Cutler

[18] Figure C.1 shows the entire distribution of HRR estimates.

[19] Because these estimates exclude all movers who are diagnosed with new chronic conditions during their move years my estimates can also be interpreted as providing additional support for Finkelstein *et al.* (2016) by showing that their qualitative findings are robust to relaxing their maintained assumption that expensive health shocks do not coincide with moves.

et al. (2019). Specifically, I use their measures for each HRR's fraction of "cowboy" physicians who consistently recommend intensive care beyond clinical guidelines and the fraction of "comforter" physicians who consistently recommend palliative care for the seriously ill. [20] Consistent with the hypothesized mechanisms, a standard deviation increase in the cowboy share is conditionally associated with a 0.16 standard deviation increase in the IV, whereas a standard deviation increase in the comforter share is associated with a 0.17 standard deviation decrease in the IV.

Finally, I test whether the claims–based instrument for medical spending has power to explain variation in medical expenditures that are not reflected in Medicare claims, for example among the MCBS sample on Medicare Advantage. The answer is yes. For each MCBS respondent enrolled in traditional Medicare I calculate the difference between the comprehensive MCBS expenditure measure and the corresponding claims–based measure from CMS administrative files. Univariate regression reveals that a standard deviation increase in the instrument is associated with a 0.04 standard deviation increase in expenditures not processed by Medicare compared to a 0.07 standard deviation increase in expenditures processed by Medicare. These coefficients only decline by about 25% when I add the comprehensive set of covariates described below. Thus, the identifying variation in medical spending comes partly from services covered by Medicare and partly from services that are covered entirely by a combination of Medicare Advantage plans, Medigap plans, employer plans and OOP spending.

[20] I thank Jon Skinner for sharing these data.

2.5.2 The Effect of Medical Expenditures on Survival

Table 2.2 reports average marginal effects for survival functions, using 1,000 bootstrap replications to calculate standard errors. [21] In addition to the health covariates featured in the table, all specifications include the demographic and socioeconomic variables summarized in Table 2.1. First–stage coefficients on the instrument and associated F–statistics are reported at the bottom of the table and unabridged estimates from the first– and second–stage models are reported in Appendix Tables B.1 and B.2.

The model in column (1) ignores the potential endogeneity of medical spending. The positive marginal effect indicates that, all else constant, a $1,000 increase in spending is associated with a 0.06 percentage point increase in the one–year mortality rate. This counterintuitive result is consistent with the idea that people who are sicker in unobserved ways will tend to spend more on medical care and die sooner. This counterintuitive result disappears when I instrument for medical spending.

Column (2) reports results from the control–function analog to column (1). The first–stage residual measure of latent morbidity has a positive, statistically significant coefficient. This supports the view that unobserved latent health is positively correlated with both medical expenditure and mortality and that the assumed exogeneity of medical expenditure in column (1) is unlikely to hold true.

The IV results implies that a $1,000 increase in medical spending reduces the one–year mortality rate by just under half a percentage point. The first–stage F–statistic and IV coefficient reported towards the bottom of the table indicate sufficient power of IV and that a marginal dollar increase in the supply–side index of medical expenditures is associated with less than a dollar increase in total expenditures.

[21] Each replication repeats both stages of estimation.

Table 2.2: Average Marginal Effects on Mortality

Outcome: Mortality in year t+1	(1)	(2)	(3)	(4)	(5)	(6)
$1,000 in medical spending	0.06***	-0.47***	-0.42***	-0.64***	-0.88***	-0.82*
	(0.01)	(0.20)	(0.18)	(0.26)	(0.63)	(5.80)
1st stage residual morbidity		0.54***	0.49***	0.71***	0.95***	0.88*
		(0.20)	(0.18)	(0.26)	(0.63)	(5.80)
HCC index	3.10***	11.11***	10.72***	13.91***	17.77***	16.73**
	(0.31)	(3.01)	(2.67)	(3.86)	(9.42)	(86.50)
one of more ADL restrictions	1.70***	3.05***	2.96***	3.51***	4.13***	4.02**
	(0.23)	(0.56)	(0.52)	(0.70)	(1.57)	(14.31)
one or more IADL restrictions	0.50**	1.28***	1.22***	1.54***	1.92***	1.86**
	(0.24)	(0.40)	(0.37)	(0.49)	(0.99)	(8.74)
ever smoked	1.19***	1.30***	1.27***	1.31***	1.34***	1.33***
	(0.22)	(0.25)	(0.24)	(0.26)	(0.30)	(1.32)
underweight BMI	2.67***	2.26***	2.31***	2.15***	1.99**	2.01*
	(0.36)	(0.43)	(0.42)	(0.49)	(0.71)	(3.74)
health = poor	3.09***	7.19***	6.85***	8.52***	10.46***	9.85**
	(0.32)	(1.67)	(1.52)	(2.23)	(5.02)	(46.17)
health = fair	1.48***	2.76***	2.67***	3.19***	3.81***	3.61**
	(0.26)	(0.54)	(0.52)	(0.73)	(1.61)	(14.96)
health = very good	-1.52***	-2.39***	-2.34***	-2.69***	-3.08***	-2.95**
	(0.26)	(0.45)	(0.43)	(0.54)	(1.04)	(9.44)
health = excellent	-2.40***	-3.81***	-3.70***	-4.27***	-4.91***	-4.70**
	(0.39)	(0.67)	(0.62)	(0.79)	(1.64)	(14.83)
insurance type covariates			x	x	x	x
health care quality covariates				x	x	x
environmental covariates					x	x
state dummies						x
1st-stage coefficient on IV		0.87***	0.96***	0.80***	0.65***	0.67**
		(0.17)	(0.17)	(0.16)	(0.19)	(0.29)
F-statistic on the IV		27	32	25	11	5
number of person-years	44,697	44,697	44,697	44,697	44,697	44,697
number of people	22,206	22,206	22,206	22,206	22,206	22,206

Notes: The table reports average marginal effects expressed as percentage point changes in the one–year probability of death. All models include age, sex, age interacted with sex and with an indicator for over 90, and indicators for race, educational attainment, marital status, and living children. Columns (2)–(6) instrument for medical spending. Column (3) adds indicators for insurance coverages: Medigap, Medicaid, and Medicare Advantage. Column (4) adds HRR–level measures of CMS's hospital compare index, and per capita measures of the numbers of acute care hospital beds, primary care physicians, medical specialists, and hospital admissions for ambulatory care–sensitive conditions. Column (5) adds HRR–level measures of automobile mortality, homicide mortality, fine particulate matter, mean winter low temperature, mean summer high temperature, share urban, median income, high school graduation rate, and Census division dummies. Column (6) replaces the Census division dummies with state dummies. Standard errors are calculated using 1,000 bootstrap replications and clustered by hospital referral region. Asterisks indicate statistical significance at the 1% (***), 5% (**) and 10% (*) levels.

Columns (3)–(6) show results from repeating estimation of the IV survival function after incrementally adding additional covariates. These specifications address my concern that the marginal effect of medical spending in column (2) may be biased toward zero if my adaptation of the Finkelstein *et al.* (2016) decomposition does not fully purge latent health. For example, some of my estimated between–HRR "supply–side" variation in expenditures could be caused by people opting in or out of private insurance coverage at the time of their moves. These adjustments could introduce bias due to adverse or advantageous selection on latent health. Column (3) addresses this concern by adding separate indicators for whether each person was enrolled in a Medigap plan, a Medicare Advantage plan, or received Medicaid benefits. Adding these covariates reduces the estimated return to spending by about 10%.

A second concern is that the return to spending may be understated if higher–expenditure HRRs have a higher marginal impact on health per dollar spent, for example because they have higher–quality medical care providers. I test this hypothesis by adding controls for hospital quality in column (4). The additional covariates include HRR–specific measures of the number of hospital beds per capita, primary care physicians per capita, specialists per capita, discharges for ambulatory care–sensitive conditions among Medicare beneficiaries, and CMS's "Hospital Compare" index of hospital quality. [22] Indeed, I find that adding this set of proxy measures for hospital quality moderately increases my estimated return to spending to -0.64.

A related concern is that some regions may have higher medical expenditures due to environmental conditions that impair population health, attenuating my estimate

[22]This index is primarily derived from measures of the shares of patients who receive "timely and effective" care upon arrival at hospitals: `https://data.medicare.gov/data/archives/hospital-compare`. An example is the share of heart attack patients who are given aspirin. To measure average quality for each HRR, the shares are first averaged across all measures and hospitals in each HRR for each reporting period, and then averaged over all reporting periods in a year, and finally averaged over years.

for the return to spending. Column (5) addresses this concern by adding a set of proxy measures for local environmental conditions. These include automobile mortality, homicide mortality, average concentrations of fine particulate air pollution $PM_{2.5}$, average winter minimum temperature, average summer maximum temperature, the fraction of people living in urban areas, median income, the high school graduation rate, and dummy variables for the nine Census regions. Intuitively, I find that adding these controls further increases the estimated return to spending to -0.88. Finally, column (6) further tightens the controls for unobserved environmental conditions by replacing the Census region dummies with state dummies, forcing the identification to come from within–state variation in the HRR index (the average state has 6 HRRs). The resulting estimate for the return to spending is nearly the same at -0.82 despite the drop in statistical power. [23]

Taken together, columns (2) through (6) define a range of estimates for the return to medical spending from -0.88 to -0.42. I use column (3) as my main specification for calculating VSL. This model utilizes all of the microdata describing individual health and insurance coverages. In comparison, adding the additional HRR–level covariates and state dummies in columns (4), (5) and (6) presents a tradeoff. It mitigates potential bias from spending being correlated with other spatially varying determinants of health, but in the absence of bias it reduces identifying variation in the instrument and statistical precision. Indeed, the 95% confidence intervals on the estimated effects of medical spending in columns (4), (5), and (6) include the point estimate from column (3). Another reason why I feature the model in column (3) is that it generally yields the largest VSLs among the specifications in Table 2.2. Therefore, my choice to feature this specification works against the hypothesis that VSL measures based on seniors' medical care choices fall below VSL measures based

[23]The large standard errors in column (6) are driven by a few outliers.

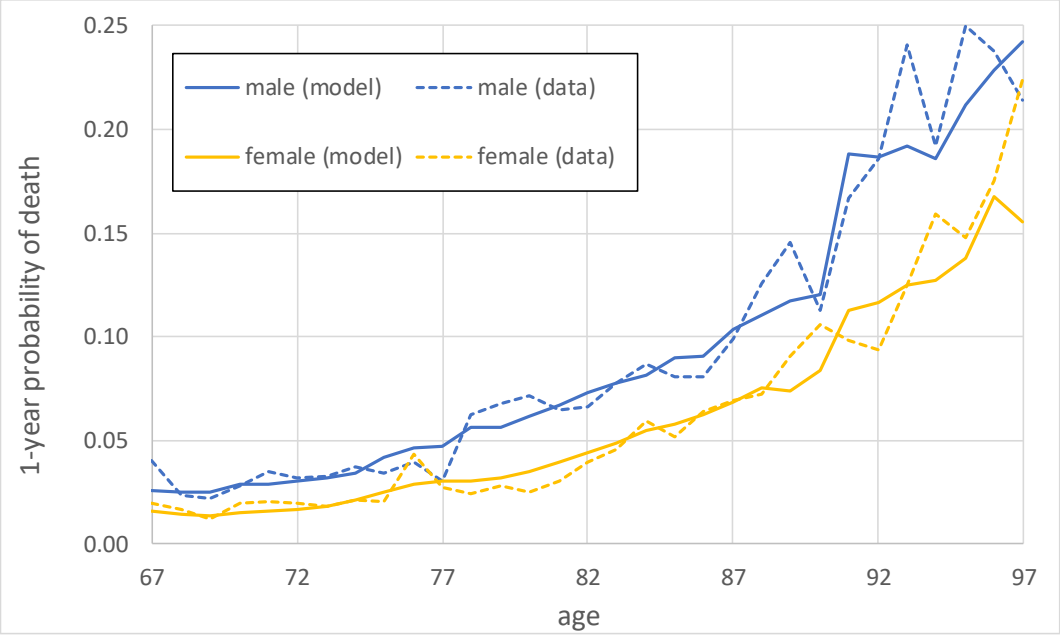
on workers' occupation choices. Section 2.6 presents my full range of VSL results from a comprehensive sensitivity analysis to alternative specifications including those in Table 2.2 and many others. As shown in Figure 2.5, this featured model falls near the middle of the distribution of VSL estimates for every given age.

2.5.3 Model Fit

The marginal effects of the health measures in Table 2.2 are intuitive and quantitatively important. For example, a standard deviation increase in the HCC morbidity index of observable chronic illnesses is associated with a 11 to 18 percentage point increase in the one-year probability of death across the IV models. Mortality is also conditionally higher among people with basic and instrumental limitations in activities of daily living, a history of smoking, a BMI that classifies them as being underweight, and a relatively poor subjective assessment of their own health. The reference category for self-reported health is "good". Moving from "good" to "poor" is associated with a 7 to 10 percentage point increase in the probability of death, whereas moving from "good" to "excellent" is associated with a reduction of 4 to 5 percentage points.

Figure 2.3 shows model fit by comparing its predictions for one–year mortality rates by integer age and sex to the data. Model predictions closely approximate mortality through age 87. Beyond age 87 the model continues to capture the upward trend in average mortality but it does not reproduce much of the idiosyncratic year–to–year variation around the trend. This improves my assessment of model fit because idiosyncratic deviations from the trend after age 90 are likely to reflect statistical imprecision caused by declining sample size.

Figure 2.3: Predicted and Actual One–Year Mortality Rates for Males and Females



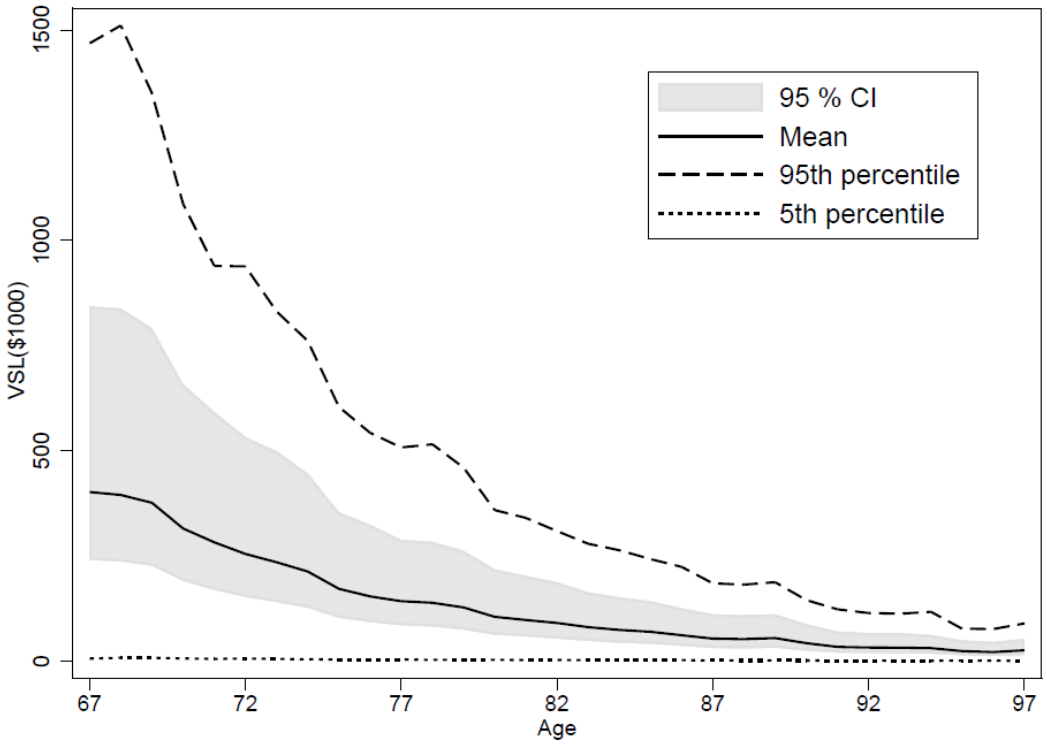
Note: The dashed lines show one–year mortality rates by age and sex in the data. The solid lines show model predictions from col (3) of Table 2.2.

2.5.4 Value of a Statistical Life

Figure 2.4 plots the VSL profile from age 67 to age 97 based on the model in column (3) of Table 2.2. This figure highlights several important features of my results. First, my VSL estimates for seniors are an order of magnitude below the prevailing wage–hedonic estimates for workers who are, on average, in their early 40s and in much better health. The solid line in the figure shows the mean VSL by age. At age 67 the mean VSL is about $402,000.

Second, the mean VSL declines with age in a near–monotonic fashion. This curvature is driven by the data. It is worth reiterating that my econometric model does not embed assumptions for the parametric form of utility or the rate of time preference. Rather, the shape of the curve reflects individuals' decisions about how much of their

Figure 2.4: VSL Estimates: Age 67 to Age 97



Note: See the text for details

own money to spend on medical care, given their current health, wealth, preferences, and beliefs about the return to spending. Analyzing the underlying components of the VSL equation (2.8) reveals that the downward trend in age results from dividing the individual coinsurance rate, which is relatively flat in age, by the return to medical spending, which increases in age. These trends are shown in Figure C.2. Intuitively, the decision not to spend more on one's health when the return to doing so is relatively high reveals that the VSL must be relatively low.

Third, my estimates for the mean VSL are reasonably precise. The shaded region in Figure 2.4 defines the bootstrapped 95% confidence interval on my estimate for the age–specific mean. It is asymmetric around the mean because the VSL is inversely

proportional to the estimated return to medical spending. Even at age 67 the upper bound of the confidence interval is below $1 million.

Finally, Figure 2.4 shows that there is substantial heterogeneity in the VSL conditional on age. The dotted and dashed lines denote the 5^{th} and 95^{th} percentiles in the distribution of my age–specific estimates. At age 67 the 95^{th} percentile is approximately $1.5 million. [24] However, the variation across people at a given age declines sharply with age.

Viewing these results through the lens of the life–cycle model can explain why the mean and variance of VSL both decline in age. As people get older their health tends to decline, as does the variance in remaining life expectancy. Negative health shocks reduce the expected future quantity and quality of life, creating a disincentive to invest in probabilistic life extension. While medical spending increases in age (Figure 2.2 panel F), it does not increase by enough to reduce the marginal return to further spending, yielding a decline in both the mean and variance of the VSL. In Section 2.7.1 I document similar patterns arising from heterogeneity in health conditional on age.

2.6 A Systematic Sensitivity Analysis

I take a systematic and comprehensive approach to testing the robustness of my main VSL estimates to modifying features of the research design. My approach is inspired by Leamer (1983), Banzhaf and Smith (2007), and Greenstone *et al.* (2013). First I define a set of potential modeling decisions along each dimension of my research design. Then I report VSL estimates derived from every possible combination of modeling decisions.

[24] Figure C.3 further illustrates the within–age heterogeneity in VSL by showing the distribution among 70–year–old people. Within that group, 93.74% of people have VSL values below $1 million, 5.52% have VSL values between $1 and $2 million, and 0.74% of people have values over $2 million.

2.6.1 Modifiable Features of the Research Design

Including or Excluding Workers

My main estimation sample excludes data for 5,764 person–years where the beneficiary was employed at the time of their MCBS interview. This exclusion improves internal validity by sharpening my focus on medical care as the relevant market for trading consumption against mortality risk, but it threatens external validity. I can investigate this threat by adding workers to the estimation sample. Doing so tends to increase the VSL modestly. For example, repeating estimation of the model from column (3) of Table 2.2 yields higher VSL measures among workers at each age from 67 to 87 with an average age–specific differential of 38%. [25] One explanation for this differential is that at any given age healthier and wealthier workers may be willing to pay more for statistical life extension and be less likely to retire.

Using MCBS or Claims–Based Data on Medical Expenditures

MCBS provides the most comprehensive data on total and OOP medical spending. Unfortunately MCBS does not collect these data during the first year of survey participation, which is why my main specification uses data from survey years 2 through 4. Alternatively, I can use all four years of survey data if I am willing to swap the MCBS spending measures for CMS's less comprehensive measures derived from the universe of claims processed under Medicare Parts A and B. Swapping the spending measures alters my sample size in countervailing ways. It expands the sample to include some observations from MCBS year 1 while simultaneously excluding people in each year who enrolled in Medicare Advantage plans (for whom claims–based spending data

[25] This age range accounts for 99% of workers in the MCBS sample. There is a near monotonic decline in the number of workers per integer age. I observe 28 people working at age 87, but never more than 15 people at older ages.

are unavailable). The net effect of these adjustments is to increase my sample size by 6%. More importantly, repeating the estimation for each sample reveals whether my findings are driven by how people respond to price adjustments by insurers that cannot be observed in claims data from fee-for-service Medicare.

Alternative Instrumental Variables for Medical Expenditures

My main specification for the instrument in equation (2.12) followed Finkelstein *et al.* (2016) in using dummies for 5-year age bins to absorb unobserved changes in health that could have occurred around each migrant's move year. As a robustness check, I incrementally relax the exclusion restriction on the IV to allow for additional forms of sorting on unobserved health. First I reconstruct the IV after replacing the dummies for 5-year age bins in (2.12) with dummies for integer age. Then I reconstruct the IV a second time using sex–by–integer–age dummies. As a third alternative, I reconstruct the IV after extending the sample to include people who never moved. [26] This increases statistical power and yields a more nationally representative sample of seniors. Finally, I construct a fourth alternative instrument from data on end–of–life spending based on evidence from Skinner *et al.* (2005) and Cutler *et al.* (2019) that a significant fraction of spatial variation in end–of–life spending is explained by variation in physician practice style. Specifically, I use average per–patient spending during the last 6 months of life reported by the Dartmouth Atlas at the HRR level.

Alternative Parametric Forms of the Survival Function

As an alternative to my featured Gompit specification for the survival function, I repeat the estimation using a Gompertz specification similar to the ones used by

[26] This matches the featured specification in Finkelstein *et al.* (2016). They exclude movers as a robustness check.

Chetty *et al.* (2016a) and Finkelstein *et al.* (2019a). The Gompertz form assumes that the log of the mortality rate is linear in covariates. This restriction reduces model fit based on the log–likelihood function value in my main specification from Table 2.2. Nevertheless, the Gompertz model has the benefit of simplicity and familiarity, having served as a common approach to modeling mortality for almost 200 years.

Alternative Covariates in the Survival Function

All of the IV specifications include the covariates shown in Table 2.2 and summarized in the footnote to the table. I repeat estimation as I incrementally add each of the five sets of augmented covariates. Corresponding to the columns in Table 2.2, these include: (2) no additional covariates, (3) insurance plan enrollment covariates, (4) medical care quality covariates, (5) environmental covariates, and (6) state dummies.

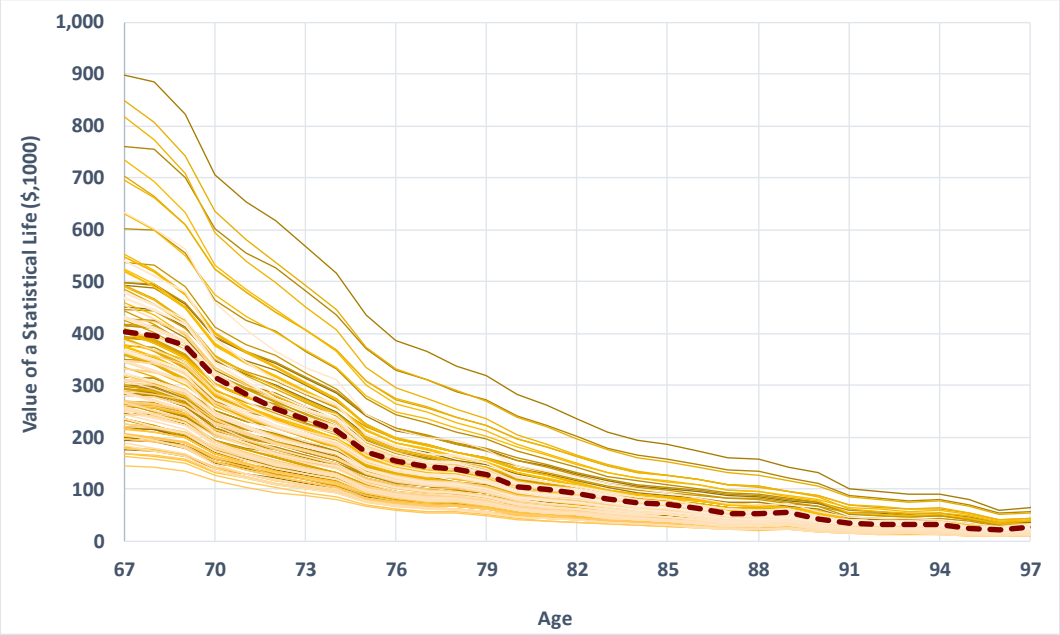
2.6.2 Results

Altogether I consider five different sets of covariates, two parametric forms for the survival function, five different instruments for medical spending, two ways of measuring medical spending, and models including and excluding workers. Considering all permutations of these modeling decisions yields 200 different models. I estimate each one and calculate the mean VSL by age.

Figure 2.5 shows results from the 200 models. The dashed line highlights my main specification from Figure 2.4. It sits near the middle of the range of estimates. Readers who disagree with my preferred modeling decisions can see how much those decisions matter relative to the alternatives outlined above.

At age 67 my preferred estimate is $402,000. The 5th and 95th percentiles in the distribution of models are $189,000 and $555,000, and the maximum is $899,000. These moments provide a partial measure of the model uncertainty in my VSL esti-

Figure 2.5: Sensitivity of VSL Estimates to Model Features



Notes: The figure shows the estimated mean VSL by age for 200 different specifications of the survival function. Each line corresponds to a different combination of modeling decisions as described in the main text. The large dashed line is my main specification from col (3) of Table 2.2.

mates. They have practical relevance because federal agencies use such moments to define benchmarks for sensitivity analysis, e.g. when using the social cost of carbon in policy evaluations (Greenstone *et al.* (2013)). Notably, every one of the 200 specifications yields mean VSL estimates that lie below $1 million at ages 67 and above, and substantially below the mean VSL estimates derived from occupation choices made by younger, healthier workers.

I use an internal meta–analysis (Banzhaf and Smith (2007)) to determine which factors cause the variation seen in Figure 2.5. Specifically, to summarize how modeling decisions affect the estimated VSL, I regress the log of mean VSL from each of the 200 models on indicators for model features. Relative to my main specification, VSL

estimates tend to increase if I add workers to the estimation sample (+24%), switch to using claims–based spending measures (+30%), or instrument using end–of–life spending (+39%). VSL estimates tend to decrease slightly if I switch to the Gompertz specification for mortality (-8%), fail to control for selection into insurance coverages (-11%), or include never–movers in the sample used to construct the instrument (-12%). As I previewed earlier, the VSL estimates decrease more substantially if I expand the covariate set to include HRR–level measures of medical care quality and environmental quality (-28% to -42%). The complete meta–regression results are reported in Table B.3.

Overall, I find that the level and curvature of the VSL–age profile is somewhat sensitive to modeling decisions, but two of its most important features are thoroughly robust. First, the VSL declines with age. Second, $1 million provides an upper bound on the VSL implied by seniors' medical expenditures.

2.7 Heterogeneity

2.7.1 Heterogeneity in the Return to Medical Spending by Health

Figure 2.6 summarizes how my estimates for the return to medical spending vary with subjective and objective measures of health. Each of the four panels reports the estimated average percentage point increase in one–year survival from a $1,000 increase in medical spending. Panels A and B stratify by self–reported measures of health. Panel (A) shows that conditional on age, the return to medical spending increases as self–assessed health declines. For example, at age 72 a $1,000 increase in spending reduces mortality by 0.8 percentage points for the average person who reports their health as "poor" compared to 0.08 for the average person who reports their health as "excellent". Panel (B) shows the same qualitative pattern such that,

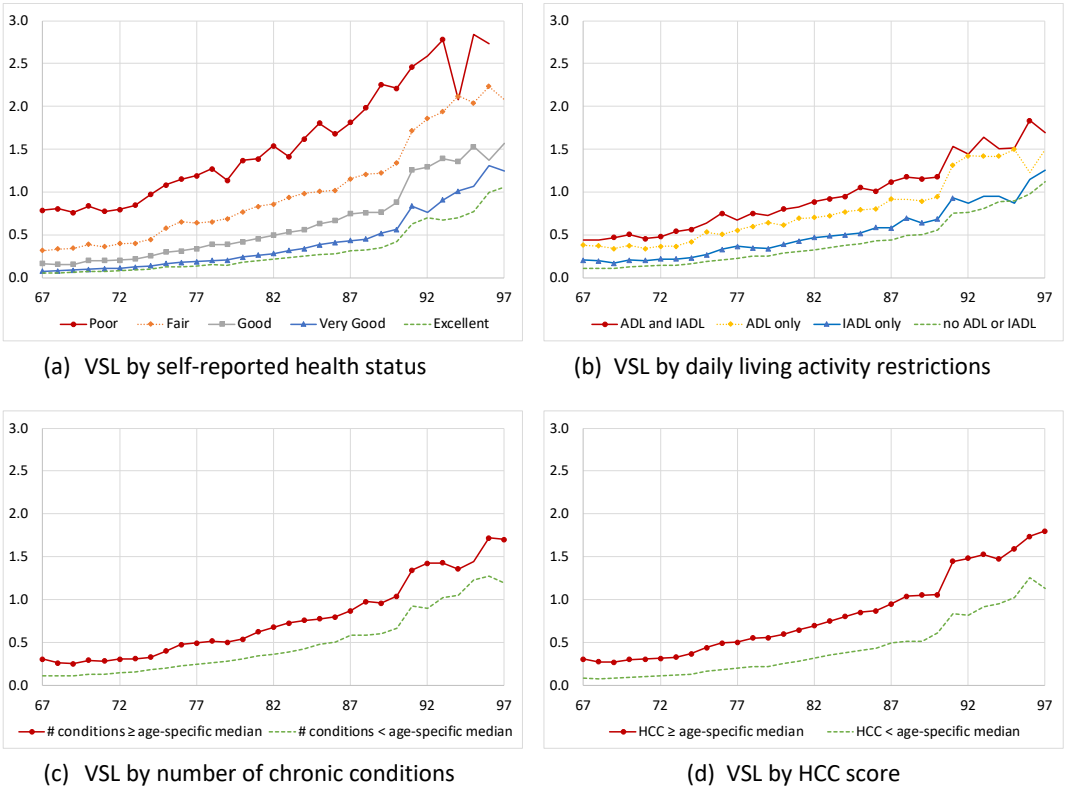
conditional on age, the return to further spending is lowest among those with no restrictions on activities of daily living, followed by those with restrictions on instrumental activities (e.g. managing money) but not basic activities (e.g. eating), followed by those with restrictions on basic but not instrumental activities, followed by those with restrictions on both basic and instrumental activities. Markers along the trend lines in each panel denote statistical significance of differences in returns between adjacent health categories. The presence of a marker indicates that the mean return at that integer age exceeds the mean return on the next lower trend line in at least 99% of bootstrap samples. Statistical precision declines with age due partly to the decline in age–specific sample sizes.

Panels (C) and (D) show that the basic pattern persists if I instead stratify by objective measures of health. In Panel (C) the age–specific return is always lower among people who have been diagnosed with fewer than the median number of chronic conditions for people of their age. In Panel (D) the age–specific return is always lower among people with HCC scores below the median for their age.

My estimates for the levels of returns and their variation with respect to health and age in Figure 2.6 essentially span the range of local average treatment effects that prior studies estimated from quasi–experimental sources of variation in expenditures within the Medicare population. For example, Huh and Reif (2017a), Clayton (2018)), Doyle *et al.* (2015a), Romley and Sood (2013), and Doyle (2011a) collectively suggest a range of marginal returns to $1,000 of medical spending from about 0.2 to 2, with relatively higher returns among sicker cohorts. Thus, even if the reader is unconvinced by my instrument for medical spending, substituting the reader's preferred estimate for the return to spending from prior literature into my VSL equation (2.8) would yield VSL measures of the same order of magnitude as the ones I report. [27]

[27] I summarize these prior results here, converting all dollars to 2010 by adjusting by the CPI.

Figure 2.6: Survival Gains from Marginal Increase of $1,000 in Medical Spending



(a) VSL by self-reported health status

(b) VSL by daily living activity restrictions

(c) VSL by number of chronic conditions

(d) VSL by HCC score

Notes: Each panel shows the average marginal effect (AME) of a $1,000 increase in medical spending on the probability of surviving to the end of the following year measured in percentage points on the vertical axis and calculated from the model shown in col (3) of Table 2.2. Markers along each trend line denote ages at which the AME exceeds the AME for the next lower trend line in at least 99% of 1,000 bootstrap samples with errors clustered by hospital referral region.

At the lower end, Huh and Reif (2017a) find that each additional $1,000 spending on prescription drugs due to the implementation of Medicare Part D reduced mortality by 0.15 percentage points. Among the younger, poorer Medicaid population, however, an additional $1,000 spending on prescription drugs led to a 2.1 percentage point reduction in mortality (Clayton (2018)). Doyle (2011a) uses a similar identification strategy as mine that leverages geographic variation in treatment intensity. Using Medicare beneficiaries who experience heart–related emergencies that lead to hospital admission through the emergency department while visiting Florida, his estimates imply that an additional $1,000 in spending (in 2010 dollars) reduced annual mortality of 0.2 percentage points. Doyle *et al.* (2015a) relies on quasi–random variation in treatment intensity due to ambulance referral patterns to evaluate the returns to spending among Medicare patients who are experiencing their first hospital admission while on Medicare and arrive at the hospital via ambulance with a subset of illnesses that have high admission rates. They estimate that an additional $1,000 in spending (in 2010 dollars) reduced annual mortality by about 1.9 percentage points. Romley and Sood (2013) re-

The stratification patterns in Figure 2.6 are not likely to be entirely causal. They may also reflect other socioeconomic factors that are correlated with health. For example, at any given age, healthier people are more likely to have a college degree and higher incomes. Attempting to disentangle these mechanisms is worthwhile but tangential to my main objective of estimating the VSL, so I leave it to future research. Regardless of the mixture of casual mechanisms underlying Figure 2.6, the observed negative relationship between health and the return to spending implies that my VSL estimates will tend to be lower for people in worse health. This occurs because the VSL is defined by the ratio of the coinsurance rate, which tends to increase in health, to the returns to survival from medical spending, which tends to decrease in health.

2.7.2 Heterogeneity in the VSL by Health

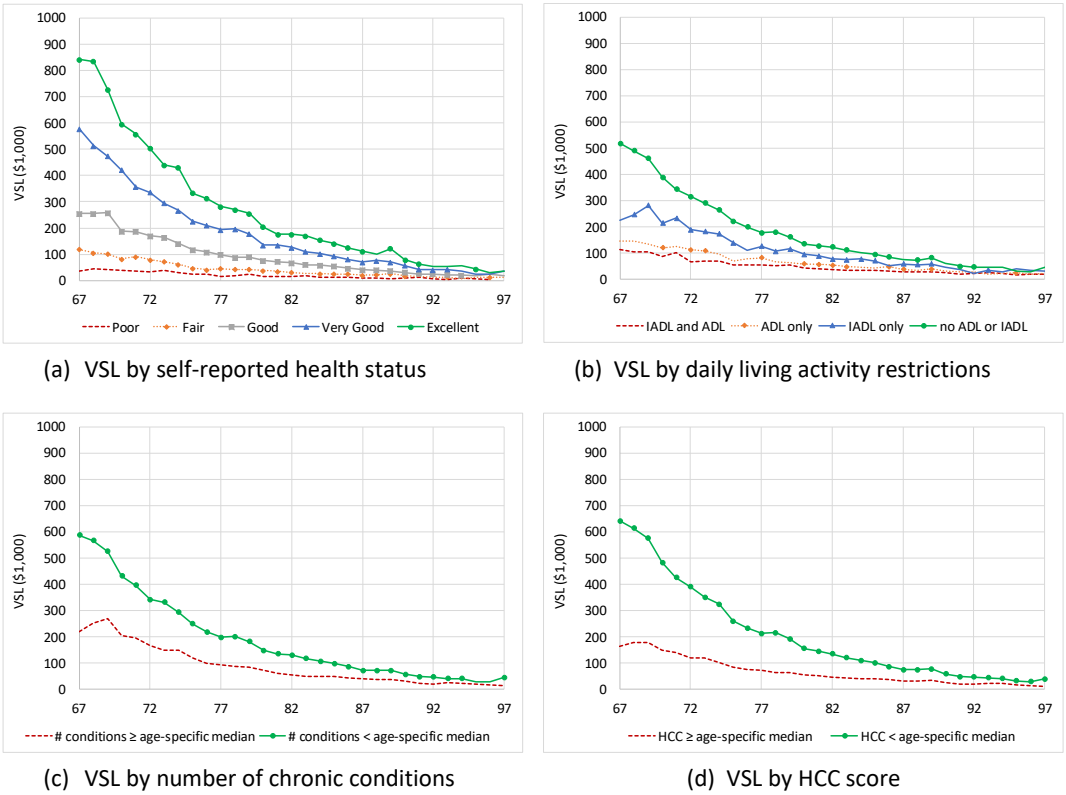
Figure 2.7 summarizes how VSL varies by subjective and objective health. I focus first on the extremes in Panel (A). At age 67, people who state they are in excellent health have an average VSL close to $850,000, more than 10 times the average VSL for people who state that they in poor health. People in poor health are more likely to have ever smoked and are diagnosed with more chronic illnesses such as kidney disease (29% compared to 6% of those in excellent health) and congestive heart failure (50% versus 11%). Conditional on age, across all categories the mean VSL increases monotonically with self–reported health. [28] The differences between health categories declines in age as relatively healthier groups experience sharper declines: VSL for people in excellent health in their early 90s is similar to VSL for people in

lies in instruments to additionally account for unobserved heterogeneity in hospital productivity and estimate that an additional $1,000 spending lowered 30–day mortality by 4.7 percentage points for Medicare patients admitted to the hospital due to pneumonia, 2.2 percentage points for congestive heart failure patients, and 1.8 percentage points for heart attack patients.

[28] In addition to the differences in the prevalence of chronic conditions, this within–age pattern is consistent with the fact that the survey question asks people to compare themselves against peers of the same age.

good health in their early 80s and to VSL for people in poor health in their late 60s. Panels (B), (C) and (D) show the same patterns emerge when the age–specific mean VSL is stratified by restrictions on activities of daily living, the number of diagnosed chronic medical conditions, or the CMS HCC score. The markers show that the differences between adjacent categories are almost always statistically significant at the 1% level from ages in the late sixties through the late eighties.

Figure 2.7: Heterogeneity in VSL by Age and Health



(a) VSL by self-reported health status

(b) VSL by daily living activity restrictions

(c) VSL by number of chronic conditions

(d) VSL by HCC score

Notes: Each panel shows the mean age–specific VSL in $1,000 (2010) dollars stratified by measures of health. The VSL is calculated from the model shown in col (3) of Table 2.2. Markers along each trend line denote ages at which the VSL exceeds the VSL for the next lower trend line in at least 99% of 1,000 bootstrap samples with errors clustered by hospital referral region.

2.7.3 Heterogeneity in the VSL by Behavioral and Socioeconomic Factors

The VSL is Lower for Smokers

Figure 2.8 summarizes how the VSL–age profile varies with behavioral and socioeconomic factors. Panel (A) highlights a large VSL gap between ever–smokers and never–smokers. At age 67 the VSL among never–smokers is approximately twice as large as among ever–smokers. This gap narrows with age as the differences in remaining life expectancy decline and is statistically indistinguishable from zero beyond age 92. These trends are consistent with the fact that smoking habits are associated with a 10–year reduction in life expectancy (Jha *et al.* (2013)) and lower quality of life. For example, COPD is twice as common among ever–smokers and lung cancer is six times as common among ever–smokers. These and other chronic illnesses may significantly reduce their expected remaining quantity and quality of life, providing an incentive to shift consumption from medical care to other forms of private consumption. [29]

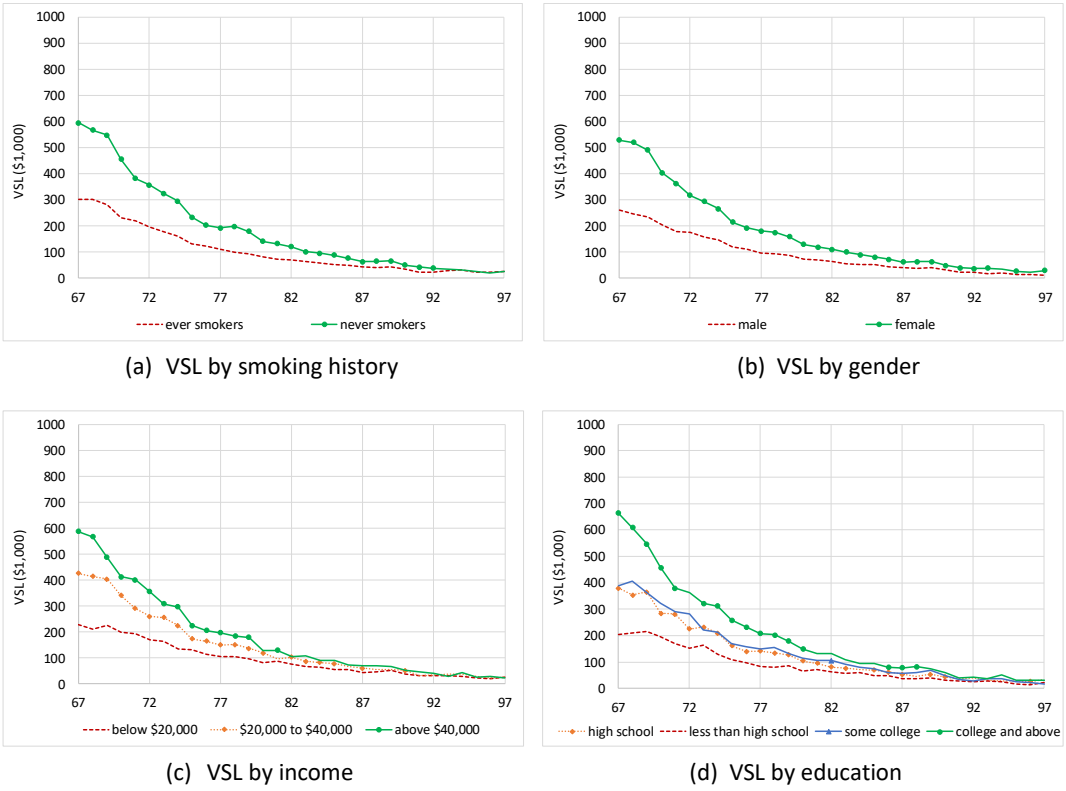
The VSL is Higher for Females

Panel (B) in Figure 2.8 shows a VSL gender gap. At age 67, the VSL is approximately twice as high for females, consistent with the higher female life expectancy. The differential declines as the difference in remaining life expectancy falls with age. This evidence validates the out–of–sample predictions made by Aldy and Smyth (2014) and Murphy and Topel (2006) based on life cycle models that incorporate expected longevity.

My evidence on the VSL–gender gap is novel. Hedonic wage studies rarely stratify

[29] My evidence of the VSL smoking gap late in life diverges from findings reported in wage–hedonic studies. For example, Viscusi and Hersch (2008) augmented a hedonic wage model with data on smoking status and found virtually no difference in the VSL estimated for workers who smoked compared to those who did not. The divergence in results could be explained by the fact that we study people at older ages at which smoking-related morbidities are more likely to have manifested.

Figure 2.8: Heterogeneity in VSL by Smoking, Gender, Income, and Education



(a) VSL by smoking history

(b) VSL by gender

(c) VSL by income

(d) VSL by education

Notes: Each panel shows the mean age–specific VSL in $1,000 (2010) dollars stratified by measures of health. The VSL is calculated from the model shown in col (3) of Table 2.2. Markers along each trend line denote ages at which the VSL exceeds the VSL for the next lower trend line in at least 99% of 1,000 bootstrap samples with errors clustered by hospital referral region.

VSL estimates by gender due to data limitations. Leeth and Ruser (2003a) show that females are less likely to work in high–risk occupations and, conditional on occupation, females have substantially lower fatality rates. According to the Census of Fatal Occupational Injuries, males account for more than 90% of all accidental deaths on the job. This makes it difficult to calculate precise occupation–by–gender fatality rates, motivating researchers to focus exclusively on males (e.g. Costa and Kahn (2004), Kneisner *et al.* (2012)). An exception is Deleire *et al.* (2013) who report

mixed evidence on the VSL gender gap for workers age 18–60 based on combining large worker samples from the Current Population Survey with non–gender–specific data on fatality risk. In contrast, data in my study capture the important differences in gender–specific health and fatality risk.

The VSL is Increasing in Income and Education

The hedonic wage literature suggests that the VSL is increasing in worker income with a cross–sectional elasticity over one (e.g. Cropper *et al.* (2011a)), Evans and Schaur (2010), Viscusi (2010), Aldy and Smyth (2014)). I excluded income from the survival model because we expect income to affect mortality risk indirectly through the covariates describing health and/or medical expenditures. Nevertheless, I can stratify my VSL estimates based on MCBS respondents' income bins to bound the cross–sectional income elasticity.

Panel (C) in Figure 2.8 shows the expected relationship between VSL and income, with the stratification between bins declining in age. This validates the prediction from Aldy and Smyth (2014) that the VSL–income elasticity will decline late in life as the scope for differences in remaining life expectancy declines. Focusing on the minimum difference in income between people in the top and bottom bins defines upper bounds on the income elasticity of 1.28 at age 67, 0.93 at age 77, and 0.77 at age 87. [30]

Because income is increasing with education, it is unsurprising to see the VSL increasing in education as well in Panel (D). Nonetheless, the magnitudes are striking. At age 67 the mean VSL among people with a college degree is more than three times as large as for people who did not finish high school and more than 50% larger than

[30]For example, if I assume that the difference in income between people in the "above $40,000" and "below $20,000" bins is approximately $20,000, then doubling income at age 67 is associated with multiplying VSL by 2.56, yielding an upper bound on the elasticity of 1.28.

for those who did not finish college. Meanwhile, I see virtually no difference between people who finished high school and did not attend college and people who attended some college but did not complete a degree.

2.8 Assessing the Influence of Revealed Preference Assumptions

So far I have followed the convention in the VSL literature and assumed that people make informed tradeoffs between consumption and mortality risk. In the context of this paper, the assumption is that Medicare beneficiaries accurately assess their OOP costs of reducing their mortality risk, perhaps with the assistance of family members and physicians. In reality, this assumption is unlikely to always hold true because some people do not fully understand their treatment options and billing procedures, even with help from family and physicians. I capitalize on the fact that the MCBS includes ancillary questions that allow me to assess how the VSLs implied by medical spending vary across people based on their knowledge and decision autonomy.

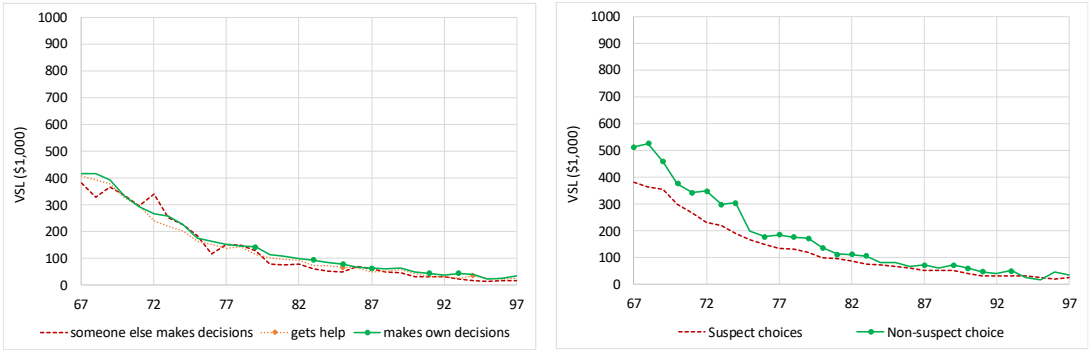
2.8.1 The VSL is Insensitive to Who Makes Health Care Decisions

The MCBS asks people whether they usually make health insurance decisions on their own, receive help making decisions and who helps them, or rely on others to make decisions for them. In cases of Alzheimer's disease or other impairments, the proxy who makes health insurance decisions also responds to the MCBS. For these patients my VSL measures are best interpreted as a reflection of family–level valuations because the proxy decision–makers are almost always family members.

Panel (A) of Figure 2.9 stratifies my VSL estimates based on who usually makes health insurance decisions. There is little difference between the 67.6% of beneficiaries who usually make their own decisions, the 27.6% who get help, and the 4.8% who rely on someone else to make decisions for them. As a result, narrowing the focus to the

subset of people who make their own medical care decisions yields virtually the same VSL measures as my featured specification (Figure 2.4). Thus, consistent with the simplifying assumptions of the theoretical model in this study, the distinction between individual– and family–level valuations of mortality risk reductions for beneficiaries does not appear to be quantitatively important for my estimates.

Figure 2.9: Heterogeneity in VSL by Decision Process and Knowledge



(a) VSL by who makes health care decisions

(b) VSL by knowledge and agency

Note: Each panel shows the mean age–specific VSL in $1,000 (2010) dollars. The VSL is calculated from the model shown in col (3) of Table 2.2. Panel (A) stratifies the results based on who makes medical care decisions for the beneficiary. Panel (B) stratifies the results based on whether I observe evidence causing us to suspect that the beneficiary may not be fully informed. See the text for definitions. Markers along each trend line denote ages at which the VSL exceeds the VSL for the next lower trend line in at least 99% of 1,000 bootstrap samples with errors clustered by hospital referral region.

2.8.2 The VSL Increases Slightly with Health Care Knowledge

The MCBS also allows me to evaluate potential effects of some people not being fully informed about their costs and benefits of medical care. For information frictions to attenuate my VSL measures, the frictions would have to increase the marginal return to medical spending. This could occur, for example, if "behavioral hazard" causes people to systematically underuse beneficial treatments (Baicker *et al.*

(2015)). However, I note that my estimated returns to spending span the local average treatment effects from prior studies of contexts in which undertreatment due to behavioral hazard seems unlikely, e.g. inpatient spending on heart–attack patients admitted through the emergency room (Doyle (2011a)).

MCBS data do not facilitate investigation into specific information frictions, but they do contain several signals about whether beneficiaries are likely to be more or less informed. I use these signals to classify decisions about annual medical care expenditures as "suspect" or "non–suspect" for the purpose of revealing preferences, borrowing terminology from Bernheim and Rangel (2009). I classify decisions as "non–suspect" if I have no reason to suspect that the decision–maker is less than fully informed. I classify decisions as "suspect" if I suspect that conventional revealed preference assumptions may not strictly hold in the data because one or more of the following statements about the beneficiary is true: (1) does not make their own health insurance decisions, (2) has assistance managing money, (3) does not realize that OOP costs vary across Medicare Part D prescription drug plans, (4) suffers from dementia and/or depression, or (5) does not think they know most of what they need to know about Medicare. [31] These criteria lead me to classify 82% of all person–years of expenditure decisions as suspect. Importantly, this classification does not mean that revealed preference logic necessarily fails for these observations, only that I have reason to suspect that it might.

Panel (B) of Figure 2.9 shows that non–suspect choices are associated with slightly higher VSL measures even conditional on age. This is consistent with the hypothesis that less informed beneficiaries have higher marginal returns to medical spending,

[31] The Part D knowledge question asks respondents whether it is true or false that "Your out–of–pocket costs are the same in all Medicare prescription drug plans." The correct answer is false. The Medicare general knowledge question asks people to report "How much do you think you know about the Medicare program? Do you know... [just about everything/most/some/a little/almost none] of what you need to know about the Medicare program?"

e.g. due to behavioral hazard. However, it can also be explained by the fact that the people making non–suspect choices are slightly healthier (e.g. 1.8 fewer chronic conditions, 36% of a standard deviation reduction in the HCC score). In any case, the differences between the suspect and non–suspect VSL measures are small, suggesting that heterogeneity in information frictions is unlikely to substantially attenuate my main VSL estimates.

2.8.3 Imperfect Physician Agency Would Bias the VSL Estimates Upwards

The presence of physicians differentiates medical decisions from the occupation choices that have traditionally been used to infer VSL. Ideally, physicians would help patients understand their options, strengthening the credibility of the revealed preference assumptions. However, a potential concern is that Medicare's fee–for–service payment methods may incentivize some physicians to recommend more treatment than under perfect agency. While I cannot directly evaluate the importance of this concern for my results, I expect it to work against my finding that the VSL is low compared to wage–hedonic studies. All else constant, higher medical spending due to breakdowns in physician agency driven by fee–for–service payment would lower my estimated returns to marginal medical spending and subsequently inflate my VSL estimates.

2.8.4 Deriving an Upper Bound on the VSL

As a final step in evaluating the scope for information frictions to attenuate my estimates, I derive upper bounds on the VSL by making an extreme assumption about beliefs. I assume that people ignore insurance when they make spending decisions and instead falsely believe that they will pay the entirety of their medical bills out of pocket. This increases my estimate for the mean VSL to approximately $1.3 million

for those in their late 60's and $100,000 for those in their late 90's. [32] These values approximately double if I make the additional ad hoc adjustment of dividing my estimated returns to spending by a constant that forces my model to match the lower bound point estimate on average returns to spending from recent quasi-experimental studies of the Medicare population (Huh and Reif (2017a), Clayton (2018)), Doyle *et al.* (2015a), Romley and Sood (2013), and Doyle (2011a)). Even under these extreme assumptions, the resulting upper bound on VSL for ages in the high sixties is approximately one–quarter of the standard wage regression estimates derived from younger workers' occupation choices.

2.9 The Value of a Statistical Life Year

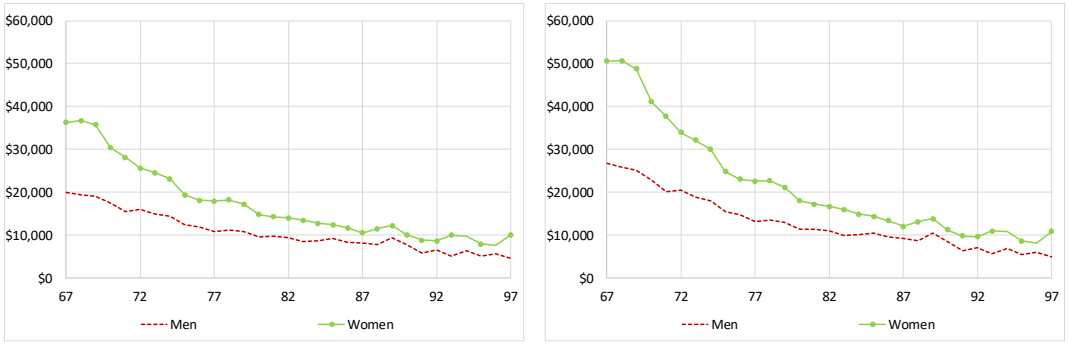
Policy analyses often rely on annuitized VSL estimates to monetize the benefits of policies that modify life expectancy among older populations. A conventional but arbitrary value for one statistical life year (VSLY) is $100,000. Revealed preference evidence from workers suggests that the VSLY is an inverse U–shaped function of age (Aldy and Viscusi (2008a)) [33] . I add to this literature by using my VSL estimates to provide direct evidence on the VSLY for seniors.

Figure 2.10 shows the value of a statistical life year by age and gender, and Appendix Table B.4 reports the values underlying the figure. I calculate these measures by combining my age–by–gender–specific VSL measures with age–by–gender–specific

[32] These values are also analogous to what Hall and Jones (2007a) call the "social value of life" because they can be reinterpreted as extending revealed preference logic to incorporate taxpayer expenditures on Medicare. My $1.3 million estimate for those age 67–69 is very similar to the measures they calibrate from macro data on medical spending. My use of microdata on health, demographic and socioeconomic characteristics introduces more curvature so that my estimates decline more steeply with age. Mechanically, I calculate this simply by replacing each person's observed coinsurance rate with a coinsurance rate of one.

[33] Despite the evidence in Aldy and Viscusi (2008a) the US Office of Management and Budget (U.S. Office of Management and Budget (2003)) gives the following instructions to economists tasked with cost–benefit analysis of federal programs who choose to use VSLY measures: "you should adopt a larger VSLY estimate for senior citizens because senior citizens face larger overall health risks from all causes and they may have accumulated savings to spend on their health and safety".

Figure 2.10: Value of a Statistical Life Year by Age and Gender



(a) 3% discount rate (b) 7% discount rate

Note: Each panel shows the mean age–by–gender–specific value per statistical life year in $1,000 (2010) dollars. The VSL is calculated from the model shown in col (3) of Table 2.2. Panel (A) uses a discount rate of 3% and Panel (B) uses a discount rate of 7%. Markers along each trend line denote ages at which the underlying VSL estimate exceeds the VSL estimate for the next lower trend line in at least 99% of 1,000 bootstrap samples with errors clustered by hospital referral region.

information on expected life years remaining from the US life tables. Panels (A) and (B) of the figure report the VSLY for the US Office and Management and Budget's recommended range of discount rates for valuing mortality reductions: 3% to 7% (U.S. Office of Management and Budget (2003)). Even at the upper–bound discount rate, my VSLY estimates at age 67 are well below the commonly used benchmark values. The differences by gender and the decline with age are consistent with a strong relationship between the health stock and the value of statistical life extension.

With a 7% discount rate my estimates imply a mean VSLY of $51,000 for women at age 67. Figure C.4 shows the sensitivity of my VSLY estimates to the 200 alternative specifications for the survival function discussed in Section 2.6. The 95th percentile in the distribution of models for females age 67 is $71,000 and the maximum is $105,000. In fact, this is the only specification that ever yields an average VSLY over $100,000 for men or women of any age beyond 67.

2.10 Conclusion

I linked US seniors' Medicare records to survey data on their health and medical spending, estimated their value of a statistical life, and analyzed heterogeneity in VSL measures by age, health, income, demographics, knowledge and agency. My results imply that the conventional wage–hedonic estimates for the VSL overstate by an order of magnitude what seniors are willing to pay for medical care that marginally increases their own survival probabilities. Likewise, under standard assumptions for discount rates I find values per statistical life year (VSLY) that are less than half the size of values that are commonly used to assess the benefits of technologies, policies, and regulations that affect the health of US seniors. I also find that the VSL increases in health, income and remaining life expectancy. These findings have potential to improve the efficiency and equity of a wide range of government activities that affect seniors' health and longevity.

My finding that the VSL for seniors increases sharply with their health and life expectancy implies that the VSL should not be treated as a statistic that is invariant to many of the policies that it is used to evaluate. Simply multiplying VSL by the number of premature deaths avoided by a policy will bias the benefit measure toward zero for policies that also reduce morbidity. Such life–saving policies may trigger a virtuous cycle in which deaths are averted directly, but health is also improved, the value of life increases, and people make greater subsequent investments in their health. Extending my analysis to directly model how this dynamic complementarity works through the VSL to modify the benefits of regulations that simultaneously affect morbidity and mortality is an important task for future research.

Chapter 3

A UNIFIED FRAMEWORK FOR VALUING HEALTH AND MORTALITY

3.1 Introduction

One of the primary goals of public policy is to improve human health and longevity. Federal agencies pursue this goal by regulating markets to target inputs to health production such as pollution, health care, and food and drug safety. People over age 65 are the main beneficiaries of many such policies because their advanced age makes them more vulnerable to pollution and less resilient to disease compared to younger adults. In addition to being the primary beneficiaries of US Medicare programs and regulations on air pollution, senior citizens are also the wealthiest and fastest growing age group, projected to constitute a quarter of the US population by 2050. This population aging makes it especially important to consider how seniors will be affected by policies targeting health and health care. A key challenge for evaluating the efficiency and equity of these policies is to understand the economic implications of *complementarity* between the quantity and quality of life. Put simply, policies that improve expected future health may also improve longevity and the benefits of surviving to future periods; and policies that increase the probability of surviving to future periods may also improve quality of life and the value of future health improvements.

The standard approach to evaluating public policies that improve both health and survival is to estimate the benefits of reducing morbidity and mortality independently and then add them together. This independence assumption may lead to underestimation or overestimation of benefits. For example, Murphy and Topel (2006) suggests

the independence assumption may lead to underestimation through *dynamic complementarity* because policies that improve longevity may increase the willingness to pay (WTP) for future quality of life improvements, as individuals expect to enjoy better health for a longer lifetime. On the other hand, Gerking *et al.* (2014) suggests that the independence assumption may lead to overestimation through *static complementarity* because estimates of the WTP to reduce unconditional mortality risk may embed some of the WTP for concomitant reductions in morbidity risk and vice versa. The challenge is to develop an integrated framework for evaluating the distributional welfare implications of policies targeting morbidity, mortality, or both, that accounts for static and dynamic complementarity between the quantity and quality of life.

In this paper, I combine the ideas of Grossman (1972) and Murphy and Topel (2006) into a unified framework for evaluating the efficiency and equity of policies affecting the health and longevity for seniors. I develop and estimate a life-cycle model of consumption and medical spending for seniors who face uncertainty about their future health and longevity and can modify their life expectancies by purchasing health care. I estimate the model using Medicare administrative records linked to the Medicare Current Beneficiary Survey (MCBS). Then I use the results to assess how much consumption seniors are willing to forgo in exchange for a marginal reduction in mortality risks and how much they are willing to pay to reduce the risk of being diagnosed with diseases. Finally, I use the model to calculate the monetary benefits of the reductions in morbidity and mortality during the 2000's that the US Environmental Protection Agency attributes to Clean Air Act.

My life-cycle model tracks people's consumption and health from age 66 to age 95, in the spirit of De Nardi *et al.* (2010) and Hall and Jones (2007b). Individuals who differ in their asset holdings, health and permanent income choose how much to spend each period on consumption, savings and health care, while facing uncertainty about

their future health and survival. Importantly, the utility is a CRRA specification that is sufficiently flexible to allow me to identify whether consumption and health are complements or substitutes. The cost of health care reflects the structure of Medicare payment plans and the evolution of health depends on age, medical spending and idiosyncratic health shocks. The realized health stock at the end of each periods affects the probability of surviving to the next period.

There are two key challenges to identifying the health production function: (i) the health stock is not directly observable, and (ii) latent features of the health stock may be correlated with medical spending and future health. Taken together, these two challenges imply that no single proxy measure of health can fully capture the dynamics of health, and the resulting measurement errors pose a threat to identification. I overcome these challenges by adapting insights from the literature estimating production functions for human capital. This threat to identification parallels the main challenge with identifying production functions for human capital (Cunha *et al.* (2010); Cunha and Heckman (2008) ; Hai and Heckman (2015)). I adopt the econometric approach from that literature, drawing especially on Cunha and Heckman (2008), to estimate a dynamic latent factor model of health. This data-driven approach requires me to observe multiple proxy measures for the evolution of each individual's health. I meet this requirement by leveraging a rich set of subjective and objective measures for health reported in MCBS and Medicare records. Specifically, I use the linked administrative-survey data to estimate the measurement systems that identify the distribution of latent health factors that evolve with age. [1] The latent factor model overcomes the endogeneity problem created by measurement errors by imposing covariance restrictions that effectively require the measurement errors as-

[1]Related literature on estimating the dynamics of health using multiple health indicators includes Heiss *et al.* (2009) and Halliday (2008). For a detailed review, see Lange and McKee (2012)

sociated with different proxy measures for health to be independent of one another for all ages. Intuitively, I assume that patients and doctors make different types of errors when they imperfectly characterize the patients' health.

After estimating the health production function, I use a simulated method of moments to estimate the utility parameters. I employ moment conditions based on the mean age profiles of net assets, medical expenditure, survival probability, and health. To accomplish this, I supplement the MCBS data with information on assets and income from the Health and Retirement Study (HRS). Although the model parameters are jointly identified, each parameter is intuitively linked to key sources of variation in the data that are reflected in particular moment conditions. The evolution of wealth identifies parameters describing the rate of time preference and risk aversion; the evolution of medical expenditures with age identifies the elasticity of substitution between consumption and health; and the marginal rate of substitution between consumption and health is identified from the gradient of health status and medical expenditure with respect to age.

The model estimates yield several insights. First, combining my estimates for the marginal effect of medical expenditures on health and the effect of health on survival implies that, on average, a $1,000 increase in medical expenditures increases the survival probability by 0.3 percentage points, consistent with the range of estimates in prior studies (Doyle (2011b); Doyle *et al.* (2015b); Huh and Reif (2017b)). Second, the marginal cost of reducing mortality risk decreases with age, and is very low for survival probability. This is consistent with the findings in Fonseca *et al.* (2013) who finds health production exhibits high productivity at low levels of survival but diminishes with the level of spending. Third, consumption and health are complements so that the marginal utility of consumption is increasing in health.

My VSL-age profile is consistent with the hypothesis that the VSL for seniors

declines with age (Viscusi and Aldy (2007); Cameron and DeShazo (2013); Aldy and Smyth (2014)). I calibrate the size of the change in risk to generate a VSL of $4.8 million (year 2010 $) at age 67 to account for the age discounting for seniors suggested by Viscusi and Aldy (2007) . My estimates then imply that the VSL gradually declines as people age, with the sharpest decline observed in the early 70s. This decline is driven by the evolution of health and life expectancy. As people age they become more susceptible to adverse health shocks, which cause them to increase health spending. However, their inability to completely reverse the shocks reduces their health stock and the marginal utility of consumption. Therefore VSL measures decline with illness and age. For example, at age 67, the average VSL for individuals in the top quintile of health is four times the average VSL for individuals in the bottom quintile.

My estimates also yield insights about the WTP for disease prevention, which can be aggregated into measures of the value of statistical illness avoided (VSI). Intuitively, I find that WTP increases with the extent to which a disease is expected to reduce the future health stock. This WTP declines with age for the same reasons that the VSL declines with age. To illustrate the quantitative implications, I use my estimates to calculate the VSI for two common diseases: Chronic Obstructive Pulmonary Disease (COPD) and non-fatal Acute Myocardial Infarction (AMI). At age 67, the value of avoiding a new diagnosis of COPD is $650,000, which then gradually declined to around $200,000 at age 94. In comparison, the VSI for AMI, commonly known as a heart attack, is around $100,000 at age 67 and $40,000 at age 94. COPD has a much larger VSI because my estimates imply that it has a greater impact on the health stock. Using the measurement system, I find that COPD has a much larger impact on future expected health compared to a nonfatal heart attack.

To assess the importance of static complementarity between the quantity and quality of life, I use my model to decompose the WTP to avoid the increases in

morbidity and mortality effects by calculating the WTP for morbidity conditions on survival probabilities. The decomposition reveals that, at age 67, approximately 20% of the VSI for COPD is due to its negative effect on life expectancy, whereas 80% is due to a lower quality of remaining expected life. My findings also provide empirical support for the dynamic complementary hypothesis in Murphy and Topel (2006). For instance, I show that a 10% reduction in all-cause mortality starting at age 66 increases VSL by $125,000 at age 75 and VSI for COPD by $12,000.

I investigate the implications of static and dynamic complementarity to demonstrate how the model can be used to inform public policy by using my model to monetize the health benefits of the Clean Air Act (CAA) using the US Environmental Protection Agency's estimates for the casual reductions in mortality among people over age 65, during the 2000's as well as their reduction in incidences of chronic illnesses. I determine that the CAA created $0.72 trillion in annual benefits, generating a benefit-cost ratio of 12:1 in 2010. These benefits reflect dynamic complementarity. In particular, I estimate that the reduction in mortality attributed to CAA increased the average VSL by $180,000 (or 8%) at age 67. Importantly, my estimates for all of these interrelated improvements to health and survival are internally consistent and derived from the same underlying model. Looking ahead to future research, this framework offers the potential to yield new insights about evaluate the distributional welfare implications of proposed changes to the CAA and other policies targeting morbidity, mortality or both.

Overall, this study advances the literature on evaluating the benefits of reducing mortality and morbidity risk reduction in several ways. First, prior studies using life-cycle models have ignored the evolution of health and its impact on WTP (Rosen (1988b); Moore and Viscusi (1990); Johansson (1996); Murphy and Topel (2006)). I extend this literature by incorporating medical expenditures as a choice variable

and I show that doing so allows me to match the life-cycle profiles of consumption, health, and medical spending. Second, prior studies that have modeled preferences over health have assumed additive separability in consumption and health, which implies a relatively flat consumption trajectory as individuals smooth consumption over the life-cycle (Hall and Jones (2007b); Murphy and Topel (2006)). I find that allowing for non-separability in consumption and health is important for my quantitative results. Third, this paper is the first life-cycle study to use the dynamic latent factor model to address the endogeneity problems with identifying a Grossman-style health production function. Finally, by modelling the twin uncertainties in health and survival, this study accounts for complementarities in risk in valuing disease prevention (Bauer *et al.* (2018); Cameron and DeShazo (2013)).

3.2 Related Literature

This paper builds on the extensive literature on valuing health. Estimates for WTP to reduce mortality and morbidity shape the benefits and costs in cost-benefit analyses of regulations affecting human health. I extend this literature by developing the first unified dynamic model for consistently estimating the WTP for changes in morbidity and mortality risk while recognizing the potential for complementarity between them. By explicitly focusing on seniors, my study also serves to fill an important gap in much of the prior literature on valuing human health, which has focused on younger populations. [2]

Most of the existing evidence on the value of reducing mortality risk has been derived from hedonic wage models. [3] Relatively few studies have used life-cycle models

[2]Gerking *et al.* (2014) provides a static framework to evaluate parents' WTP for mortality and morbidity risks faced by themselves and their children. Their results are based on stated preference methods using field data for cancer and leukemia.

[3]An in-depth review of VSL studies is provided in Cropper *et al.* (2011b). For meta-analyses of VSL literature, see Mrozek and Taylor (2002); Viscusi and Aldy (2003) and Kochi *et al.* (2006)

to estimate VSL-age profiles [Rosen (1988b); Moore and Viscusi (1990); Johansson (1996); Hall and Jones (2007b); Aldy and Smyth (2014)]. Among these studies, the two that are most closely related to my work are Murphy and Topel (2006) and Hall and Jones (2007b), both of which calibrate life-cycle models and use them to evaluate the benefits of increasing life expectancy. I extend these studies by using microdata to directly identify the parameters of utility and health production functions while allowing the utility to be non-separable in consumption and health.

The framework developed in this study also relates to the growing literature on life-cycle models with health uncertainty. Several studies model medical expenditures as an exogenous shock to the budget constraint (Palumbo (1999); De Nardi *et al.* (2010); French and Jones (2011); Pashchenko and Porapakkarm (2017); De Nardi *et al.* (2017)). I extend this literature by modelling medical spending as a choice variable, which helps me to identify marginal rates of substitution between consumption, health, and mortality risk. This feature allows my model to capture the essence of health capital evolution following Grossman (1972). This feature also allows health risks to be endogenous, recognizing that seniors may choose to prioritize their quality of life over quantity by avoiding expensive medical treatments that yield marginal increments in expected longevity and/or have undesirable effects. A few studies have explicitly model medical expenditure as endogenous choices (Fonseca *et al.* (2020); Khwaja (2010); Ozkan (2014)). I extend this literature by treating health as a latent variable that can be mapped onto several proxy measures, rather than focusing on a particular proxy measure of health.

This study also relates to the literature on heterogeneity in VSL across subpopulations. While there is debate over the shape of the VSL-age profile, many studies have suggested an inverted-U shape (Murphy and Topel (2006); Aldy and

Viscusi (2008b); Cameron and DeShazo (2013); Aldy and Smyth (2014)). [4] Other studies have reported evidence of higher VSLs for women compared to men (Leeth and Ruser (2003b); Viscusi (2004); Murphy and Topel (2006); Aldy and Smyth (2014)), and higher VSLs for healthier people relative to sicker people (Ketcham *et al.* (2020)). [5] Prior studies have also reported a positive effect of income on VSL, with estimates ranging from 0.5-0.7 at the lower end (Mrozek and Taylor (2002); Viscusi and Aldy (2003); Viscusi and Masterman (2017)), to over 1 at the higher end (Costa and Kahn (2004); Kniesner *et al.* (2010)). I add to this evidence by documenting how VSL varies with respect to age, health, and income, with the life-cycle model helping to discipline the sources of this variation.

Compared to the vast empirical literature on VSL, there has been relatively little work on estimating the willingness to pay for reduced morbidity. It is important to understand the trade-offs people make between health and consumption because many public policies target health. Indeed, the U.S EPA's highest priority for long-term research is to calculate the monetary benefits of reducing morbidity risks (US EPA (2005)). Murphy and Topel (2006) also speculate that improvements in quality of life due to medical technologies could have greater value to people than the associated improvements in longevity.

Most prior work on valuing morbidity has used cost-of-illness (COI) methods. COI methods use the present discounted value of lost earnings and medical costs to measure the fiscal benefits of reducing the number of cases of any given illness. For instance, US EPA (2011) uses COI methods to estimate the benefits of reducing the numbers of non-fatal myocardial infarctions and hospital admissions for respiratory

[4]Ketcham *et al.* (2020) report a declining VSL for seniors aged 67 to 97 based on revealed preference evidence from medical spending decisions.

[5]In contrast, Alberini *et al.* (2004) in a stated preference study finds no significant differences in VSL for people with or without chronic conditions like cancer or lung disease

diseases. A limitation of the resulting measures is that they exclude the utility cost of pain, suffering, and reduced functionality. My work is more closely related to a small number of studies that have estimated the WTP to avoid specific illnesses. For example, Blomquist *et al.* (2011b) and Brandt *et al.* (2012) use stated preference methods to elicit parents' WTP for treatments to reduce their children's asthma symptoms. Other studies have valued prevention including Milligan *et al.* (2010) providing estimates of WTP for cancer prevention, Jacobs *et al.* (2011) reporting estimates for cardiovascular diseases and Viscusi *et al.* (2012) providing the estimates for gastrointestinal diseases. Bauer *et al.* (2018) argue that incomplete annuity markets make treatment worth more than prevention, even if the resultant effects on longevity are the same. Hummels *et al.* (2016) computes the marginal disutility of diseases for workers and finds that worker disutility comprises 20% of wage gains from increased firm sales. Cameron and DeShazo (2013) computes the how different illness profiles modify the VSL, and Cameron *et al.* (2010) finds that WTP to avoid diseases vary substantially across diseases. [6] My work connects these strands of literature and extends them to generate VSL-age profiles and VSI-age profiles for different diseases while documenting how these profiles reflect the static and dynamic complementarity between survival and health.

3.3 The Late Life-Cycle Model

The model depicts seniors who are permanently out of the labor force, choosing how much to spend on consumption and health later in life. Abstracting from

[6] An alternative approach to valuing morbidity risks is to estimate the number of life-years lost due to disability or premature death. This approach is traditionally used in the Quality Adjusted Life Years (QALY) (developed by Fanshel and Bush (1970); Torrance *et al.* (1972))and Disability-Adjusted Life Years (DALY) literature, where researches calculate QALY weights for certain illness using time trade-offs and other procedures. Such methods are more commonly used to calculate the cost-effectiveness of a particular intervention. For an overview of the QALY literature, see Cutler and Richardson (1997)

modelling labor market and retirement decisions keeps the model computationally tractable and approximates the data reasonably well because less than 10% of Americans over the age of 65 were working full-time in 2010 (BLS, 2010). [7] Agents are assumed to value consumption and health and face uncertainty about their own future health and survival. Against this background, the agents optimally choose medical expenditure and savings.

I start with an overview of the model environment and timing (section 3.3.1). Then, I introduce specifications for health technology (section 3.3.2), preferences over health and consumption (section 3.3.3) and the resources available to the agent (section 3.3.4). I conclude by combining all of the model features into a recursive formulation of the agent's dynamic optimization problem (section 3.3.5).

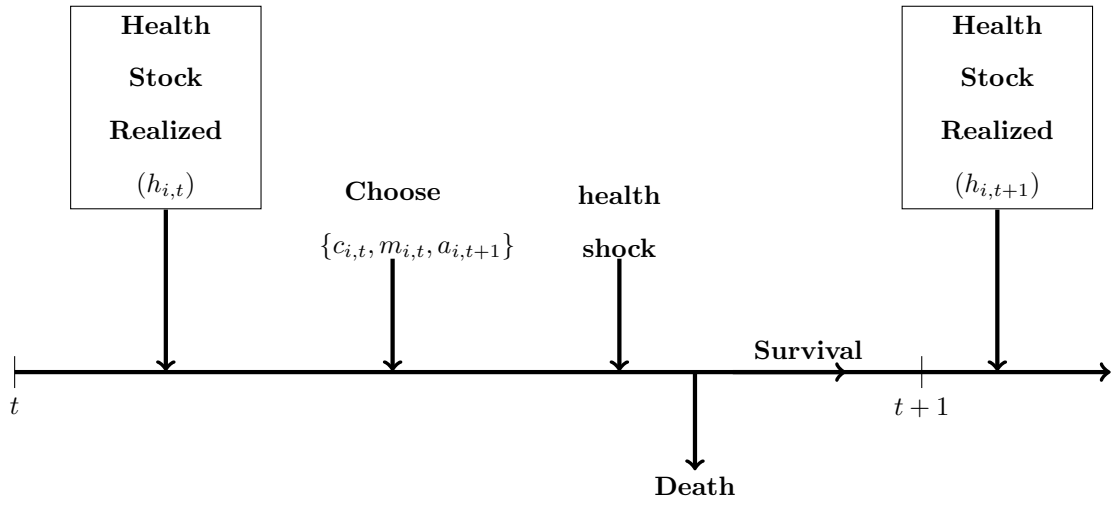
3.3.1 Environment

The model is formulated in discrete time, where t denotes age. Figure 3.1 summarizes the timing of decisions and shocks. The life-cycle begins at age 66 and ends at a terminal period T, when the individual reaches age 95. The model has 30 periods, each consisting of a year. Individuals start the life-cycle with asset holdings, $a_{i,t}$ and health stock, $h_{i,t}$. In each period, t individuals receive exogenous non-asset income and expect their health stock to depreciate. Individuals can slow the expected rate of depreciation by investing in health through medical expenditures, $m_{i,t}$. These expenditures can be interpreted as preventive care or maintenance costs. Individuals simultaneously make consumption decisions, $(c_{i,t})$,which inturn determines their asset holdings through the budget constraint. At the end of each period, they experience a health shock, which affects their end of period health stock, $h_{i,t+1}$. Finally, the

[7] In the Medicare Current Beneficiary Survey, approximately 13% of seniors aged 66 and above were working in 2010.

individuals experience a survival shock that depends on $h_{i,t+1}$. Individuals who die consume their remaining wealth in the last period.

Figure 3.1: Model Timing



3.3.2 The Health Technology

Health evolves following a partially stochastic technology. The health stock in period, $t+1$ depends on the current stock of health, medical expenditures, and an idiosyncratic shock as follows:

$$\ln h_{i,t+1} = \alpha_{0,t} + \alpha_{1,t} \ln h_{i,t} + \alpha_{2,t} \ln m_{i,t} + \nu_{i,t+1} \tag{3.1}$$

$$\nu_{i,t} \sim N(0, \sigma^2_{\nu,t})$$

where, $m_{i,t}$ represents medical expenditures, $\alpha_{1,t}$ captures the persistence of health, $\alpha_{2,t}$ is the elasticity of medical expenditures, $\alpha_{0,t}$ represents changes in health over the life-cycle independent of medical expenditures, and $\nu_{i,t+1}$ denotes the idiosyncratic health shock. All the parameters of the health production function are age-specific. The health shock is assumed to be distributed normally with mean 0 and age-specific

variance of $\sigma^2_{\nu,t}$. In essence, I assume the health shock can both positively and negatively affect the latent health stock next period. An example of a positive health shock could be a move to an area with less pollution. The health production function captures the essence of a Grossman (1972) style health capital dynamics. [8] [9]

The end-of-period health stock is then mapped into a separate deterministic function denoting the probability of surviving to the next period, $\Phi(h_{i,t+1})$. I follow Chetty *et al.* (2016b) and Finkelstein *et al.* (2019b) and adopt a Gompertz specification in which the log of the hazard rate, $1 - \Phi(h_{i,t+1})$ is assumed to be a linear function of the health stock:

$$\begin{aligned} \log[1 - \Phi(h_{i,t+1})] &= \phi_{1,t} + \phi_{2,t} h_{i,t+1} \\ \Phi(h_{i,t+1}) &= 1 - \exp(\phi_{1,t} + \phi_{2,t} h_{i,t+1}) \end{aligned} \tag{3.2}$$

Thus, survival depends on medical expenditure through the effect of medical expenditures on health. The opportunity to invest in health in order to attempt to postpone death is a crucial aspect of this model [Galama and Van Kippersluis (2019)].

3.3.3 Preferences

Each period, individuals enjoy flow utility from consumption and health:

$$u(c_{i,t}, h_{i,t}) = A + \frac{(\gamma c_{i,t}^{\theta} + (1-\gamma) h_{i,t}^{\theta})^{\frac{1-\sigma}{\theta}}}{1-\sigma}, \tag{3.3}$$

where θ denotes the elasticity of substitution between consumption and health, γ is the share of consumption, σ is the coefficient of relative risk aversion, and A is a

[8] In contrast to Grossman (1972), where time spent on exercise or other activities and other health behaviors is viewed as investment in health, I focus exclusively on medical expenditures as the only investment available to individuals to slow down the depreciation of their health stock

[9] The health production function can be augmented by adding an interaction between $m_{i,t}$ and $h_{i,t}$, in order to represent expenditures on curative care as in Ozkan (2014).However, I find that the interaction term does very little to explain the next period's health stock, so I exclude it for simplicity.

positive constant that ensures that utility from living exceeds the utility from death. [10] The specification in (3.3) previews the main trade-off that individuals must make: to choose whether to forgo current consumption in exchange for better expected health in the future. The parameters, γ, and θ influence the value of life-extension by scaling the relative utility from being alive conditional on the level of consumption and health. The idea of allowing utility to depend on the health stock is a notion that follows from Grossman (1972). The parameter θ captures the substitutability/complementarity between consumption and health, i.e., if $\theta < 0$, individuals enjoy consumption more when they are healthy. [11] The specification simplifies to the one from Hall and Jones (2007) in the special case where $\theta = 1$.

3.3.4 Budget Constraint

Current period income is defined as the sum of asset income and non-asset income, where the later is modeled as a deterministic function of exogenous permanent income, I_i and age following De Nardi *et al.* (2010). Specifically, non-asset income is expressed as:

$$y_{i,t} = f(I_i, t), \tag{3.4}$$

where, I_i is assumed to include income from all non-asset sources including, social security benefits, government transfers, and spousal non-asset income. Individuals may also derive a second source of income from the return on any asset holdings, at a risk-free interest rate, r. Thus, the total annual income available to the individual

[10] This follows from Hall and Jones (2007b). Since estimates for σ typically exceed 1, the second term in 3.3 is negative, so that positive values for the constant A is needed to ensure that agents receive positive utility from living.

[11] Complementarity is consistent with the findings of Finkelstein *et al.* (2013) that the marginal utility of consumption declines with the number of chronic diseases that an individual has been diagnosed with.

in period t is :

$$x_{i,t} = ra_{i,t} + y_{i,t} \tag{3.5}$$

From these resources, the individual decides how much to spend on non-medical consumption, medical expenditures, and how much to save. Given the structure of the public health insurance system in the United States, and the role of Medicare for people over age 65, I model annual out-of-pocket expenditures as a function of total medical expenditures, an annual deductible D and a co-insurance rate ψ:

$$oop(m_{i,t}) = \begin{cases} m_{i,t} & m_{i,t} < D \\ \psi(m_{i,t} - D) + D & m_{i,t} \geq D \end{cases} \tag{3.6}$$

This setup approximates the insurance structure of Medicare Parts A and B (basic coverage for everyone above age 65). If total medical expenditures are less than the deductible stipulated by Medicare, then individuals pay the full price. If total expenditures exceed the deductible, then they pay the deductible and a proportion of the additional amount. [12] For simplicity I assume that, individuals do not have access to any other supplemental insurance coverage.

The budget constraint is completed with the law of motion for asset accumulation and an additional no borrowing constraint: $a_{i,t+1} \geq 0 \ \forall t$. It can be written as :

$$c_{i,t} + a_{it+1} + oop(m_{i,t}) = (1 + r)a_{i,t} + y_{i,t} = x_{i,t} + a_{i,t} \tag{3.7}$$

Thus, each period assets must be allocated to out-of-pocket medical cost, non-medical consumption, or reinvested.

[12] The out-of-pocket function is similar to the one used in Ozkan (2014), but does not depend on insurance coverage.

3.3.5 Dynamic Optimization Problem

The agent's optimization problem can be written as the Bellman equation in 3.8 subject to the health production function, the survival function, the out-of-pocket function, the budget constraint (equations 3.1, 3.2, 3.6, 3.7 respectively) and the no-borrowing constraint:

$$V_{i,t}(a_{i,t}, h_{i,t}, I_i) = \max_{\{c_{i,t}, m_{i,t}\}} \{u(c_{i,t}, h_{i,t}) + \beta \, \mathbb{E} \, [\Phi(h_{i,t+1}) V_{i,t+1}(a_{i,t+1}, h_{i,t+1}, I_i)]\} \quad (3.8)$$

where, $\beta \, \epsilon \, (0, 1)$ is the discount factor, $\Phi(h_{it+1})$ is the probability of survival to age $t+1$, conditional on the realization of the health stock, h_{t+1}, that is observed after choosing medical expenditures in t. The individuals first observes h_t, I_i and a_t at the beginning of the period, and then makes decisions about $c_{i,t}$ and $m_{i,t}$. The next period's asset holdings are determined from the budget constraint based on these choices and $y_{i,t}$. At the end of the period, the agent experiences iterative shocks to health and mortality. Agents may die at the end of the period but may not leave debt as enforced by the borrowing constraint. The optimal decision rules are solved by backward recursion after discretizing the state variables and decisions (see Appendix A.3 for details).

3.3.6 Initial Conditions

I complete the model by defining the initial conditions and a set of measurement equations that relate the unobserved latent health to observed proxy measures in the data. Since individuals in this model start their life-cycle at age 66, the initial conditions are defined by the initial distribution of the state variables : $\{a_{66}, h_{66}, I_{66}\}$. Part of this distribution can be directly observed from data: $\{a_{66}, I_{66}\}$. The remaining challenge is to identify the initial distribution of health. I compensate for the non-existence of a single comprehensive measure of health by introducing a set of

measurement equations for unobserved health at each time period.

3.3.7 Measurement System

I formulate a latent factor model for health that adapts approaches used in the literature on human capital production (Cunha and Heckman (2008); Cunha *et al.* (2010); Hai and Heckman (2015); Agostinelli and Wiswall (2016)). This allows me to map the unobserved variables from the model to measurements in the data. This process addresses the multidimensional nature of health and measurement error created by using any single proxy measure in place of health. The measurement equations for health are as follows:

$$Z_{i,t,k} = \mu_{t,k} + \lambda_{t,k} \ln h_{i,t} + \epsilon_{i,t,k} \qquad \forall k = 1, ..., K \tag{3.9}$$

where, the $Z_{i,t,k}$ are the observed proxy measures for the log of health. The unconditional distribution of the latent factor $\ln h_{i,66}$ is assumed to be normal. I explain how the measurement system is identified in section 3.5 after I summarize the data.

3.4 Data

The model is estimated using data from two sources : i) Medicare Current Beneficiary Survey (MCBS) responses linked to Medicare administrative records on file at the US Centers for Medicare and Medicaid Services (CMS) and ii) the Health and Retirement Study (HRS).

3.4.1 MCBS

The MCBS is a rotating panel survey of a nationally representative sample of approximately 16,000 beneficiaries randomly drawn from the Medicare population. Each respondent is followed for up to four consecutive years, regardless of any change

in address or transfer to long-term care facilities. In the event of cognitive impairment, due to a health shock, for instance, Alzheimer's disease, a chosen proxy responds on their behalf. The MCBS questionnaire contains rich information on individuals' socioeconomic characteristics, like self-assessed health, limitations on activities of daily living, and health behaviors such as smoking, drinking, and exercise. This complements the information available in administrative records on the same individuals' diagnoses of chronic medical conditions, gross and out-of-pocket medical expenditures, demographics, and death dates.

I match MCBS records from 2005 to 2011 to respondents' Medicare administrative records for the same period. I use data on all respondents between the ages of 66 and 95 who were not working at the time of their MCBS surveys and had positive medical expenditures. This yields information on 18,717 individuals observed over 35,432 person-years. Appendix Table B.5 provides additional details on sample construction.

My sample construction embeds four main data cuts. First, I drop individuals who are still in the labor force beyond age 65. This is done to abstract from modelling retirement decisions in order to keep the model tractable. The second data cut drops individuals under age 65 because their Medicare eligibility is derived from poverty and/or medical conditions that make them a non-representative subset of the population. I also cap the maximum age at 95 because very few people (approximately 1%) live beyond this age. Finally, I dropped a small number of outlier observations where individuals had medical expenditures above $ 100,000 (less than 2% of the sample). The average number of survey years per person (2) is half of the maximum possible due to attrition from death, occasional missing variables, and the inability to observe four years of survey data for people who entered MCBS before 2005 or after 2008.

3.4.2 HRS

I complement the MCBS with information from the RAND Health and Retirement Study (HRS) dataset. Starting in 1992, the HRS provides a nationally representative longitudinal sample of people aged 51 to 61 years with a initial cohort of 12,654 individuals surveyed every alternate years. In 1998 the HRS expanded their survey population to include older and younger adults. For this study, I focus on individuals aged 65 and over from 2004 to 2012 to approximately match with the MCBS time frame. Limiting my sample to people ages 65-95 means that, I work with 5 different HRS birth cohorts (Cohort 1: 1909-1909; Cohort 2: 1920-1924; Cohort 3: 1925-1929 Cohort 4: 1930-1934, and Cohort 5: 1935-1946).

The MCBS and HRS files provide complementary information, with similar overlap. The HRS data also contain health histories, both self-reported measures and diagnosed illnesses, as well as insurance choices and medical expenditures. However, the HRS reports medical expenditures in intervals rather than continuous dollar amounts, making MCBS the preferred source of information on medical spending. In contrast, the HRS reports a richer set of continuous measures for wealth and income, making it the preferred source of information on individuals' financial portfolios.

Overall, the HRS data serves three purposes. First, they allow me to estimate the income function. Second, they allow me to construct wealth moments from the information on personal assets. Lastly, since the HRS health measures are similar to those in the MCBS, I construct the initial distribution of the state variables, using variances and covariances observed in the HRS data. To maximize consistency between the datasets, I follow the same sample selection procedure as the MCBS. [13]

[13]Specifically, I restrict the sample only to retired individuals, aged 66 to 95. Observations with any missing information on health measures are also dropped. Total medical expenditures were not reported in any of the waves from 2004 to 2012. Sample selection on income and wealth is described below.

This yields information for 8,778 individuals observed for 22,587 person-years.

3.4.3 Description of Key Variables

Measures of health

I use five sets of proxy measures for each individuals' health each year. The first measure is CMS's Hierarchical Condition Categories (HCC) score. It is computed using a risk adjustment model to produce an index of individual morbidity that synthesizes information about individuals' medically diagnosed chronic illnesses and demographics from CMS administrative records. CMS uses the HCC index to adjust capitation payments to Medicare Advantage plans based on their enrollees' health expenditure risk. [14] The index is a function of age, gender, indicators for numerous chronic illnesses, and the initial reason for Medicare eligibility. [15] The raw scores are monotonically increasing in illness, so I take a negative log transformation in order to normalize the latent health factor to be monotonically increasing in the health measure. The second measure is based on the Body Mass Index (BMI) derived from the 'height' and 'weight' variables reported in MCBS. I construct an indicator variable to represent healthy weight for those who are neither underweight nor overweight, i.e., BMI is between 18.5 and 25.

All of the remaining health measures are based on self-reported information. The third measure is the self-reported health status (SRHS), which is derived from a survey question asking individuals to rate their health on a scale starting from 'excellent'(SRHS=5) to 'poor'(SRHS=1). The last two measures are the counts of the number of restrictions on Activities of Daily Living (ADL) and Instrumental Activi-

[14] Additional background information on the risk adjustment model can be found at `http://www.nber.org/data/cms-risk-adjustment.html`.

[15] CMS only provides raw scores starting in 2007. I predict scores for the previous two years using regression analysis and demographic and health information and their interactions.

ties of Daily Living (IADL). ADL limitation measures track whether individuals are able to perform independent tasks like eating, bathing, and walking, whereas IADL measures track whether individuals are able to independently perform more complex tasks such as managing finances, cooking, and grocery shopping. The maximum number of ADL limitations is 6, and for IADL it is 5. [16]

Medical Expenditures, Income and Assets

The linked CMS-administrative data reports out-of-pocket costs and total medical expenditures for each respondent each year. The medical expenditure measure includes inpatient care, outpatient care, long-term care in skilled nursing facilities, hospice care, ambulatory surgical centers, physicians for procedures and office visits, anesthesia, and other procedures such as dialysis and durable medical equipment. However, my measures exclude expenditures on prescription drugs because this information is only available for the subset of people enrolled in Medicare Part D. On average, drug costs constitute 13% of total medical expenditures (De Nardi *et al.* (2016)). So, my measure of Medicare costs is slightly understated. Using the CPI-U, I convert the nominal amounts into constant 2010 US $. [17]

I define net assets, non-asset income, and permanent income following De Nardi *et al.* (2010). Non-asset income includes income from social security benefits, any government transfers, retirement benefits, and pensions. For permanent income, I

[16] I flip the order so that an individual with 0 ADL limitations receives a score of 6 and someone with a maximum number of limitations (6) receives 0.

[17] The MCBS also reports comprehensive data on medical expenditures. However, the MCBS does not collect data for the first year of survey participation. While CMS reports observation for all years, it only uses claims data processed under Medicare Parts A and B for the first year of survey participation and misses out on all people enrolled in Medicare Advantage (for whom claims-based spending data are unavailable). Therefore, the choice of medical expenditure data entails a trade-off between completeness and sample size. I use the CMS claims-based data for my main analysis and plan to extend my work to repeat the estimation using the smaller-sample but more comprehensive spending measure in future drafts of this paper.

first aggregate income from all sources in addition to non-asset income like earnings while working or any unemployment benefits for all individuals for all years they are observed. Permanent income is then defined as the average of that income, so it is constant for each person over time. The non-asset income function, $y = f(I_i, t)$, is estimated as a function of permanent income and a cubic polynomial in age. The HRS measure of net assets includes all assets less mortgages and other debts. I use the information on the value of housing or real estate, automobiles, money market accounts, savings accounts, Treasury bills, individual retirement accounts, Keoghs, stocks, the value of a farm or business, mutual funds, bonds, and 'other' assets.

3.4.4 Summary Statistics

Table 3.1 reports summary statistics for key variables by age. Starting with health measures, the raw HCC score increases with age, representing a deterioration of health over time. The same pattern holds for the numbers of ADL and IADL restrictions. In comparison, self-reported health is relatively stable, with only a very slight decline in age. This is likely to reflect the relative nature of the question as people are asked to report how their health compares to other people of the same age. The BMI measure indicating the fraction of people in the healthy range increases with age, likely due to selection on mortality.

Consistent with the observed trends in health proxy measures, average health spending increases over time. Average medical expenditures almost double between age 66 and age 95, while out-of-pocket expenditures increase by approximately 32%. This can be attributed to the coverage provided by Medicare health insurance. Turing to other financial variables, non-asset income increases slightly from age 66 to age 75, then declines to $17,000 at age 95. The age-gradient of net assets is relatively flatter from age 66 to age 85 and then declines sharply towards the end of the life-cycle.

Table 3.1: Summary Statistics of Key Variables by Age

	Age 66	Age 75	Age 85	Age 95
Health Measures				
HCC scores	0.90	1.12	1.34	1.33
Number of ADL conditions	0.46	0.42	0.72	1.38
Number of IADL conditions	0.49	0.46	0.81	1.53
Health Status (1: Poor; 5 : Excellent)	3.51	3.47	3.37	3.26
BMI[18.5,25]	0.29	0.36	0.47	0.54
Health Expenditure				
Medical Expenditures (2010 $)	6,900	7,908	8,632	12,642
Out-of-Pocket Cost (2010 $)	1,178	1,303	1,365	1,552
Variables from HRS				
Non-Asset Income (2010 $)	20,286	20,851	19,961	16,764
Net Asset (2010 $)	224,770	209,182	200,900	126,573

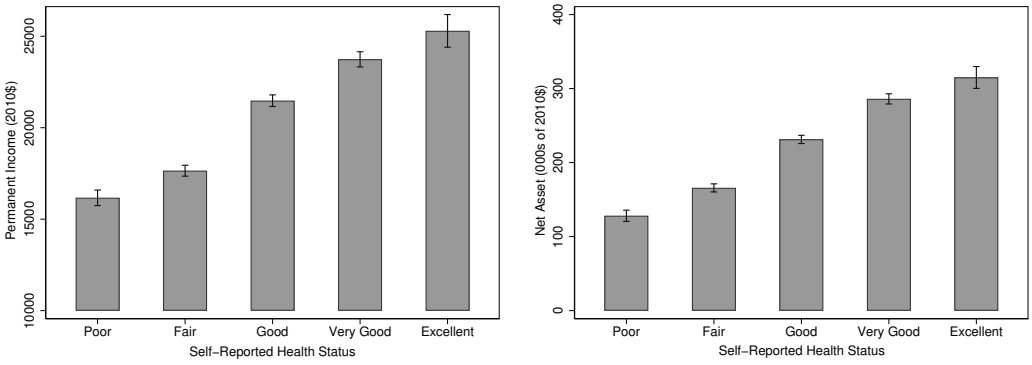
Note: All health measures, except HCC scores, come from MCBS based on survey responses. All spending and financial variables are adjusted to the year 2010 dollars using the CPI. The HCC scores and health spending measures are drawn from CMS administrative files. Non-asset income and net assets are drawn from the RAND HRS data.

Additional summary statistics including other demographic variables are reported in the appendix (Table B.6).

Figure 3.2 reports average income and average wealth by self-reported health status. Wealth and permanent income are both positively correlated with health. Similar patterns can be observed for other subjective health measures available in the HRS that are similar to the MCBS. (Appendix Figure C.5).

Figure 3.3a show how medical expenditures vary by income. Since I use the CMS data on gross and out-of-pocket medical expenditures, Figure 3.3a uses current income

Figure 3.2: Permanent Income and Net Asset Distribution by Health

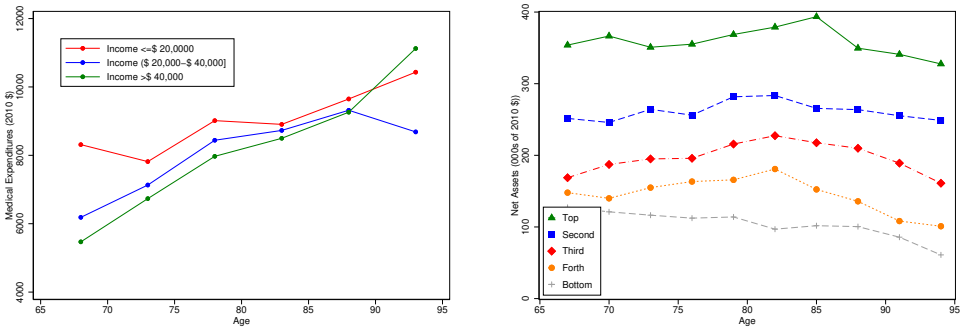


(a) Permanent Income and Health Status (b) Net Asset and Health Status

Note: Permanent income is calculated from the HRS as the average income over all the years an individual is observed. The figures in both panels use the self-reported health status reported in the HRS.

intervals rather than permanent income derived from the HRS. As shown in Figure 3.3a, the bottom income group spends the most on health care, approximately 1.5 times as much as the top income group at age 68. De Nardi *et al.* (2016) attributes almost all differences to nursing home spending, as the poor tend to spend more on nursing home care. The gap in medical consumption fades away after age 70. Figure 3.3b shows the trends in asset deccumulation over age for different permanent income groups. Income rich individuals tend to deplete their assets at a later stage in life and at a slower rate compared to poorer individuals. For the top two permanent income quintiles, asset deccumulation starts after age 85 and also at a slower rate compared to individuals at the bottom two quintiles. This is consistent with the findings in Dynan *et al.* (2002) and De Nardi *et al.* (2010).

Figure 3.3: Medical Expenditure and Net Asset by Income



(a) Medical Expenditure by Income Group

(b) Net Asset by Permanent Income Quintiles

Note : In panel (a), the income variable is drawn from the MCBS based on survey responses. It is the aggregate individual income from all sources and is reported in intervals. The permanent income in panel (b) is calculated from the HRS averaging over all years an individual is observed.

3.5 Estimation

I use a two-step estimation procedure following Gourinchas and Parker (2002); De Nardi *et al.* (2010) and Hai and Heckman (2015). In the first step, I estimate auxiliary processes that affect model predictions but are assumed to independent of the structural parameters targeted by my model. In the second step, preference parameters are calibrated using the simulated method of moments.

3.5.1 External Calibration

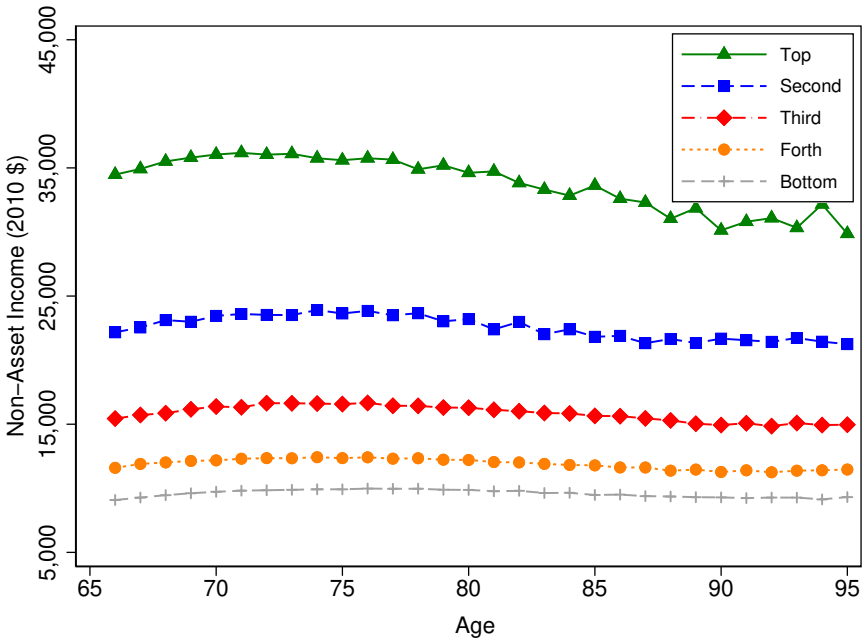
Non-asset income

The non-asset income function, $Y_{i,t} = f(I_i, t)$ is estimated as a cubic function of age and quadratic in permanent income. The basic regression specification is as follows :

$$Y_{i,t} = \xi_i + \Pi_1 age_{i,t} + \Pi_2 age_{i,t}^2 + \Pi_3 age_{i,t}^3 + \Pi_4 I_i + \Pi_5 I_i^2 + u_{i,t}$$

Figure 3.4 shows the resulting prediction for non-asset income by permanent income quintile, using the individual fixed effects to normalize the predictions to represent the means for cohort 5 (1936-1945). People in the top income quintile receive an average of $35,000 of annual income from non-asset sources. Figure 3.3b shows that the average asset holdings for an individual at the top quintile is $350,000 or about 10 years of income.

Figure 3.4: Average Non-asset Income by Permanent Income Percentiles



Other Parameters

Based on the structure of the out-of-pocket cost in equation (3.6) in the model, I estimate the parameters for the co-insurance rate (ψ) and deductible (D) directly from MCBS data. I estimate $D = \$246$ and $\psi = 0.16$. [18]

[18]This approximates the complex structure of Medicare payment plans. For instance, in 2010, all expenses were covered under Medicare Part A except for inpatient hospital stays and skilled nursing facilities. In the case of inpatient hospital stays, a deductible of $1,100 covered Medicare beneficiaries for the first 60 days. After this period, they were required to pay a daily co-insurance of

I set the interest rate, r to be 0.04 which is widely used in the literature (French and Jones (2011); Fonseca *et al.* (2020)). I also set the terminal period, T to 95 years. Table 3.2 summarize the parameters calibrated outside the model.

Table 3.2: Externally Calibrated Parameters

Description	Parameter	Value	Source
Co-Insurance Rate	ψ	0.16	MCBS sample
Deductible	D	\$ 246	MCBS sample
Interest Rate	r	0.04	Fixed parameter
The Maximum possible lifespan	T	95	Fixed parameter
Income Function	$y_{i,t}(I_i, t)$	-	HRS Sample

3.5.2 Estimating Structural Parameters

I first estimate the parameters describing the health measurement system, the initial distribution of health, the health production function, and the survival function parameters. Then, I estimate the remaining utility function parameters describing preferences. Individuals enter the model at age 66 with initial assets, a_t, health,h_t, and permanent income. I construct the initial distribution from data on the subset of individuals who turned 66 during my study period (2004-2012). Their initial health is drawn from the distribution of health, estimated using the measurement system parameters, then factor scores are obtained. The covariances between latent health and permanent income is also estimated in this step. Together these results are used to define the initial joint distribution of assets, health, and permanent income.

\$275 for days 61-90, and \$550 after that. For stays in skilled nursing facilities, the daily co-insurance was \$137.5 for days 21-100. Under Medicare Part B, the Medicare beneficiaries paid annually \$155 in deductibles and 20% as co-insurance.

[19] This initial distribution accounts for the empirical correlations between health, mortality and income.

I then simulate the distribution of medical expenditures and health over the remaining life-cycle and use these simulated data to compute model predictions for moments (M_s) that I observe in the MCBS and the HRS data. Then, I solve for model parameters to minimize the weighted distance between the simulated moments and the data moments(M). More formally, the vector of parameters to be estimated is denoted by Ω:

$$\hat{\Omega} = \underset{\Omega}{argmin}(M - M_s(\Omega))W(M - M_s(\Omega))', \tag{3.10}$$

where, W represents the weighting matrix. The weighting matrix is set to be the inverse of the diagonal variance-covariance matrix of moments computed by bootstrapping. The set of parameters to be estimated is summarized in Table 3.3 below. I use four sets of moment conditions describing the evolution of mean wealth, health, medical expenditures, and survival probabilities over the life-cycle. The construction of the age profile for different moment conditions is detailed below.

Moments

Net Assets: The asset-age profile is constructed from the HRS data, accounting for cohort effects. Following French and Jones (2011), I first run an individual-specific fixed effects regression with controls for integer age dummies and household size. The age profile is then created after normalizing the individual-specific fixed effects to represent the mean for Cohort 5 (birth year 1935-1946) and fixing the household size to 3.

Medical Expenditure: The medical expenditure profile is constructed from MCBS data using a similar method. First, an individual-specific fixed effects regression with

[19] This is drawn from the same HRS birth cohort (cohort 5: BY 1935-1946)

Table 3.3: Parameters to be Estimated

Description	Parameters
Preference Parameters	
Utility of being alive	A
Discount Factor	β
Elasticity of substitution between consumption and health	θ
Share of Consumption	γ
CRRA coefficient	σ
Parameters of Health Production Technology	
Persistence of health factor (5 age-groups)	$\alpha_{1,t}$
Elasticity of medical expenditures (5 age-groups)	$\alpha_{2,t}$
Intercept (5 age-groups)	$\alpha_{0,t}$
Standard Deviation of Health Shock (5 age-groups)	$\sigma_{\nu,t}$
Parameters of Survival Function	
Effects of Health	$\phi_{2,t}$
Constant	$\phi_{1,t}$

Note: The parameters of the health production function and survival function are first estimated using the dynamic latent factor model. Then the preference parameters are estimated using Simulated Method of Moments (SMM).

age dummies is estimated to compute the age effects, and then the individual fixed effects are normalized to represent means for cohort5.

Health Status: Again, using the MCBS, I construct an average health status profile using a fixed-effect regression model with age cohort dummies, adjusting cohort effects to be representative of Cohort 5.

Survival Probability: The point estimates for the survival probability at each age

is calculated from the US life tables using the probability of death between age t and age $t+1$.

Table 3.4 summarizes the targeted moments. I use 96 moments to estimate the 5 preference parameters in Table 3.3.

Table 3.4: Targeted Moments for Calibration

Targetted Moments	# Moments
Mean survival probabilities by age [age 67-90]	24
Average net wealth by age [age 67-90] (2010 US $)	24
Average health status by age [age 67-90]	24
Mean annual medical expenditure by age [age 67-90] (2010 US $)	24

3.5.3 Identification

Factor Model and Measurement System

Identifying the parameters describing the health production function estimation requires normalizing the location and scale of the factors (see Anderson *et al.* (1956)). I follow the algorithm in Cunha *et al.* (2010) to estimate the measurement parameters in the initial period. Details of the normalization and estimation of the measurement parameters in the initial period are described in Appendix A.4.

The main challenge in the identification of the health distribution comes from the fact that health is unobserved and that there are no natural scale and location. The initial conditions are assumed to be drawn from a joint normal distribution, which therefore implies health is normally distributed :

$$\Omega = (a_{66}, \ln(h_{66}), I_{66}) \sim N(\mu_\Omega, \sigma_\Omega),$$

To set the scale, I normalize the factor loading with respect to the log of HCC

scores implying that, the latent health loads into the HCC measure in the same manner for all ages. The location is set by normalizing the mean of the log of latent health to a positive constant 1. Then, I exploit the multiple proxy measures of health available in the data, which involves making assumptions about the covariance structure of measurement errors. Specifically, I assume the measurement errors are contemporaneously uncorrelated across measures, independent over time and of the latent health stock in the model. The proxy measures are a combination of subjective and objective health measures. The covariance restrictions simply imply that the errors made by physicians when diagnosing diseases are different from the patient's misjudgment of their own health. These normalization and covariance restrictions allow me the identification of all other factor loading and the location of the health measures. Then, the variance of health can be computed by using the covariances between health measures and their factor loadings as shown in Section A.4.2.

Identification of Structural Parameters

The health technology parameters are estimated from the factor scores computed using the parameters from the measurement system. Details are described in Appendix Section A.4.3. Using the measurement parameters, I construct a residual measure of health which is composed of errors and the latent health. Substituting this residual measure into the health production function, I instrument the residual measure with other proxies. This identification strategy follows from the assumption underlying the use of multiple proxy measures of health with uncorrelated errors. This compensates for the lack of instrumental variables required to identify the parameters of the function in the presence of measurement errors. While this assumption seems strong, they are standard in the human capital literature of skill formation and parallels the formulation of the evolution of latent health in this study. Therefore,using other

proxy measures as instruments for a given proxy measure of health helps identify the parameters of the health production function.

The survival function is estimated similarly. Due to the non-linear specification of the survival function, I use a control function approach to estimate its' parameters. For a given residual measure of health, other proxy measures are used as instruments.

Although the model is over identified, each parameter can be intuitively linked to observable sources of variations in the data that are captured by particular sets of moments. The asset profile contains information that helps to identify the discount factor, β, as well as the CRRA coefficient, σ. Intuitively, the observed wealth profile in Figure 3.3b showing gradual savings for most of the sample through their early 80s, followed by a steeper decline at the end of life-cycle is consistent with a high coefficient of relative risk aversion and a low discount factor.

The evolution of average medical expenditure and health over the life-cycle contain information that helps to identify the elasticity of substitution between consumption and health. Changes in medical spending between periods help identify the elasticity, θ. A higher value of θ would imply that individuals will choose to spend more on health care later in life in order to to increase their health by enough to maintain the utility derived from non-medical consumption. The value of A is identified from the average survival probability profile. The larger the value of A, the longer the life expectancy individuals attempt to achieve, which motivates them to spend more on health to delay death.

Lastly, the gradients of health and medical expenditures with age help to identify the parameter, γ, which governs the importance of consumption relative to health. A flatter trajectory of health and a steep increase in medical expenditures would be consistent with a lower value of γ, as it would imply individuals placing a higher value on health relative to consumption.

3.6 Estimation Results and Model Fit

In this section, I discuss the main estimation results. In particular, in section 3.6.1, the estimates of the measurement parameters are discussed, using the method discussed in the appendix section A.4. Next I discuss the estimates of the health production function and survival function in section 3.6.2 using the measurement parameters in the first stage. In the last two parts of this section, the goodness of fit of the model and the estimated preference parameters are discussed.

3.6.1 Measurement System

Table 3.5 report the initial distribution of the state variables, $(a_{66}, \ln(h_{66}), I)$. The mean of the log of health at age 66 is normalized to 1. I normalize the health factor to the inverse of the raw HCC scores so that the variances and covariances of the latent health factor and the health production function parameters are interpreted relative to this normalization. [20] As expected, the three state variables are moderately positively correlated.

Parameter estimates for the measurement system and age-group-specific variance decomposition are reported in Appendix Table B.7. The health factor captures most of the variation in the transformed HCC scores (between 76% and 84% depending on age group). Signal-to-noise ratios are lower for the other observable measures of health. The latent factor only captures 13 percent of the variation in self-reported health status. At the opposite extreme, the health factor explains less than 1.1% of the variation in the BMI variable. This is plausible given in Table 3.1, the summary statistics for BMI suggest people become healthier over age, which could be because

[20] The initial distribution of the state variables is constructed using the HRS data combined with the measurement parameters estimated in the initial step from MCBS. Alternatively, the measurement parameters can be constructed using the HRS health measures. This alternative approach yields similar estimates of the joint distribution of health, assets, and permanent income.

Table 3.5: Initial Joint Distribution of Assets, Health and Permanent Income

a_{66} : Net Asset	$\log h_{66}$: Log Health	I : Permanent Income
Mean		
224.769	1.000	22.842
(9.532)	(0.068)	(0.681)
Variance-Covariance Matrix		
66085.106		
(4315.667)		
134.963	0.515	
(16.851)	(0.095)	
1610.967	7.979	295.674
(172.866)	(1.164)	(30.697)
Correlation Matrix		
1.000		
0.319	1.000	
(0.037)		
0.364	0.282	1.000
(0.034)	(0.038)	

of selection on mortality.

3.6.2 Health Production and Survival Function

Table 3.6 reports the health production function parameters. The model is estimated separately for people in each of five age bins in order to account for cohort effects: (66-70, 71-75, 76-80, 81-85, 86-95). There are several notable results. First, the coefficient on log health shows that health tends to be very persistent in the late

60s. As people get older, the estimated coefficient on log health declines, indicating a faster rate of decline. Second, the elasticity of health with respect to medical expenditures is monotonically increasing in age. This would suggest that conditional on current health, a 90-year old individual will tend to have higher returns to medical spending compared to an 80-year old. This finding is consistent with the results reported by Ketcham *et al.* (2020), who find that the returns to medical spending with respect to survival increase with age. Third, the health production function intercept decreases with age, suggesting that health tends to decline at a faster rate as people get older. Lastly, the variance of the health shock increases with age, indicating that, individuals become more vulnerable to health shocks as they age.

Table 3.6: Parameter Estimates of the Health Production Function

	(1)	(2)	(3)	(4)	(5)
	Age 66-70	Age 71-75	Age 76-80	Age 81-85	Age 86-95
Log (Current Health) [α_1]	0.996***	0.946***	0.894***	0.862***	0.812***
	(0.043)	(0.056)	(0.046)	(0.098)	(0.136)
Log (Medical Expenditure) [α_2]	0.008	0.016*	0.0201**	0.0239*	0.0297*
	(0.008)	(0.010)	(0.010)	(0.014)	(0.018)
Intercept Constant [α_0]	-0.032	-0.015	-0.048*	-0.061**	-0.072**
	(0.022)	(0.033)	(0.028)	(0.033)	(0.039)
Variance of Health Shock [σ^2_ν]	0.019	0.025	0.050***	0.065***	0.08***
	(0.014)	(0.018)	(0.018)	(0.022)	(0.028)
N	4,836	3,773	4,444	3,521	2,121

Note: The table shows the estimates for the health production function. The dependent variable is the log of next period's health. Each column represents separate estimates by age bins. The standard errors are computed using 1,000 bootstrapped replications clustering on individuals. Asterisks indicate statistical significance at the 1% (***), 5% (**) and 10% (*) levels.

Table 3.7 reports estimates for the survival function parameters. I mitigate po-

tential bias from measurement error in health by using a control function approach. The estimation approach follows from the algorithm in health production function i.e. I instrument for a given endogenous proxy variable with other proxies based on the assumption that measurement errors across proxy measures are uncorrelated. The control function residuals are statistically significant, indicating that unobservably sicker people die sooner and that ignoring measurement errors in the health measures biases the estimates of survival function parameters.

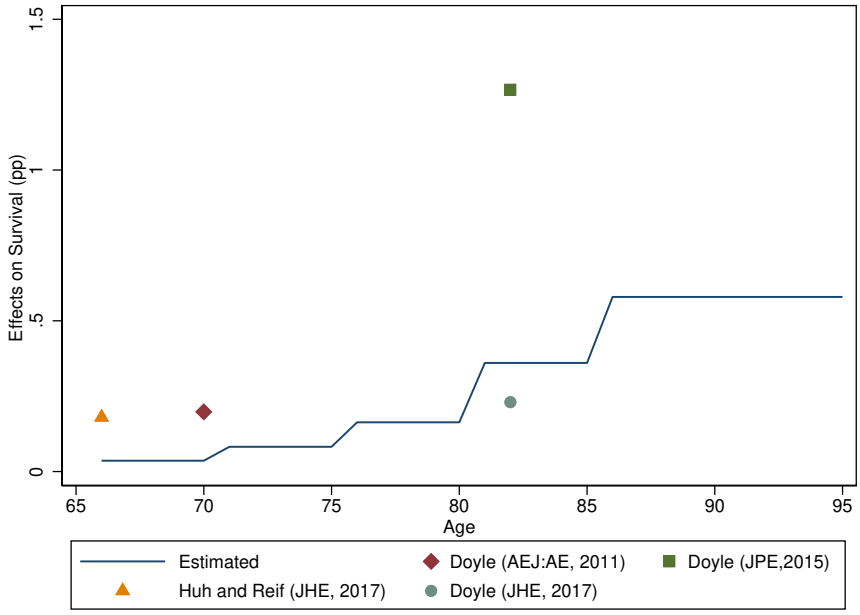
Table 3.7: Parameter Estimates of Survival Function

	(1)	(2)	(3)	(4)	(5)
	Age 66-70	Age 71-75	Age 76-80	Age 81-85	Age 86-95
Log (Health) [ϕ_2]	-2.274***	-2.138***	-2.108***	-2.470***	-2.374***
	(0.2586)	(0.254)	(0.208)	(0.190)	(0.255)
Intercept [ϕ_1]	-1.984***	-1.804***	-1.470***	-0.567***	-0.045
	(0.251)	(0.222)	(0.206)	(0.179)	(0.245)
Residual	2.004***	1.988***	1.896***	2.236***	2.157***
	(0.262)	(0.262)	(0.210)	(0.201)	(0.255)

Note: The table shows the estimates for the survival function. The dependent variable is the one-year probability of death. Each column represents separate estimates by age bins. The standard errors are computed using 1,000 bootstrapped replications clustering on individuals. Asterisks indicate statistical significance at the 1% (***), 5% (**) and 10% (*) levels.

To assess how the implied returns to medical spending compare with estimates in the literature, I use the parameters reported in Table 3.6 and 3.7, to calculate the returns to medical expenditures in terms of its effect on mortality. Figure 3.5 shows that my life-cycle estimates are broadly consistent with the collective evidence on heterogeneity in the returns to medical spending from prior literature. Each point in the figure shows the estimated effect of a $1,000 increase in spending along with the average age of the study sample. The solid line shows my age-specific estimates.

Figure 3.5: Estimated Marginal Returns to $1,000 Medical Expenditure



Notes: The figure plots the marginal effects of medical expenditure as percent point changes in the one-year probability of death. The solid line plots the combined marginal effects using the results in Table 3.6 and 3.7. The point estimates report the corresponding marginal effects in the literature for the average individual in those studies.

My findings suggest that, on average, a $1,000 increase in spending increases the survival probability by 0.30 percentage points. The marginal effects of spending on survival are relatively low for ages 65-70 (0.04 percentage points) and then increase with age, rising to 0.58 percentage points after age 85. Thus, the marginal cost of reducing mortality risk is relatively small for people with low survival probabilities, and then it increases at an increasing rate. For instance, the marginal cost of reducing one-year mortality risk by 10% for the 90-year-old is $2,600 compared to $7,500 for the average 70-year-old. These estimates fall within the the range suggested by the existing literature. [21]

[21] At the lower end, Huh and Reif (2017b) estimates that implementation of Medicare Part D

3.6.3 Preference Parameters

Table 3.8 reports the preference parameters estimated using the simulated method of moments. In cases where my estimates are comparable to prior literature, the results tend to be similar. [22]

Table 3.8: Preference Parameters

Description	Parameters	Values
Coefficient of Relative Risk Aversion	σ	3.025
Discount Factor	β	0.961
Utility of being alive	A	6.455
Elasticity of substitution between consumption and health	θ	-0.485
Share of Consumption	γ	0.650

The coefficient of relative risk aversion (3.025) is close to the value of 3.189 estimated by Fonseca *et al.* (2020) and somewhat smaller than in De Nardi *et al.* (2010) who obtain an estimate of 3.81. One potential explanation for this difference is that De Nardi *et al.* (2010) treats medical expenditures as an exogenous shocks rather than as a choice variable. On the other hand, my estimate for the discount factor is

reduces mortality by 0.036 percentage point for 66-year olds and increases their drug utilization by $244; this translates to a reduction in mortality by 0.15 percentage points for each $1,000 spending increase in prescription drugs. For a similar younger Medicare population, Doyle (2011b) leverages spatial variation in treatment intensity and estimates mortality reductions of 0.2 percentage points for an additional $ 1,000 of end-of-life expenditures (in 2010 dollars) for Medicare beneficiaries experiencing heart–related emergencies leading to a hospital admission through the emergency department during their visit Florida. Similar estimates are reported by Doyle *et al.* (2017) for an older population (average 82 years) using quasi-random variation in hospital assignment due to ambulance referral patterns to estimate the returns to 90-day hospital spending among Medicare patients who receive care following a health emergency. In an earlier study, for a similar population, Doyle *et al.* (2015b) estimates much higher returns to one-year spending following the initial hospital spending induced by the emergency: reduction in mortality risks is approximately 1.26 percentage points for each $1,000 additional spending.

[22] I am in the process of estimating the standard errors for the parameters. Future versions of the paper will incorporate the bootstrapped standard errors.

0.961, which is close to the estimate from De Nardi *et al.* (2010) (0.972) but somewhat smaller than the estimate from Hall and Jones (2007b) (0.992).

My results imply that the consumption share parameter is 0.65. I also find that the elasticity of consumption with respect to health is negative (-0.485). This implies that consumption and health are complements, so that individuals enjoy consumption more when they are in better health. This helps to explain why consumption is not smoothed over the life-cycle. As individuals age and their health stock declines, they derive less utility from non-medical consumption.

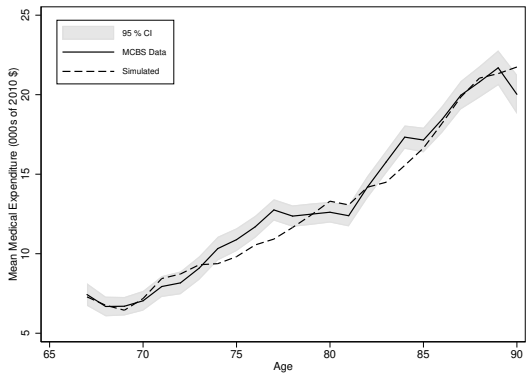
Finally, the parameter defining the utility of being alive, A, is sufficiently large to ensure that flow utility is positive over the relevant values of consumption, so that individuals value life extension. This parameter influences the level of the VSL associated with a given risk. The ability to identify A differentiates my framework from model where the survival probability is exogenous, and the value of A is calibrated to match the peak VSL, for instance (Aldy and Smyth (2014)).

3.6.4 Model Fit

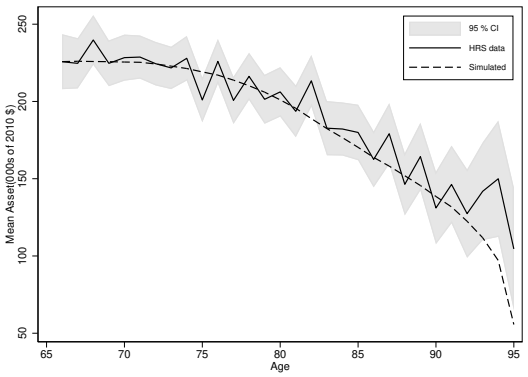
Figure 3.6 shows how the model simulations fit the data moments. The life-cycle profiles estimated using the data are illustrated in black solid lines, together with the shaded 95% confidence bands. Panel 3.6a shows the evolution of annual average medical expenditures using the MCBS over the life-cycle. Average medical expenditure grows steadily over the life-cycle due to deterioration of health. Panel 3.6b illustrates the profile for annual average net asset holdings from HRS. The average 1940 cohort's assets remain steady until age 75 when it starts declining gradually at a slow rate. Panel 3.6c illustrates how the average self-reported health status (1=poor; 5=excellent) reported in MCBS vary over the life-cycle and panel 3.6d exhibits the mean survival probability profile taken from the US Life Tables 2010. The survival

probability profile only reports the point estimates, hence it does not contain the confidence bands. The dashed lines plot the life-cycle profiles using simulations from the model.

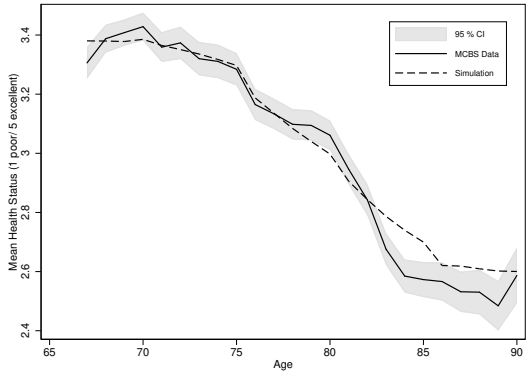
Figure 3.6: Model Fit



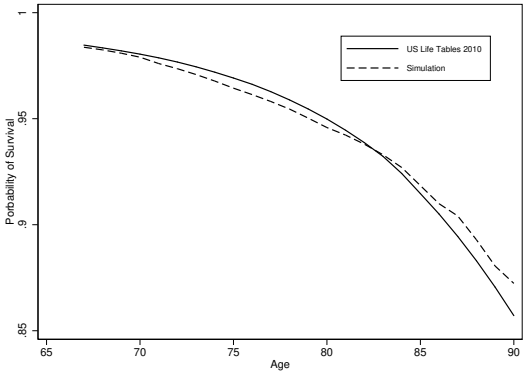
(a) Mean Medical Expenditure



(b) Mean Asset



(c) Mean Health Status



(d) Probability of Survival

Notes: Age Profile for Data simulation. The age profiles for medical expenditure in panel 3.6a and health status 3.6c are drawn from MCBS, while panel 3.6b reports age profile for mean assets from HRS. The age-profile for the survival probability in 3.6d is taken from US Life Tables 2010. The solid lines show the average profiles observed in data, whereas the dashed lines show the profiles simulated by the model.

The model simulations show that the model fits the data reasonably well. This is true for the life-cycle profiles of mean medical expenditure, survival probability, and

mean asset. The mean asset profile falls out of the confidence bands at the very end of the life cycle, and can be attributed to the departure from not modelling bequest motives. Bequest motives alter the rate of deccumulation of wealth by individuals, as individuals try to keep a part of their asset for their heirs. [23] Lastly, the simulated profile for mean health status lies mostly within the confidence bands except after age 83. One possible explanation for this is that, I did not model unhealthy behavior for instance, smoking or drinking, into the health production function, which could exacerbate health status more steeply towards the end of the life-cycle. Still, the model captures the overall declining pattern in mean health status. In Appendix Figure C.6, I also show how the model is able to replicate non-targetted moments of median gross medical expenditures and the mean out-of-pocket costs.

3.7 Willingness to Pay for Reduced Morbidity and Mortality

In this section, I use the calibrated late life-cycle model to estimate the average willingness to pay for mortality and morbidity risk reductions, which I aggregate into measures of the value of statistical life and the value of statistical illness. My procedure for estimating VSL is similar to Aldy and Smyth (2014) in that it uses a discrete time analog to the continuous time approach of in Murphy and Topel (2006) and Bauer *et al.* (2018).

3.7.1 Value of a Statistical Life

The EPA defines VSL as "the aggregate dollar amount that a large group of people would be willing to pay for a reduction in their individual risks of dying in a year, such that we would expect one fewer death among the group during that year on average". In conventional hedonic wage models, VSL is calculated as the aggregate

[23] This will be incorporated for future alternative specifications of the model

wages needed to compensate workers for the marginal death on the job each year. I make a similar calculation to derive the aggregate amount needed to compensate seniors for a marginal death each year.

Consider a small perturbation to the age t survival probability, $\Delta\Phi_t$, so that, the new survival probability at that age is $\Phi_{i,t} + \Delta\Phi_t$. The survival probabilities in future periods remain constant. Changing the age t survival probability alters the optimal level of medical expenditure and savings for the individual. This in turn, affects the asset holdings and the continuation value, $\mathbb{E}_t[\Phi(h_{i,t+1})V_{i,t+1}(a_{i,t+1}, h_{i,t+1}, I_i)]$. Taking all those changes into account, the VSL can be calculated by comparing the baseline value function, $V_{i,t}(a_{i,t}, h_{i,t}, I_i|\Phi_{i,t})$ with the counterfactual value function, $V^*_{i,t}(a_{i,t}, h_{i,t}, I_i|\Phi_{i,t} + \Delta\Phi_t)$ that embeds the new survival probability. In order to calculate the willingness to pay for this one-time change in the survival probability, I calculate the change in assets, $\Delta a_{i,t}$ needed to make the individuals indifferent between the two scenarios:

$$V_{i,t}(a_{i,t} + \Delta a_{i,t}, h_{it}, I_i|\Phi_{i,t}) = V^*_{i,t}(a_{i,t}, h_{i,t}, I_i|\Phi_{i,t} + \Delta\Phi_t)$$

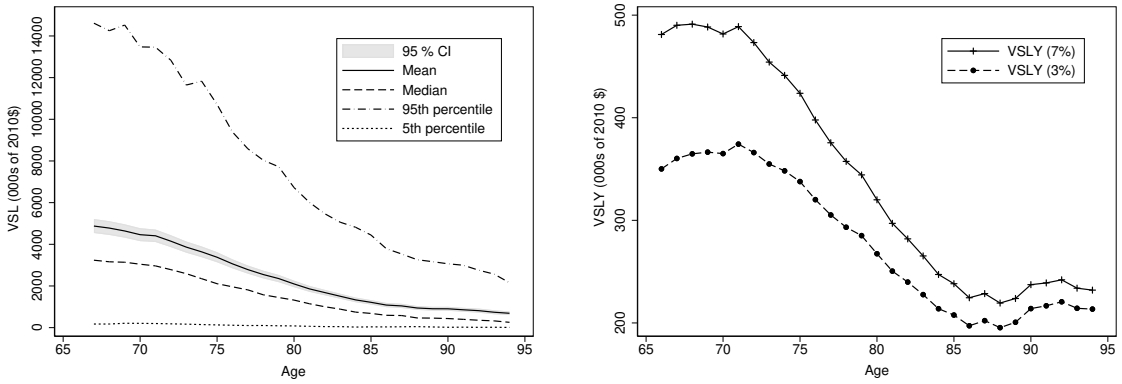
Thus, $\Delta a_{i,t}$ is the amount of assets the individual is willing to pay in the status quo in exchange for an increase in life expectancy. The VSL can then be defined in this context as the ratio of the change in wealth to the change in the survival probability. The change in mortality risk translates to one fewer deaths in a population of $\frac{1}{\Delta\Phi_t}$ identical individuals, so that aggregating willingness to pay produces the following VSL measures:

$$VSL_{i,t} = \frac{\Delta a_{i,t}}{\Delta\Phi_t}$$

Given the state variables, the VSL will vary across the population with respect to asset holdings, permanent income and health.

Importantly, the curvature of the utility function implies that the level of VSL will depend on the size of the perturbation in the survival probability, $\Delta\Phi_t$. Indeed, I find that VSL is a concave function of $\Delta\Phi_t$. To calibrate the level of VSL at age 67 to match estimates from prior literature, I choose $\Delta\Phi_t$, so that the VSL approximately matches the estimates of 4.8 million (2010 $) for workers aged 55-62 in Viscusi and Aldy (2007). [24]

Figure 3.7: Distribution of VSL and VSLY Over the Life-Cycle



(a) VSL over the life-cycle

(b) VSLY over the life-cycle

Notes: Both VSL and VSLY are reported in 1,000s of 2010 $. Panel 3.7a depicts the mean VSL profile over the life-cycle with the 95% confidence intervals. The figure also reports the median, 95th percentile, and 5th percentile of the distribution of VSL over the life-cycle. Panel 3.7b reports the VSLY profile based on 3% and 7% discounting of life expectancy.

Figure 3.7a plots the VSL profile for ages 67 to 94. The figure highlights several important findings related to the magnitude, shape and distribution of VSL over the life-cycle. First,as a benchmark for comparison, well-identified hedonic wage models for prime-aged workers yield VSL measures from $8 to $10 million [Lee and Taylor (2019b); Kneisner *et al.* (2012)]. The solid line in Figure 7a plots the mean VSL by age. Thus, the size of the difference between my estimates and those derived from

[24]Ketcham *et al.* (2020) reports substantially lower VSL measures for people over age 67 based on higher mortality risks.

wage-hedonic models grows with age. The solid line in the figure plots the mean VSL by age. At age 67, the average VSL is $4.7 million, which is also the peak VSL over the remaining life-cycle. The shape of the VSL curve is common to all simulated agents, i.e., it falls as people age. The mean VSL is around 30% higher than the median at age 67, and this gap declines as individuals age. There are stark differences in VSL between the 95th percentile and individuals at the 5th percentile, reflecting substantial heterogeneity in the WTP for reduced mortality risk. This stems from heterogeneity in health and income. To illustrate this heterogeneity, I examine the distribution of VSL at age 70. Figure C.7 shows that VSL measures for most types of individuals lie between $0 and $10 million, although the long tail generates a few VSL values as high as $27.5 million. This variation creates a wedge between the mean ($4.0 million) and the median ($2.8 million) at age 70. The standard deviation (not shown in Figure 3.7a) also declines with age, from $5 million at age 67 to $0.5 million at age 94. The VSL declines faster for people at the top of the income distribution because, they deccumulate assets faster as they age.

Policy evaluations sometimes use an alternative statistic, the value of a statistical life year (VSLY). I calculate the VSLY in Panel 3.7b by combing the average VSLs in Panel 3.7a and the discounted life expectancy reported in the US Life Tables for 2010. I calculate the age-specific VSLY values using the Office of Management and Budget's recommended range of discount rate for valuing mortality risk reductions [U.S. Office of Management and Budget (2003)]. Using a 7% discount rate yields a $480,000 VSLY for an average senior at age 67. The VSLY then declines over the remaining life-cycle, consistent with the hypothesis that the value of life extension declines with age as the health stock depletes. To further explore the heterogeneity, I examine variation in the VSL with health and income.

3.7.2 Heterogeneity in VSL

Heterogeneity by Health

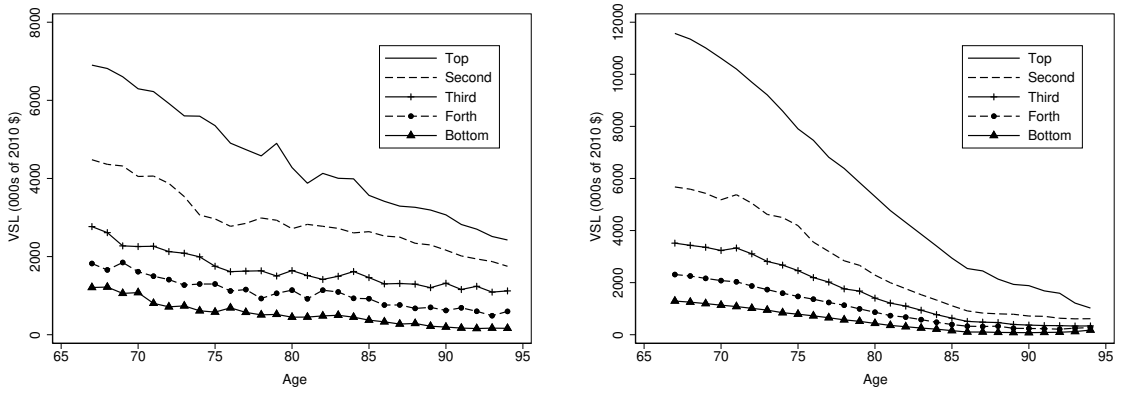
Figure 3.8a summarizes how the VSL varies with health. At age 67, the average VSL is about $7 million for people in the top health quintile, which is almost three times the average VSL for people in the bottom quintile. People in the bottom quintile are more likely to suffer from chronic conditions and functional limitations. The gap between health quintiles declines with age because of the declining difference in life expectancy. As another way to see how the VSL varies with wealth and health, notice that individuals in the top quintile at age 92 have similar VSLs to people in the third quintile at age 70.

Heterogeneity by Income

Figure 3.8b plots the VSL estimates by income quintiles. At the top of the income distribution, the average VSL is $11 million at age 67, which is an order of magnitude larger than the VSL of an individual at the bottom of the income distribution. Further, the average VSL for a person in the top income quintile at age 90 is similar to the VSL of a person in the third quintile at age 80. As in the case of health, the difference between VSL measures between different income groups narrows as people age: at age 94, the average VSL in the top income quintile is three times the average VSL in the bottom quintile.

Previous studies have suggested that VSL should increase with income, and there is a wide range of estimates for the income elasticity. At the lower end, Viscusi and Aldy (2003) reports measures in the range of 0.5 to 0.6 based on a meta-analysis of 50 wage-risk studies spanning ten countries. Mrozek and Taylor (2002) reports similar estimates. In a more recent study, Viscusi and Masterman (2017) estimates

Figure 3.8: Heterogeneity in VSL



(a) VSL by Health Quintiles

(b) VSL by Income Quintile

Notes: Both figures report VSL in 1,000s of 2010 $. Panel 3.8a reports the VSL stratified by health quintiles while panel 3.8b reports VSL by income quintiles for the simulated population using the model.

an elasticity of 0.5 to 0.7 for the US and just over 1 for non-US countries. Estimates from the higher end of the spectrum in the literature, suggest an elasticity over 1. For example, Murphy and Topel (2006) suggests the income elasticity should be 1.33, based on a calibrated life-cycle model. Kniesner *et al.* (2010) estimates an average income elasticity of 1.44 while Costa and Kahn (2004) use historical wage data from 1940 to 1980 to generate an estimate from 1.5 to 1.7. Viscusi *et al.* (2012) shows a downward trend with age.

Similar to Aldy and Smyth (2014), I use my model to estimate the elasticity with respect to permanent income and current annual non-asset income. [25] Appendix Table B.8 reports the elasticity implied by my model for various age-groups with respect to both measures. Consistent with Aldy and Smyth (2014), both measures follow a declining trend with age. Younger seniors tend to have more life-years left, so they are able to spread out increased income over longer periods of greater con-

[25] Aldy and Smyth (2014) estimated average elasticities of 0.81 and 0.15 at age 60 with respect to permanent income and realized income, respectively

sumption, raising utility and hence WTP to reduce mortality risk. I find a permanent income elasticity of 0.81 for people in their early 70s and 0.57 in their late 90s. The corresponding values with respect to non-asset annual income are larger (1.15 and 0.85). Since non-asset annual income is an estimated function of permanent income and age, the variation in current income is less than that of permanent income.

3.7.3 Willingness to Pay for Morbidity

My approach to calculating the estimated WTP to reduce morbidity risk parallels my VSL calculations. I call the resulting measure the "value of a statistical illness". The value of a statistical illness (VSI) is defined as the WTP to avoid one case of a particular illness. Bauer *et al.* (2018) defines VSI as "the aggregate WTP in order to eliminate a disease risk that is expected to befall them". To fix ideas, suppose that at age t, there is a probability q_t of next period's health being reduced by δ_t due to a particular disease, in which case next period's health will be lower relative to the baseline. This is achieved by changing the intercept in the health production function in equation 3.1. The expected reduction in health causes both survival probabilities $\Phi_{i,t}(h_{i,t+1})$ and the expectations at age t of future values, $\mathbb{E}_t[\Phi(h_{i,t+1})V_{i,t+1}(a_{i,t+1}, h_{i,t+1}, I_i)]$ to change in the subsequent period. Equating the baseline value function with the counterfactual (next period's health stock with the shock) implicitly defines the WTP to reduce the expected reduction in health. This unconditional WTP measure can be further decomposed into the WTP to avoid the reduction in health (conditional on survival) and the WTP to avoid the associated increase in mortality risk (conditional on health).

I calculate the unconditional WTP for a change in health as an equivalent variation measure of the reduction in assets that would be required for the individual to be indifferent between the two scenarios. Let, $V^*_{i,t}(a_{i,t}, h_{i,t}, I_i)$ be the counterfactual value

with the changed health state:

$$V_{i,t}(a_{it} - WTP_{i,t}, h_{it}, I_i) = V^*_{i,t}(a_{it}, h_{it}, I_i)$$

$WTP_{i,t}$ denotes the unconditional WTP for the change in health, i.e the reduction in assets that makes the individual indifferent between the status quo and a counterfactual deterioration in health. Then the value of statistical illness (VSI) for avoiding a particular disease can be defined as:

$$VSI_{i,t} = \frac{WTP_{i,t}}{q_t}$$

Thus, VSI is the age t willingness to pay for avoiding a disease that caused the decline in the health stock.

To decompose this unconditional measure into the WTP for conditional improvements in health and mortality, let $\tilde{V}_{i,t}(a_{it}, h_{it}, I_i)$ be the counterfactual value function with the changed health state next period but the survival probability fixed at its baseline level.

$$V_{i,t}(a_{i,t} - WTP^{morb}_{i,t}, h_{i,t}, I_i) = \tilde{V}_{i,t}(a_{i,t}, h_{i,t}, I_i)$$

This equality isolates the WTP for an improvement in conditional morbidity risk ($WTP^{morb}_{i,t}$).

Hence, the willingness to pay to avoid illness conditional on the survival probability, which I define as "value of conditional statistical illness" (VCSI), is defined as:

$$VCSI_{i,t}\frac{WTP^{morb}_{i,t}}{q_t}$$

The associated WTP to avoid the decline in mortality caused by the illness can be derived from the difference between the two WTP measures:

$$WTP^{mort}_{i,t} = WTP_{i,t} - WTP^{morb}_{i,t}$$

Note that this is the average WTP. The marginal WTP or VSL, can be recovered by using the difference in survival probabilities between the two scenarios be $\Delta\Phi_{i,t}$:

$$VSL_{i,t} = \frac{WTP_{i,t}^{mort}}{q_t \Delta\Phi_{i,t}}$$

3.7.4 WTP to Reduce the Risk of Chronic Obstructive Pulmonary Disease and Heart Attacks

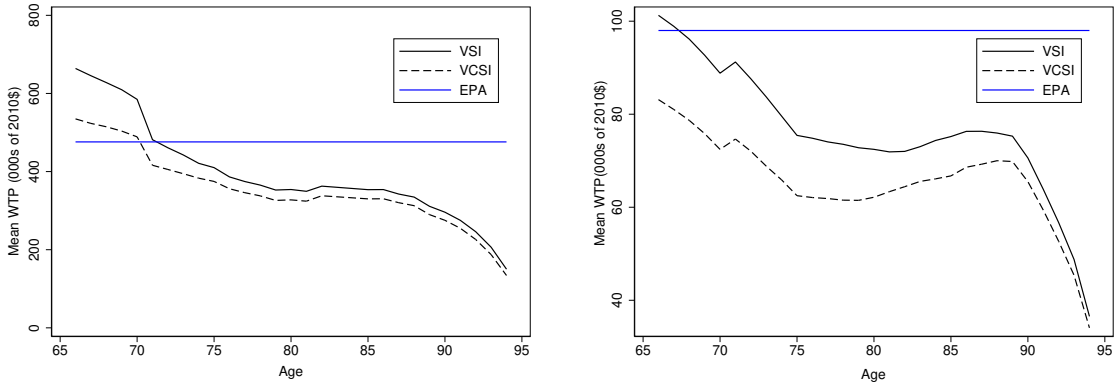
To demonstrate the model's implications for the value of reducing morbidity risk, I calculate the VSI and VCSI profile for common diseases targeted by public policies. Specifically, I consider diseases that are mitigated by EPA regulation of air pollution. This allows me to compare my WTP estimate to those used by the EPA in their benefit-cost analysis of the Clean Air Act. Chronic Obstructive Pulmonary Disease (COPD) and acute myocardial infarction (AMI, i.e., heart attacks) are common morbidities that represent the two largest components of the EPA's estimated morbidity benefits of the Clean Air Act. To map these conditions, into the measure of health in the model, I first calculate the age-specific probability of diagnosis of each condition and the marginal impact on proxy health measures. Combining the probability and marginal impact, I calculate the expected reductions in the latent health measure in the model using the estimated parameters of the measurement system. The age-specific expected reduction in health measure is then introduced into the model through the intercept of the health production function as shocks.

COPD is a group of lung diseases that includes Chronic Bronchitis (CB) and Emphysema. [26] EPA's calculation of morbidity benefits focuses on CB only. Reductions in incidences of CB contributes more than half of the total morbidity benefits of the Clean Air Act (CAA). The EPA uses an estimate of $476,000 (2010 $) per avoided

[26] Both CB and Emphysema are lung conditions that block airflow and creates breathing difficulties.

case. [27] Since, my data doesn't allow me to identify CB and Emphysema separately from COPD, I focus on COPD and in Section 3.8, I transfer the estimates of VSI and VCSI for COPD to calculate morbidity benefits of reduced cases of CB.

Figure 3.9: The WTP for Disease Prevention for COPD and AMI Conditional on Survival



(a) VSI and VCSI for COPD (b) VSI and VCSI for AMI

Notes: The figure plots the value of disease prevention for two diseases: COPD in panel 3.9a and AMI in panel 3.9b conditioning on surviving the onset of these diseases. The solid line illustrates the value of an avoided case of the disease and constitutes the WTP to avoid changes in health and mortality induced by the disease. The dashed line show the decomposition of this WTP into the WTP to avoid the change in quality of life holding mortality risks constant. difference between the two lines produce the WTP for the change in mortality risks. The solid blue line shows the WTP estimates used by EPA. All WTP values are adjusted to 2010 $.

Figure 3.9a shows the life-cycle profile for the WTP to avoid a case of COPD. The figure highlights several important features. First, the solid black line shows the average total WTP to avoid a case of COPD conditional on surviving the onset of COPD. At age 67, the estimated WTP to avoiding a case of COPD (or VSI) is

[27]The EPA's estimates are derived from two studies: Viscusi *et al.* (1991) and Krupnick and Cropper (1992). Viscusi *et al.* (1991) reports the WTP to avoid a severe case of CB while Krupnick and Cropper (1992) estimates the elasticity of WTP with respect to the severity of CB. Since the average pollution-related CB case is not severe, the EPA adjusts estimates from the literature by making assumptions about severity of an average pollution-related CB case, the WTP to avoid a severe case of CB, and the elasticity of WTP with respect to severity.

$650,000, which is approximately 40 percent higher than the measures used by the EPA (solid blue line). Second, the WTP declines over the life-cycle, with the sharpest decline observed at younger ages. This is intuitive because COPD is irreversible so that the deterioration in expected remaining lifetime health is greater at younger ages. Third, my estimates suggest that the EPA overestimates WTP for seniors beyond age 70. Lastly, decomposing my WTP estimates into the WTP to avoid the reductions in quantity and quality of life associated with increased mortality and conditional morbidity reveals WTP for mortality accounts for about 21 % of my estimates, and this share gradually declines as people age. The difference between the solid and the dashed lines in Panel 3.9a represents the willingness to pay to avoid the change in survival probability induced by the disease.

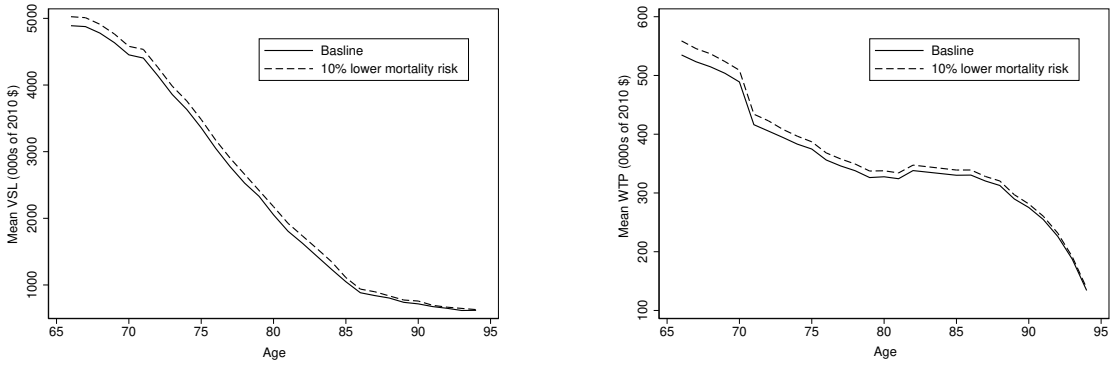
Acute Myocardial Infarction (AMI) constitutes approximately 30% of the EPA's estimated morbidity benefits from regulating air pollution. The EPA sets the value per avoided case of (non-fatal) AMI at about $91,000 (2010 $). They arrive at this value by averaging the estimates of discounted direct medical expenditure over a 5-year period following a heart attack. Figure 3.9b shows my WTP estimates for an avoided caseof non-fatal AMI. The average WTP is around $98,000 at age 67. The VSI per avoided case generally declines to around $40,000 at age 95 with a slight increase between ages 81 and 90. [28] Toward the end of the life-cycle, the VSI exhibits a rapid decline as individuals decumulate their wealth and have fewer years life-years left. Decomposing WTP into conditional morbidity and mortality implies that mortality accounts for a fifth of the total WTP at age 67, and this fraction declines as people age.

[28]The rise towards the end of the life-cycle mainly reflects the severity of the impact on health stock caused by a heart attack relative to earlier years.

3.7.5 Dynamic Complementarity between Health and Mortality

Murphy and Topel (2006) hypothesizes health improvements tend to be complementary. Their calibration exercise suggests that mortality improvements between 1970-2000, raised the value of a 10% reduction in all-cause mortality post 2000 by 18%. This finding has important implications for policies that target health and longevity, such as, the Clean Air Act. Since past improvements in health raise the value of further progress, the WTP for reductions in morbidity and mortality can be endogenous to policy. Ignoring this form of dynamic complementarity may cause the EPA and other federal agencies to understate the benefits of their policies.

Figure 3.10: Baseline VSL and VCSI with 10 Percent Reduction in Mortality Risk at all Ages



(a) VSL (b) VCSI for COPD

Notes: The solid line show the VSL estimates and VCSI for COPD estimated from the baseline model while the dashed line show the corresponding estimates with a 10% reduction in mortality at all ages

Figure 3.10 provides direct evidence to test the hypothesis in Murphy and Topel (2006). I use my estimates to run counterfactual simulations in which I reduce mortality risk by 10% at all ages. Figure 3.10a plots my VSL estimates in both the baseline model and the counterfactual scenario. A 10% reduction in mortality risk

increases VSL at age 67 by 3%, equivalent to $130,000. Intuitively, individuals place a higher value on reducing short-term mortality risks, because their life expectancy improved. Since consumption and health are complements, seniors are able to enjoy consumption over a longer life-span, hence the increase in VSL. Figure 3.10b shows the corresponding increase in WTP to to avoid COPD conditional on survival for COPD. A 10% reduction in mortality risk increases WTP by 4%, equivalent to $23,000 at age 67. Again, with a longer life span, individuals put higher values on avoiding debilitating disease like COPD. In both cases, the gains in value decline with age. Therefore, older seniors place lower values on future health progress, given current improvements.

3.8 Policy Implications of the Unified Approach to Valuing Health and Longevity

The conventional approach to calculating the benefits of policies that reduce mortality and morbidity risks is to assume a constant VSL for every death avoided (regardless of health, wealth, and age) and to add the resulting measures to the medical costs of avoided illnesses. My framework makes three important refinements to the conventional approach. First, my revealed preference estimates reflect how the VSL evolves with health, wealth and age beyond age 67. [29] Second, my framework accounts for static complementarity between health and mortality. For instance, my results suggest that 21% of the WTP to avoid a case of COPD reflects the WTP to avoid the increase in mortality risk caused by the disease. Third, my framework accounts for endogeneity of the VSL and VSI due to dynamic complementarity between WTP to reduce mortality and morbidity risks.

[29] Age-adjusted VSL has significant impacts on estimated benefits from a policy. For instance, EPA's use of senior discounts for VSL in the Clear Skies Initiative reduced estimated benefits by approximately 30% [US EPA (2002)]EPA does a single age-adjustment based on whether the individual is above or under 65 at the time of death. They provided a conversion factor for seniors of 0.63. Viscusi and Aldy (2007) for the same policy applied their age discounted estimates to arrive at a 40% lower benefits estimates compared to benefits estimated using EPA's constant VSL approach

To demonstrate how much these refinements can collectively matter for policy evaluation, I use my framework to reconsider the EPA's most recent cost-benefit analysis of the CAA (US EPA (2011)). The EPA estimated that the CAA's benefits in 2010 were $1.4 trillion (2010 $), generating a benefit-cost ratio of 25:1. The EPA's benefit estimate is driven by their assumption of a constant $8.1 million VSL (2010 $). In EPA's calculations, premature deaths avoided account for $1.3 trillion (95%) of the total monetary benefit, and 75% of these avoided deaths are assumed to accrue to people over age 65. Morbidity benefits account for only 4% of the total benefits. These benefits are calculated as the benefits from reduced incidences of diseases adjusting by fatality rates for each disease. The first column in Table 3.9 summarizes the EPA's reported total benefits.

In the second column of Table 3.9, I update the EPA's benefit calculations for people over age 65 by replacing the EPA's estimates for the values for mortality and morbidity with my estimates of VSL and VCSI for each age bins. Detailed accounting of benefits by age-bins are reported in the Appendix (Tables B.9 and B.10). [30] The values implied by my estimates are shown in bold. Strikingly, replacing the EPA's estimates with my estimates for seniors reduces the monetary benefits of the CAA by nearly half. While the morbidity benefits are reduced by 6%, the sharp reduction in total benefits occurs because the value from reduced mortality declines by roughly 50%. Nevertheless, this dramatic reduction does not change the EPA's conclusion that the CAA has positive net benefits. Under my calculations, the benefit cost-ratio is 12:1.

Importantly, my benefit estimates account for any endogenous increases in the value of health and the value of life that were realized by US seniors as of 2010

[30] I make no adjustments to EPA's computed benefits for people under age 65, nor do I make adjustments for the EPA's morbidity benefits from reduced hospitalizations or benefits from improvement in visibility and crop yields

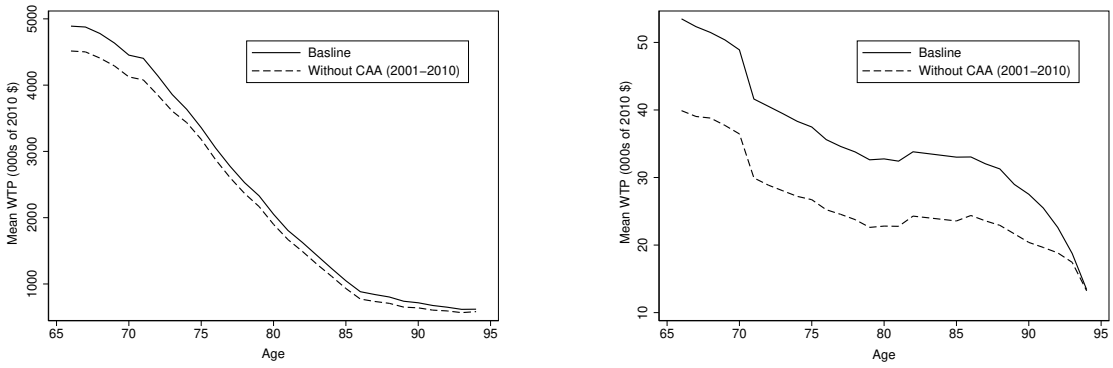
Table 3.9: Revisiting the Benefits of Clean Air Act for the Year 2010

Categories	Benefits (million $ 2010)		
	EPA	Baseline	W/O CAA
Mortality			
PM Mortality	1,300,000	**610,000**	**580,000**
Ozone Mortality	35,000	**16,000**	**15,000**
PM Morbidity			
Chronic Bronchitis	26,000	**25,000**	**23,000**
Non-fatal Myocardial Infarction	15,000	**13,000**	**11,000**
Other Morbidity Benefits	9,000	9,000	9,000
Ozone Morbidity			
Total Ozone Morbidity Benefits	1,300	1,300	1,300
Other Benefits	43,000	43,000	43,000
Total : All Categories	1,430,000	**720,000**	**680,000**

Note: The benefits in the second column are obtained using the age-distribution of premature deaths avoided and number of cases of each disease computed from the concentration-response functions reported by the EPA. The numbers in bold are computed using the estimates from this study, while all other numbers are taken from US EPA (2011). The other benefits include ecological and visibility benefits.

as a result of the morbidity reductions and longevity improvements caused by the Clean Air Act. To demonstrate the impact of this dynamic complementarity on the measured benefits, I repeat the benefit calculations after using my calibrated model to estimate the VSL and VCSI in a counterfactual scenario with zero mortality reduction induced by the CAA during the 2000s. According to estimates in US EPA (2011), the CAA reduced premature deaths by 110,000 in 2000 and 160,000 in 2010. Using linear interpolation to fill in the gaps in between these years, the CAA avoided 1,375,000

Figure 3.11: Baseline VSL and VCSI With and Without CAA



(a) VSL (b) VCSI for COPD

Note: The solid line show the VSL estimates and VCSI for COPD estimated from the baseline model while the dashed line show the corresponding estimates without the cumulative mortality reduction induced by CAA between 2001-2010.

premature deaths between 2001 and 2010. Figure 3.11a shows the reductions in VSL and VCSI for COPD for the counterfactual scenario where these deaths were not avoided and mortality rates were higher. The VSL at age 67 is now reduced by $375,000 (8%), while the VCSI for COPD declines by $130,000 (25%). These gains diminish with age. I use these counterfactual values to compute the benefits of the CAA in the third column of Table 3.9. Comparing the second and third columns of Table 3.9, reveals that mortality reductions induced by the Clean Air Act between 2001 and 2010, increased the policy's benefits by $40 billion (6%). Almost all this difference can be attributed to the increase in VSL.

3.9 Conclusion

This article developed a novel integrated framework for evaluating policies that simultaneously affect morbidity and mortality while accounting for static and dynamic complementarity between them. Estimating my model using administrative data on people over age 65 yielded several important empirical findings. First, the

conventional wage-hedonic estimates will tend to overstate the VSL for non-working adults aged 66 and above because I find clear evidence that the VSL declines with age. Second, I estimate how the value of disease prevention depends on the severity of the disease in terms of its effects on morbidity and mortality, using chronic obstructive pulmonary disease and acute myocardial infarction as examples. I find that approximately one-fifth of the value of preventing a disease like COPD is due to its effect of mortality. Lastly, I find that the Clean Air Act increased the value of a statistical life by approximately 8% (yielding $40 billion in benefits) in 2010 by increasing the expected quantity and quality of life for people over age 65. Therefore, dynamic complementarity in health accounts 6% of the total health benefits derived from the Clean Air Act.

Turning to future research, the framework developed in this study has potential to yield new insights in across a wide variety of contexts. For instance, my framework could be used to re-assess the distributional welfare effects of health insurance policies such as the Affordable Care Act [Black *et al.* (2019)] or Medicare [Khwaja (2010)]. It could also be used to evaluate the benefits of different types of investments in medical research distinguishing between the benefits of preventing and treating disease [Bauer *et al.* (2018); Rheinberger *et al.* (2016)]. Another challenge for future research is to consider how people value risk reductions experienced by others, given that public programs targeting health risks are typically financed through taxes. Prior studies have suggested that the external benefits of this health-focused altruism may be substantial [Smith (2007); Dickie and Gerking (2007); Jacobsson *et al.* (2005)]. In principle, the model developed in this study could be augmented to incorporate health altruism within the family for married couples.

Chapter 4

EFFECTS OF DEPRESSION ON ILLICIT DRUG USE IN YOUNG ADULTS: EVIDENCE FROM THE 9/11 TERROR ATTACKS (WITH FRANCESCO AGOSTINELLI AND ALEX RIVADENEIRA ACOSTA)

4.1 Introduction

The economic cost of health has been recently the focus of an important debate. In particular, stress-related conditions such as depression and anxiety have been associated with significant and rising economic costs. Furthermore, those conditions have the potential to trigger behaviors that could exacerbate them, lead to new ones, or even induce death. Greenberg *et al.* (2015) estimate that major depressive disorder (MDD) alone accounts for $210.5 billion (2012 U.S $) in 2010, 50% of which is attributed to direct medical and suicide related mortality costs. More than 60% of the direct costs is accounted by cormorbid conditions.

In the medical and epidemiological literature depression has been associated with various health conditions. Depression has been found to increase the risk of heart failure (Abramson *et al.* (2001); Williams *et al.* (2002)); increase complications for people with diabetes (De Groot *et al.* (2001)); increases risk of dementia Cherbuin *et al.* (2015); and elevate all-cause mortality (Pratt *et al.* (2016)). With regards to health behavior, the literature has emphasized that depression reduces healthy behaviors like exercising Wang and Yang (2013) and increased risky sexual behavior Averett and Wang (2012). Among youths, depression has been associated with higher rates of juvenile crimes (Cuellar *et al.* (2004)), and lower educational outcomes Currie and Stabile (2006); Fletcher (2007). One behavior of crucial relevance that is associated

with mental illness or depression is illicit drug use. There is high correlation between mental illness and drug use disorder. According to the Substance Abuse and Mental Health Services Administration (2017), in 2016, 28% of adults aged 18-25 who were suffering from any mental illness also had a past year substance use disorder [1] . Moreover, 35 % of adults aged 18-25, with serious mental illness also had a past year substance use disorder.

Illicit drugs remains a problem in the US with an estimated 31.8 million people aged 12 and above reported using some kind of illicit drug in the past month in 2018, corresponding to 11.7% of the population. The prevalence of the use of illicit drugs is highest among young adults and adolescent groups. Moreover, the rise in mortality of middle-aged (45-54) white Hispanics in the US has been mainly attributed to the rise in mortality from drug overdoses, suicides, and alcohol induced mortality which has been described as "death of despair"(Case and Deaton (2017)). The number of deaths from drug overdoses has also been on the rise since 2012 for different types of drugs like synthetic opioids, methamphetamine and cocaine with a total of 72,000 overdose deaths in 2017 (Centers for Disease Control and Prevention (2017)). A more alarming fact is that in 2016, annual prevalence for use of any illicit drug across different-age groups was the highest for college students (43 %) (Schulenberg *et al.* (2017)).The National Drug Intelligence Center (2011) estimated the total societal cost of illicit drug use to be $221 billion (2015 US $), two-thirds of which are losses in productivity.

Various factors have been put suggested as the contributing factors to the initiation of drug abuse. These factors include pressure from peer groups (Lundborg (2006)); cultural and social factors when individuals imitate others (Bandura (1986));

[1]Substance use disorder, as defined by National Alliance on Mental Illness, is abuse of any substance including tobacco, alcohol, drug that leads to clinical impairment or distress.

coping with stress in early adolescence (Wills (1986)); tobacco and alcohol as gateway drugs (Kandel (2002)); substitution of alcohol (Cameron and Williams (2001)); personal characteristics like impulsiveness (Perry and Carroll (2008)); and adverse family relationships. Additionally, mental health is cited as an important factor in stimulating the consumption of illicit drug use. For adolescents, evidence suggests that depression is associated with higher levels of smoking uptake and progression (Audrain-McGovern *et al.* (2011)) and increased risk for early life initiation of alcohol or illicit drug use (Tang and Orwin (2009)).

Both mental illness and illicit drug use often start in adolescence, and certain mental health conditions have been identified as risk factors that initiate illicit drug intake and later lead to substance use disorder (Baigent (2012)). For instance, some individuals rely on drugs for self-medication. [2] Moreover, brain changes caused by mental illness make the effect of drugs more rewarding, making it harder to quit (Santucci (2012)). Another mechanism that connects depression and illicit drug use is that depression can cloud out one's judgment and forces him to take rash actions like consuming drugs in periods of extreme negative sentiments, a phenomenon called 'negative urgency' (Pang *et al.* (2014))

While previous studies have associated depression with illicit drug use, the direction and magnitude of any causal relationship between them is yet to be established. Disentangling the causal effect of depression on drug consumption is challenging because of simultaneity bias. Against this background, I use a natural experiment in the form of a crisis that induced an increase in depression, to understand its effects on illicit drug use.

Specifically, I use the September 11 terrorist attack as a natural experiment that

[2]The self-medication hypothesis entered in the medical literature in 1987 after Khantzian (1987), which suggests certain drugs are chosen by individuals because they alleviate distress associated with depression for example cocaine

induced an increase in depression, to study its subsequent effects on illicit drug consumption. The 9/11 attack occurred in the middle of a survey that was collecting data of young adults, which included among other variables, a depression index and detailed information about drug consumption. The survey is the National Longitudinal Study of Adolescent to Adult Health (AddHealth), which is a nationally representative survey for US students in grades 7-12. The AddHealth cohort is tracked into young adulthood with in-home interviews, with the most recent survey happening in 2008. I use the third wave for this study which mainly includes young adults aged 18-28. Prevalence of past month illicit drug use is the highest for individuals 18-25 (23.9%) (Substance Abuse and Mental Health Services Administration (2017)). The unexpectedness of the event randomly split the sample into a treatment group (after 9/11) and a control group (before 9/11), which allow me to use one group as a counterfactual for the other.

My results suggest that 9/11 induced an increase in depressive symptoms as measured by an index based on a subset of questions in the Center for Epidemiological Scale (CES-D). The index provides an aggregate measure of subjective assessment of an individual's overall mental health with regards to anxiety, depression disorders, loss of concentration, fatigue, psychological functioning and states of happiness/sadness and is suggested to correlate well with the clinical measure of depression. I find a significant jump in the index for respondents interviewed after 9/11, compared to respondents interviewed before 9/11. I use this plausible exogenous shock to mental health to identify the causal effect of depressive symptoms on health behavior, such as consumption of illicit drug and binge drinking. The identification closely follows the work of Salguero *et al.* (2011) and Perlman *et al.* (2011), where they use the British Household Panel Survey to identify the effects of 9/11 on the mental health of British residents. Their results suggest large spillover effects on residents of the

United Kingdom from the attack in New York. In my study, I additionally look at the effect on health behavior in an instrumental variable framework.

Four key findings emerge. First, comparing individuals interviewed before and after 9/11, I find that for individuals who experienced an increase in depressive symptoms the likelihood of consuming any type of illicit drug is higher. The instrumental variable results are an order of magnitude larger than the OLS results, and the direction of the estimated effects suggest that these drugs are used as a coping mechanism for individuals with depression. The significant increase in the likelihood of illicit drug consumption persists when I use a different time window in the construction of the instrument by excluding the first week after the event or using an alternative measure of depression. I also confirm ,that the results are not driven by seasonal effects that might induce a spike in drug consumption during the month of September. Second, I find that these effects of depression are heterogeneous across different types of drugs. The largest effects are observed for marijuana, which is easy to obtain and less expensive compared to other drugs like cocaine. I also document an increase in the frequency of binge drinking (consuming 5 or more drinks per day) following an increase in depressive symptoms.

Next, I investigate heterogeneity across different socio-economic and demographic groups. The effects of depressive symptoms on illicit drug use are the highest for white females and young adults over the age of 21. The results also point towards a dynamic behavioral change in substance abuse i.e. depressed young adults tend to substitute alcohol with drugs as they grow older. Stratifying the sample by education, I find college students with greater cognitive abilities are also more likely to use drugs to deal with depressive symptoms compared to non-college going students or lower ability individuals.

Last, I consider the effects of illicit drug use on outcomes relating to productiv-

ity. Specifically, I consider two productivity outcomes: sleeping at school or work and missed school or work days. Both outcomes have implications for education attainment and efficiency in the workplace. Identifying the causal effects of drug consumption on these productivity outcomes is complicated by the possibility that, depression may also independently affects these outcomes. With two endogenous variables (depression and drug consumption) and one instrument, I provide analytical bounds of the effects under the maintained assumption that both depression and drug consumption affect productivity weakly negatively. On average, both depressive symptoms and illicit drug use have significant impacts on sleeping during school/work. The effects are much larger for students compared to non-students.

Overall, this paper makes two important contributions to the literature. First, it identifies the impact of mental illness on induction to drugs. While, the epidemiological literature have extensively studied the inter-relations between mental illness and drug use, (Kessler *et al.* (1996); Merikangas *et al.* (1998); Conway *et al.* (2006); Glantz *et al.* (2009); Swendsen *et al.* (2010)), a causal relation is yet to be established. To my knowledge, only one prior study in the economics literature studied the effect of mental illness on demand for addictive goods. Saffer and Dave (2005), using family history of mental illness as an instrument, estimates the impact of mental illness on demand of cocaine, tobacco and alcohol. I add to this literature by improving on the identification strategy considering potential impacts on a wide range of drugs and the implications on productivity measures. [3]

Secondly, this study identifies a relatively inexpensive channel to reduce drug consumption. Drug consumption is highly costly with total societal cost estimated to be $ 221 billion (2015 US $) National Drug Intelligence Center (2011). Additionally, the

[3]Since cocaine is more expensive compared to other drugs and my focus is on young adults who are likely to be budget constraint,I assess the impact on drugs which are more relevant for the age-group in consideration. Illicit drug use is also the highest for this age-group.

government estimated spending is set at $ 26.6 billion (2015 US $) for federal drug control (National Drug Control Budget, 2018). Drug abuse is also related to poorer educational outcomes, lower income, greater welfare dependence and unemployment (Fergusson and Boden (2008)). In terms of health indicators, deaths from drug overdoses, suicides and alcohol are the prime contributors to the rise in mortality rates among mid-life US adults (Case and Deaton (2017)). Drug abuse is also associated with higher crime rates (Grogger and Willis (1998)) and can also have large enduring impacts Evans *et al.* (2005). This study suggests that having policies that subsidize the treatment of depression or increasing access to mental health care can provide important complementary benefits by curbing substance use.

Third, the identification strategy contributes to the literature on how terrorist attacks affect private and social outcomes. The identification strategy follows from the work of Averett and Wang (2012), Wang and Yang (2013) and Averett and Wang (2016), who also used the same dataset and the 9/11 attack as an instrument. Wang and Yang (2013) and Averett and Wang (2012) instrumented depression in order to identify and quantify its effects on weight-related and risky sexual behaviors respectively. Averett and Wang (2016) instrumented alcohol abuse with 9/11 attack to estimates causal effects of alcohol abuse on intimate partner violence. Focusing on illicit drug use and alcohol abuse, this study contributes by focusing on an important first order effect of depression.

The paper is organized as follows. In Section 2, I describe the data and describe how the timing of the survey facilitates econometric identification using the exogenous variation. Section 3 provides details on the identification strategy and analyzes the impact of depression on drug consumption using the 9/11 shock as an instrument. Section 4 discusses the results from my baseline analysis and presents additional results on heterogeneity. Section 5 presents partial identification strategy to estimate

the causal effect of drug consumption on productivity in school/workplace. Section 6 concludes.

4.2 Data

My empirical analysis is based on the National Longitudinal Study of Adolescent to Adult Health (AddHealth) data. AddHealth started as a nationally representative survey for U.S. students enrolled in grade 7-12 during the 1994-1995 school year. The survey adopted a school-based sampling design. The first in-school interview (Wave I) was administrated to 90,118 children in roughly 130 private and public schools. A random subpopulation of children (20,745 children) were selected for the following in-home interview; and a total of three additional in-home follow-up interviews were administrated from 1996 to 2009.

My study will focus on the third in-home interview (Wave III), which spans from July 2001 to April 2002. [4] In particular, I focus on the period immediately before and after the 9/11 attack, when more than 4,000 people were daily interviewed within a 30 days span from the incident. According to Schulenberg *et al.* (2017), illicit drug use is more prevalent among individuals in their early adulthood, and Wave III, comprising of respondents aged 18-23 appears to have the population of the relevant age-group. Drug use or alcohol abuse in early adulthood can be a potential health hazard and is associated with future drug abuse disorders in future adulthood (D'Amico *et al.* (2005)); risky sexual behavior interms of sexually transmitted diseases (Howard and Wang (2006)) and suicide (Rowan (2001)). Since my identification strategy relies on utilizing the exogenous variation provided by the 9/11 event, the availability of interviews before and after 9/11 allows for identification. Since 9/11 was an unexpected event, it randomly split the sample into two groups: treatment

[4]Few pilot interviews were conducted in April and May 2001

and control. My identification strategy then relies exclusively on the fact that 9/11 was unanticipated and did not alter the organization of the survey. I discuss these assumptions below in greater details.

Add Health was originally designed to assess both psychological and physical well-being of sampled adolescents. Hence, the survey includes several depression-related questions based on the Center for Epidemiologic Studies Depression Scale (CES-D). The original CES-D is composed by 20 items of self-reported feelings during the last 7 days (see Radloff, 1977). Similarly, Add Health included one entire section of questions called "*Social Psychology and Mental Health*" where respondents were assessed with with a subset of the 20 questions in CES-D. [5] Table 4.1 shows the subset of questions relating to CES-D that were asked in Wave III of AddHealth surevy. Answers to each questions were recorded on the four point 0-3 scale: "0 : never or rarely; 1 : sometimes; 2 : a lot of the time; 3 : most or all of the time." I constructed an index out of these questions adding up the scores on each item's responses and then standardized the total score by its population standard deviation (for results interpretation). The range of total score is 0 to 27 where higher scores imply greater prevalence of depressive symptoms. The score order in Table 4.1 reflects how the responses to individual questions affect the overall index of depression.

Additionally, Wave-III contains detailed information about drug and alcohol consumption of respondents. Specifically, I used self-reported information on drug consumption to construct various indicator variables of whether individuals used different type of drugs during the last 30 days, such as marijuana, cocaine, methamphetamine, LSD, ecstasy or heroin. Finally, I also use information of alcohol consumption using the survey records of number of times in the last two weeks respondent had 5 or more

[5] All but question no. 6 in Table 4.1 were the same questions as in CES-D. Question 6 appears differently in CES-D: "You felt everything you did was an effort?"

Table 4.1: Subset of CES-D Items in AddHealth Survey

Description	Score Order
How often was each of the following things true during the past seven days?	
1. You were bothered by things that usually don't bother you	Positive
2. You could not shake off the blues, even with help from your family and your friends	Positive
3. You felt that you were just as good as other people	Negative
4. You had trouble keeping your mind on what you were doing	Positive
5. You were depressed	Positive
6. You were too tired to do things	Positive
7. You enjoyed life	Negative
8. You were sad	Positive
9. You felt that people disliked you	Positive

drinks in one day.

The Add Health data also provides information on demographics for each interviewee. Specifically, I have age, gender, race, earnings, personal income, marital information, household income and also the sum of the individual's and spouse's income. Moreover, I also use information about receipts of cash and non-cash benefits in the form of housing assistance, unemployment insurance, Aid to Families with Dependent Children(AFDC) etc. This information helps me to account for potential omitted variable bias, check robustness of my estimates and look at heterogeneity in treatment effects between certain groups of the population.

4.2.1 Summary Statistics

The main sample is constructed by restricting the Wave III data to interviews conducted 30 days before and after the event of 9/11. [6] Table 4.2 provides the demographic characteristics for the main sample (30-days) which contains 4.904 individuals. Out of this, 47% of the individuals were interviewed after the terrorist attack on 9/11. This reflects the fact that 9/11 did not have a considerable effect on AddHealth surveys and interviews were conducted according to the initial plan. The sample is composed of mainly single females and the average person is approximately 22 years old. In terms of racial composition, 79 % are white and the rest are black. A majority of the individuals (94 %) earned income from wages/salaries or from self-employment. The average personal income stands at $ 12,500 which comprises mostly of earnings. On average, around 5 % of the sample received some sort of government benefits in the form of Aid to Families with Dependent Children (AFDC), food stamps, housing assistance etc. Around half of the sample received some sort of financial assistance from friends and family. The average family income is around $60,000, around half of which is the average cumulative income of the individual and the spouse. Appendix Table B.11 compares the estimating sample with the entire sample in Wave III.

4.3 9/11 Terrorist Attack and Depression

The main empirical challenge in identifying the causal effect of depression on drug consumption is the *simultaneity* or *reverse causality* problem: changes in depression may induce changes in decision to consume drugs, which in turn, could affect depression. There is also the problem of measurement-errors in self-reported measure of

[6] To investigate robustness of my estimates, I also run the estimates on a sample of interviews constrained to 15 days before and after 9/11

Table 4.2: Differences in Demographics Between Those Interviewed Before and After 9/11

Observed Variables	Mean	Difference	Standard Error	N Obs
Age	21.660	-0.011	0.049	4,904
Male(%)	0.413	-0.049***	0.014	4,904
White(%)	0.791	-0.018	0.026	4,904
Black(%)	0.213	-0.003	0.013	4,904
Native America(%)	0.063	-0.001	0.009	4,904
Asian(%)	0.107	-0.016	0.012	4,904
Number of Times being Married	0.184	-0.008	0.011	4,901
Received Wage Income(%)	0.941	0.029	0.018	4,904
Received Dividend Income(%)	0.205	0.006	0.018	4,904
Received Food Stamps(%)	0.061	0.017	0.014	4,904
Received AFDC(%)	0.045	0.017	0.012	4,904
Received Housing Assistance(%)	0.032	0.013	0.010	4,904
Received Unemployment Insurance(%)	0.065	0.009	0.012	4,904
Received Alimony(%)	0.045	0.021	0.013	4,904
Received Support from Family / Friends(%)	0.463	-0.011	0.018	4,904
Total Personal Income($)	12,446	465	411	4,619
Earnings($)	11,500	557	458	4,216
Total Household Income($)	60,325	997	2,373	1,948
Total Couple Income($)	28,793	1,355	1,358	1,396

Notes Spending measures are adjusted to year 2010 US dollars using the CPI. The third column report the difference in means of variable before and after 9/11 and is created by running regressions of each variable on an intercept and a dummy variable "9/11" which equals 1 if the interview is taken after 9/11. The coefficients on the dummy "9/11" are reported in the table. Standard errors are clustered by birth-year. Asterisks indicate statistical significance at the 1% (***), 5% (**) and 10% (*) levels.

depression, where measurement errors are correlated with the computed depression index. In this section I describe my strategy to identify the causal effects of depression on drug consumption using 9/11 as an exogenous shock to depression. Several studies have shown that catastrophic events usually induce depression. Green (2011) documents increase in prevalence of depressive symptoms after 9/11, among students enrolled in an university far away from the attack area. Vlahov *et al.* (2002) finds increased use of alcohol and marijuana following 9/11 attacks with people reporting increases are more likely to experience post-traumatic stresses. Schlenger *et al.* (2002) records significantly higher levels of Post traumatic Stress Disorder (PTSD) among residents of New York, 1-2 months after 9/11 compared to other metropolitan areas. [7]

Previous studies were faced with the challenge of investigating whether a certain catastrophic event actually induced substantial increase in depression levels, because of the lack of pre-event data. In this study we could investigate this, because of the availability of survey data both before and after the 9/11 terrorist attack. I discuss the relevance and validity of this instrument before proceeding to discuss the results.

4.3.1 Identification Strategy

To provide evidence on the relevance of the instrument, I first investigate whether the 9/11 attack generated a significant increasing in depression using the following regression:

$$dep_i = \gamma_0 + \gamma_1 9/11 + X_i'\gamma_2 + \nu_i \tag{4.1}$$

[7] There are several other studies that tried to investigate the impact of terror attacks like Galea *et al.* (2002); Silver *et al.* (2002) for 9/11 attack and North *et al.* (1999) for Oklahoma City Bombing of 1995. However, since most of these studies report post-attack data, it is difficult to ascertain whether these findings reflect the effect of the terrorism event.

where dep_i is the standardized depression index based on CES-D described above. The instrument, 9/11 is a dummy variable equal to 1 if the individual was interviewed after the 9/11 attack and 0 otherwise. X_i is a rich vector of individual characteristics which includes age, gender, race, Peabody Picture Vocabulary Test (PPVT) test score, years of education and an index of religiosity. This is the also first stage of the two stage least-square (2SLS) estimation.

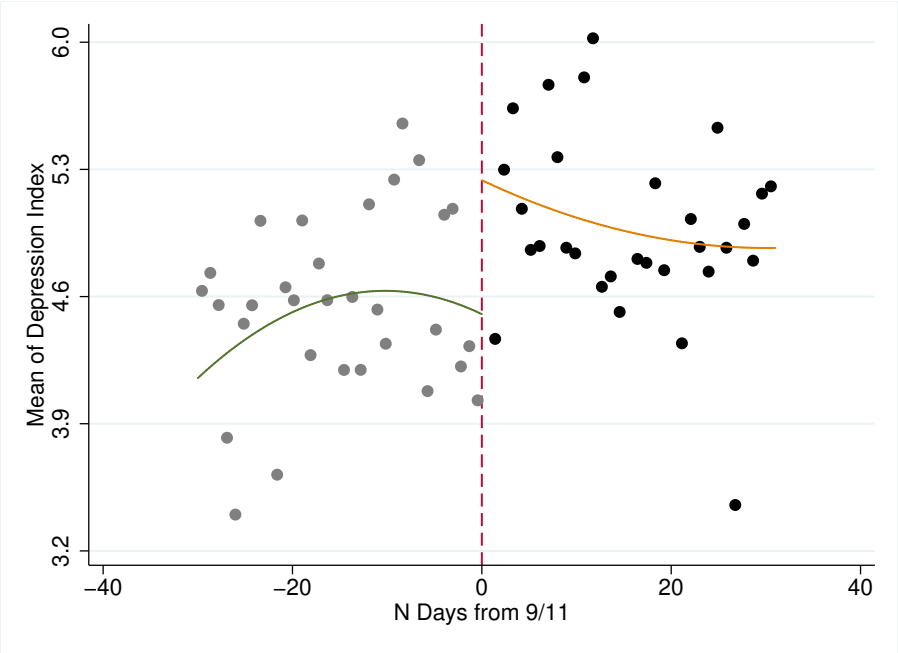
Using the predicted depression, ($\widehat{dep_i}$) from equation 4.1, I estimate the second stage,

$$drug_i = \alpha_0 + \alpha_1 \widehat{dep_i} + X_i' \alpha_2 + \epsilon_i \tag{4.2}$$

where $drug_i$ is a dummy variable equal to 1 if the individual has consumed drugs during the last 30 days and 0 otherwise. I estimate the model using seven different outcomes for drug consumption in the last 30 days : whether the individual consumed any type of drug, whether the individual consumed specific types of drugs such as marijuana, cocaine, methamphetamine, drug intake through injections and a composite category of other types of drugs including LSD, ecstasy, heroine etc and biweekly frequency of 5 or more alcoholic drinks.

Using 9/11 as an instrument for depression requires, first, that 9/11 created an increase in depression, and second, that 9/11 is uncorrelated with any other determinants of drug consumption. Figure 4.1 provides the evidence that respondents interviewed on or after 9/11 exhibited an increase in depressive symptoms. The scatter plots depict the CES-D, averaging over all individuals on each interview dates within a period of 30 days before and after 9/11. Separate best-fit curves drawn before and after the event suggests there has been a substantial increase in depression symptoms immediately after the 9/11 attack. Furthermore, I formally test the statistical significance of this increase using regression analysis in Section 4.4.

Figure 4.1: The CES-D Index Before and After 9/11 (Within 30 days)



Notes: The y-axis plots the continuous CES-D index and the x-axis shows each dates within 30 days before and after the 9/11 terrorist attack. Each scatter plot average all the CES-D scores for all individuals in a particular interview date. The dashed vertical red line indicates the date of September 11, 2001. The curves are best-fit curves across all the scatter points before and after 9/11.

The second requirement of validity is untestable. However, the 9/11 could be seen as a random treatment since it occurred unexpectedly during the middle of a survey. Crucially, the timing of the survey did not obey any selection criteria, so the fact that some people were interviewed before and after 9/11 could be considered as good as random. Hence, my identification strategy relies on the fact that those interviewed before and after 9/11 are comparable, with the only difference between both groups being the timing of the survey. I test if that was the case by comparing both groups in the 30 day window on several observables. The balance test reported in Table 4.2 show that both the treatment group (interviewed on or after 9/11) and control

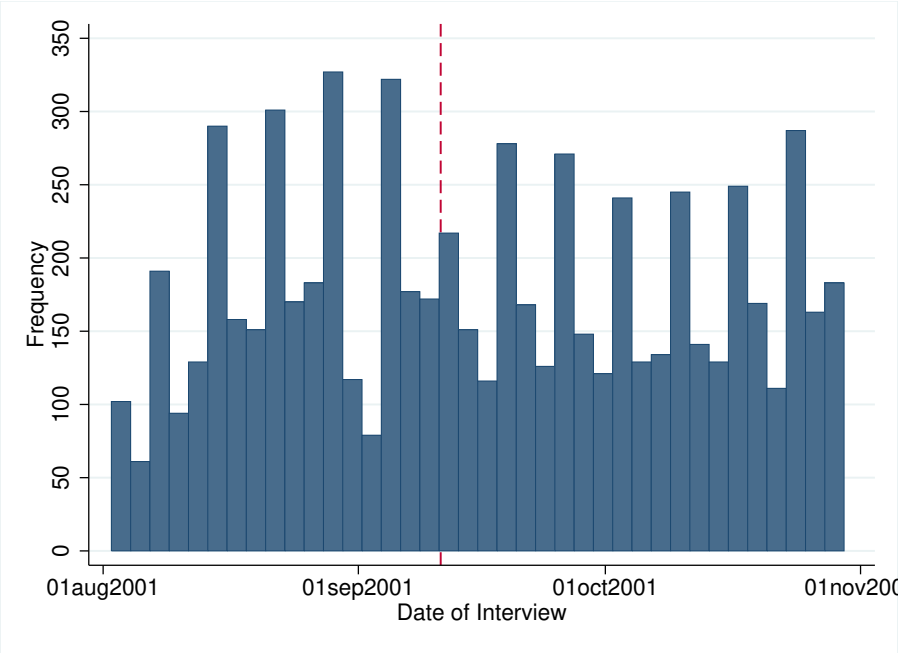
group (interviewed before 9/11) exhibits no statistical difference in both economic and demographic characteristics with the exception of the male variable. This finding justifies the exogeneity of the assignment of treatment, 9/11 reflecting the fact that the event was unexpected. Appendix Table B.12 additionally suggests that the two groups exhibit statistically significant differences in illicit drug use and alcohol intake. The group interviewed after 9/11 are more likely to consume drug mainly marijuana and cocaine and are also likely to engage in binge drinking (consuming 5+ alcoholic drinks per drink).

Another concern related to the 9/11 shock could be that it might have affected the regular execution of the survey by postponing it, changing the sample, or redirecting the survey to other groups. Such reorganization could threaten my identification strategy if the treatment and control groups turn to be heterogeneous due to a direct effect of the shock on the survey's structure, instead of through the proposed mechanism, namely, drug consumption. While the balance test results in Table 4.2 suggests that this might not be the case, I provide further justification, that the 9/11 event had no impact on the interview routine. Specifically, I test to see if 9/11 generates any organizational shock in the surveying procedure of Wave III. Figure 4.2 plots the frequency of interviews conducted before and after 9/11. The vertical red line indicates the day of the attack i.e. September 11,2001. The figure suggests interviews were not postponed or cancelled after 9/11, signifying that the event did not impact the survey routine which could otherwise have biased my estimates.

Finally, the exclusion restriction could be violated if the 9/11 shock affected drug consumption through a channel different than depression. I conduct a range of robustness checks to provide evidence that the exclusion restriction is satisfied.

The baseline estimates of the effect of depression on drug consumption is based on a 30-day time window used in the construction of the instrument. I addition-

Figure 4.2: Distribution of the Interview Dates Around September 11, 2001



Notes: The figure plots the frequency of interviews conducted just before and after the event of September 11, 2001 (indicated by the vertical red line).

ally consider a time frame of 60 days; the instrument takes the value of 1 for any observation coming from interview made within 30 days prior to 9/11 attack and a value of 0 for interviews 30 days following the day of attack. I investigate in the subsequent analysis whether the results of this study is robust to the alternative time boundaries used in the construction of the instrument. Another concern that may arise with the identification strategy is that the exogenous variation in depression were not induced by the unexpected event of 9/11 but due to an unobserved seasonal effect of the month of September. In order to address this concern, I ran a placebo test to investigate the effect of the month of September by replicating the analysis with information from Wave IV of AddHealth data set. Thus, I choose the cut-off date to be September 11, 2008. If there are any unobserved seasonal effect, then the

estimates using Wave IV would show significant impact of 9/11 on illicit drug use in the year 2008. Lastly, I consider an alternative measure of depressive symptoms to the continuous CES-D measures used for the baseline estimates. This alternative depression measure is an indicator of whether the individual exhibited any depressive symptoms at all i.e, it takes the value of 1 if the composite CES-D score is positive and zero otherwise. Thus a value of 1 for the alternative binary depression measure indicates the individual showed at least some depressive symptoms relating to one of the 9-items used to construct the CES-D scores.

4.4 Results

Table 4.3 shows both the 2SLS and OLS estimates for the 30-day sample and for seven different outcome variables. For each specification I control for gender, race, cognitive abilities using years of education and PPVT scores [8] and an index for religiosity. Column (1) shows the results for whether the individual consumed any type of illicit drug and Columns (2)-(6) reports the same for specific types of drugs. Panel A documents the first stage relationship between the 9/11 shock and depression as measured by the parameter γ_1, while the second-stage estimates of α_1 are reported in Panel B. Panel C exhibit the OLS results disregarding endogeneity of depression. The sample size differs across columns because of missing information for the dependent variable (less than 1%). Unabridged estimates of Panel A -to C are presented in Appendix Tables B.14 and B.15.

The first-stage estimates of γ_1 in Panel A suggests that due to the 9/11 terrorist attack, there was statistically significant increase in depressive symptoms, as mea-

[8]The Peabody Picture Vocabulary Test (PPVT) is an age standardized test, intended to provide a measure of intelligence through the measurement of school aptitude and verbal ability. The Add Health dataset includes a computerized and an abridged version of the original test, provided both the raw scores and scores standardized by age (for technical details, see Halpern *et al.* (2000)).

sured by the CES-D index. On average, the 9/11 terrorist attack lead to an 0.12 of a standard deviation increase in depressive symptoms. Furthermore, a first-stage partial F-statistic value of around 17, relieves concerns relating to weak instrument as per Staiger and Stock (1997). The results from the first stage are consistent with the jump in mean depression index immediately after 9/11 shock illustrated in Figure 4.1 and also confirms the findings of studies focusing on mental health impacts following 9/11 or other terror attacks (Schlenger *et al.* (2002); Perlman *et al.* (2011); Salguero *et al.* (2011); Green (2011)). In Appendix Table B.13, I show how 9/11 affects the different components of the CES-D index. Individuals on average reported greater anxiety, depression, concentration problem and feeling of sadness following the 9/11 terrorist attack.

Panel B show the estimates from the second-stage. The baseline estimate of γ_1 suggests that an increase in depressive symptoms triggered by the terrorist attack of 9/11, increased the probability of drug consumption. Comparing individuals interviewed 30 days before and after the 9/11 terrorist attack, I find the probability of any illicit drug consumption increases by 32 percentage points for a standard deviation increase in depressive symptoms. Comparing the results with the OLS estimates reported at the bottom of the table in Panel C, the instrumental variable estimates correspond to an order of magnitude larger effects of depressive symptoms. Combining estimates from both stages, the 9/11 attack increased illicit drug use by 3.8 percentage points.

Table 4.3: 2SLS Estimates of the Effects of Depressive Symptoms on Illicit Drug Consumption and Binge Drinking

	Outcomes: Drug Consumption last 30 days						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Ever Consumed Any	Marijuana	Cocaine	Methamphetamine	Inject	Others	Alcohol
	Panel A: First Stage (IV)						
Terror Attack 9/11	0.119	0.119	0.118	0.118	0.118	0.119	0.119
	(0.028)	(0.028)	(0.028)	(0.028)	(0.028)	(0.028)	(0.028)
F-Stat	17.500	17.529	17.390	17.390	17.402	17.615	17.504
	Panel B: Second Stage (IV)						
CES-D	0.321***	0.309**	0.066	0.026	0.027*	0.154**	1.166**
	(0.122)	(0.119)	(0.045)	(0.032)	(0.016)	(0.063)	(0.524)
	Panel C: OLS						
CES-D	0.035***	0.034***	0.006**	0.004*	0.003*	0.005	0.099***
	(0.006)	(0.006)	(0.003)	(0.002)	(0.001)	(0.003)	(0.030)
N Obs	4,634	4,635	4,646	4,648	4,648	4,646	4,625

Notes The dummy variable, "9/11" is used as an instrumental variable to estimate the impact of depressive symptoms (CES-D index) on whether the individual consumes any illicit drug, any specific type of drug like marijuana, cocaine or methamphetamine etc, and biweekly frequency of 5+ alcoholic drinks per day. In each columns, I control for: gender, race, cognitive abilities (PPVT test score), years of education and an index of religiosity. The others variable is a composite category indicating whether the individual consumes LSD, ecstasy, Heroin etc. Asterisks indicate statistical significance at the 1% (***), 5% (**) and 10% (*) levels. Robust standard errors are in parenthesis.

One would expect that the OLS estimates would be upward biased given the fact that depression triggers drug consumption which in turn increases can cause depressive symptoms. The IV estimates in Table 4.3 suggest OLS estimates to be downward biased. An explanation which could be consistent with the finding is that, individuals use illicit drug as a coping mechanism with depression and therefore illicit drug use providing them with temporary relief from depressive symptoms. Indeed, according to National Institute on Drug Abuse (2010), individuals with some form of mental disorders abuse drugs inorder as a form of self-medication in order to ease the symptoms of the underlying disorder, to tackle with difficult emotions or to change their moods. Another explanation could be that OLS estimates are attenuated due to measurement error in depression. The index-based measure is constructed from a survey, which could generate error in measuring the component of "depression" that is relevant for decisions about drug use.

The effects are also heterogeneous across different types of drugs. The estimates of α_1 in Column (2)-(6) of Table 4.3 suggest that the effects of depressive symptoms on drug consumption are more heavily concentrated in marijuana. On average, a standard deviation increase in depressive symptoms increases probability of marijuana use by 31 percentage points. This is consistent with the stylized fact that marijuana is the most affordable and available drugs among the class of illicit drugs considered in this study. The second largest effect is observed to be for mostly "club drugs", which is the composite category and includes LSD, Ecstasy etc., while there is only suggestive evidence for other drugs like cocaine, methamphetamine and drugs consumed through injections. [9] According to the 30-day prevalence rates in Schulenberg *et al.* (2017), marijuana and ecstasy are the most abused drug by individuals aged 19 to 30 years.

[9]The National Institute on Drug Abuse classifies club drugs as drugs "used by teenagers and young adults at bars, nightclubs, concerts, and parties". These include GHB, Rohypnol, ketamine, MDMA (Ecstasy), methamphetamine, and LSD (acid).

The results are also consistent with prior studies focusing on the association between drug abuse and mental illness (Deykin *et al.* (1987); Ross *et al.* (1988)) [10]

Some studies investigating the effect of mental illness on drug abuse also report abuse of alcohol. In column (7), I report the effect of triggered depression on the consumption of alcohol. My estimates suggest that, a standard deviation increase in the depression index increases the biweekly frequency of binge drinking per day by 1.16 times. This result is consistent with the finding in Saffer and Dave (2005) which suggests having mental illness increases the likelihood of alcohol abuse by 25 %.

To ensure these estimates are not driven by and any unobserved seasonal or "September" effect, I run a placebo test where I replace the values using information from September 2008 corresponding to Wave IV interviews which were conducted between January 2008 and September 2009. To be consistent with my earlier analysis, I choose the cut-off date as September 11, 2008, and consider interviews conducted 30 days before and after this date. Appendix Table B.16 reports the results from both the first and second stages. The first-stage estimates show no significant changes in the CES-D index which is also confirmed by very small value of the partial F-statistic. [11] The second-stage estimates are approximately zero, signifying no effect of depression on marijuana or other drugs. These findings confirm our hypothesis that my estimates are not driven by any seasonal effect or any special effects from the month of September. [12]

[10] According to Regier *et al.* (1990), 29 % of all people with mental illness abuse drug or alcohol. Also, a study by Kessler *et al.* (1997) suggests individuals with mental illness are more likely to abuse drug. Saffer and Dave (2005) estimated that individuals with a history of mental illness are 69 % more likely to abuse cocaine.

[11] One should also bear in mind, that September 2008 marks the start of the global financial crisis, and any changes in depression would have been captured in the first-stage estimates.

[12] In future versions of this study, I plan to do a falsification check where the first stage esimtates remains the same, but the values of the dependent variable in the second stage would be replaced by the information from earlier Waves. This analysis would confirm whether the results are driven by unobserved differences between respondents interviewed before and after 9/11.

Given the unprecedented and unanticipated nature of the 9/11 terrorist attack, I also investigate whether my estimates are robust to considering an alternative time frame which excludes the first week after the attack. Appendix Table B.17 reproduces the estimates of the effects of depressive symptoms on illicit drug use for individuals interviewed 30 days before and after the 9/11 attack excluding the first week after the attack. While the first-stage estimates of the effect of 9/11 on depressive symptoms are approximately the same, the second stage estimates are marginally higher. The partial F-statistic for the first stage is lower compared to the baselines estimate, but still exceeds 10 signifying a strong instrument. On average, a standard deviation increase in depressive symptoms leads to more than 35 percentage points increase in illicit drug use and the qualitative conclusion for heterogeneity across the class of drugs considered remains the same. Overall, the estimates are quite robust to the consideration of an alternative time frame.

Next, I consider whether my estimates are robust to alternative measures of depression. In this respect, I followed Wang and Yang (2013) and constructed an index of depression based on a subset of questions in Table 4.1 : (1) You were bothered by things that usually don't bother you (2) You could not shake off the blues, even with help from your family and your friends (3) You had trouble keeping your mind on what you were doing (4) You were depressed (5) You were sad. Adding up scores to these individual five questions therefore produced an index with a range of 0 - 15. Table B.18 reports the 2SLS estimates using this alternative depression measure. The estimates are smaller but not statistically different from the baseline estimates. The second stage estimates suggest that, on average, a standard deviation increase in depression is associated with 27 pp higher drug consumption. The qualitative results in terms of heterogeneity across types of drugs still hold when using the alternative depression measure. As a final robustness check, I report the estimates in Table B.19

considering both the alternative depression measure and the alternative time boundary (30 days before and after the 9/11 event excluding first week). The results are approximately the same as the baseline estimates.

4.5 IV estimate of the Effects of Depression : For Whom?

The estimates in Table 4.3 could be interpreted as the average effect of a standard deviation increase in depressive symptoms for individuals whose depression was influenced by the 9/11 terrorist attack (complier group). Following Angrist and Imbens (1995) and Løken *et al.* (2012), this is defined as the average causal response (ACR). In this exercise, using estimates of the IV weights, I investigate how the composition the complier group is driving the estimates reported in Table 4.3. Under the assumption of monotonicity, using the decomposition of the linear IV estimand, for the case of the binary instrument (9/11), I am able to identify which group of individuals across the distribution of the depression scores influence the value of the average marginal effect of depression on drug consumption. The construction of the IV weights is explained below.

Let the depression levels take the finite set : depression, $Dep_i \in \{0, 1.....\bar{d}\}$, and using dummy variable $d_{di} = 1\{Dep_i \geq d_i\}$, I can write write the main estimating model as:

$$Drug_i = \alpha_0 + \sum_{d=1}^{\bar{d}} \alpha_d d_{di} + \epsilon_i \tag{4.3}$$

Under the previously described exclusion restriction and relevance assumptions, the IV estimand in equation 4.2 is $\alpha(9/11) = Cov(Drug_i, 9/11)/Cov(Dep_i, 9/11)$, where the causal estimate α is a function of my binary instrument. I can decompose

$\alpha(9/11)$ as an weighted average of the marginal effects, α_d :

$$\alpha(9/11) = \sum_{d=1}^{\bar{d}} w_d(9/11)\alpha_d, \tag{4.4}$$

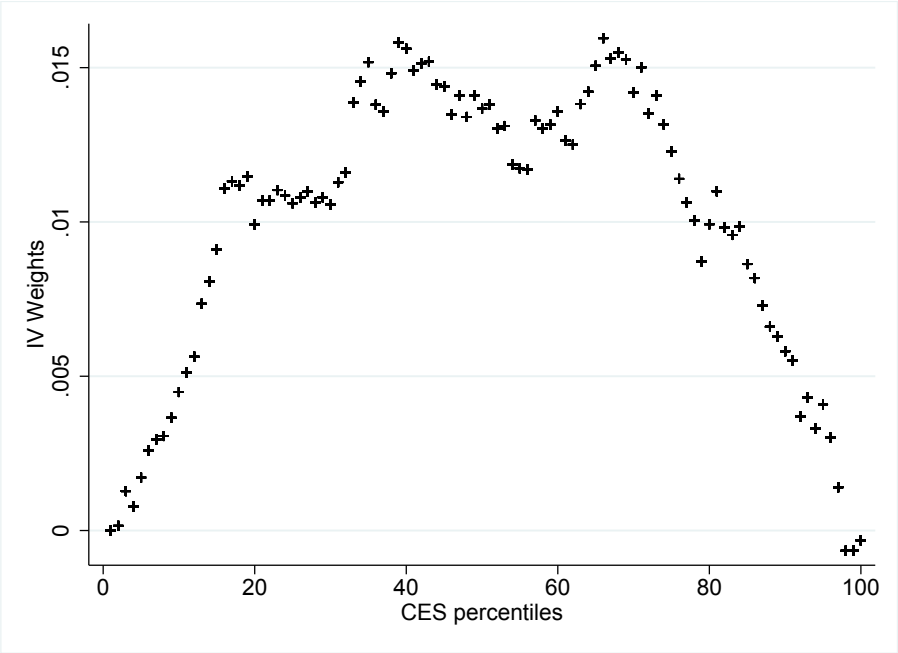
where ,

$$w_d(9/11) = \frac{Cov(d_{di}, 9/11)}{Cov(Dep_i, 9/11)} \tag{4.5}$$

The weights should sum to 1 and be positive if I assume that the event 9/11 affect everyone in one direction i.e the monotonicity assumption in Angrist and Imbens (1995). The monotonicity assumption rules out the presence of "defiers", i.e there is no one in the control group (interviewed before 9/11) who would have experienced an decreased in depressive symptoms had he been interviewed after 9/11. Given the unprecedented nature of the 9/11 terrorist attack, monotonicity is unlikely to be violated in this case. The weights, w_d here depends on the proportion of individuals, who as a result of the 9/11 shock suffered a switch in depression from less than d to at least d.

Figure 4.3 plots the distribution of weights for the IV estimates. The IV weights estimates are drawn relative to the percentile distribution of the CES-D scores. The figure reveals that the groups contributing most to the ACR based on the terrorist attack on 9/11 are those who are in the range of the 40th percentile and 70 percentile on the distribution of depression scores. There is a steep decline of the IV weighting function around the 70th percentile of depression index. Overall, most of the weights lie in the range of 20th percentile and 80th percentile. If there are non-linearities in the marginal effects ($\alpha_d \neq \alpha'_d, d \neq d'$) of depression on drug consumption, the average treatment effect will mainly be identified from the marginal effects of the individuals whose CES-D scores are around the median of the distribution.

Figure 4.3: IV Weights Relative to Percentile of CES-D Index



4.6 Heterogeneity

In this section, I investigate whether the impact of depressive symptoms disproportionately affected certain demographic groups. To do so, I repeat the estimations in Table 4.3 by gender, age, race, cognitive ability and whether the individual is a student or not.

4.6.1 Heterogeneity by Demographics

Gender

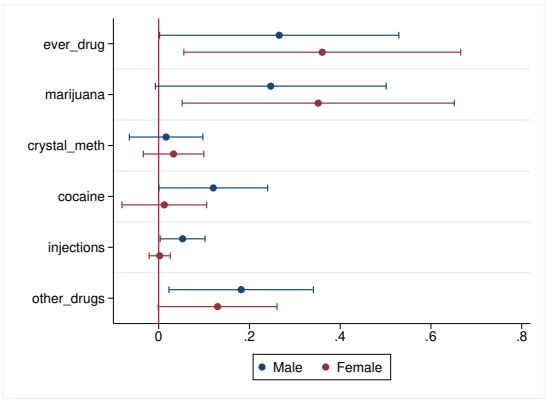
Figure 4.4 summarizes how the effects of depressive symptoms triggered by 9/11 impact drug consumption for different demographic groups, and Appendix Table B.20 reports the point estimates for the first and second-stage coefficients for each group.

The differences are not statistically different across demographic groups given the 95% confidence intervals in all panels in Figure 4.4 coincides. in Panel (a) highlights the differences by gender. The results show the 9/11 has a relatively stronger effect on depressive symptoms for females. For both females and males, those who suffer depressive symptoms are likely to use illicit drugs, however the effects are stronger for females. A standard deviation increase in CES-D index, leads to 26 percentage points increase in probability of illicit drug consumption for men which is approximately two-thirds of the effects form women. The treatment effect is mainly dominated by effects of depression on marijuana consumption for females, while males respond by increasing the frequency of binge drinking. It is well established in the literature that females are twice as likely to be depressed than males (Kessler *et al.* (1993)), but on the contrary, young adult males are likely to take drugs (Schulenberg *et al.* (2017)). My result therefore suggest, females are more likley to resort to drugs to cope with depression.

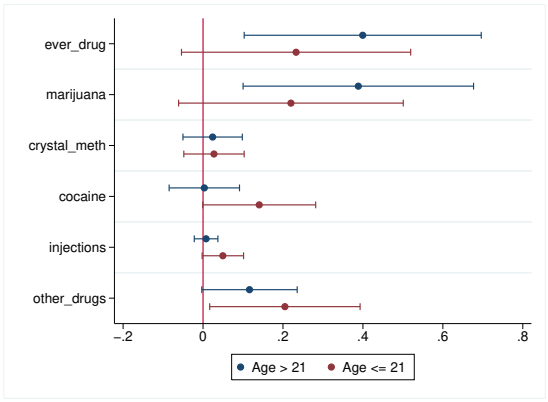
Age Group

Panel B in Figure 4.4 shows the heterogeneous effects of depressive symptoms by age-group. I split our sample into two groups: younger adults (age 21 and below) and older adults (above age 21). The minimum legal drinking age (MLDA) in US is 21 years and has been linked to positive public-health benefits after its enforcement (DeJong and Blanchette (2014)). The importance of this age in the development of substance use disorder has also been emphasized in the health literature (Sher *et al.* (2005); Chen and Jacobson (2012); Stone *et al.* (2012)). Alcohol use is also linked with drug use: some suggest it is complementary (Williams *et al.* (2004)), while other studies suggest that drug use goes down with increased alcohol use after the age of 21 (DiNardo and Lemieux (2001); Deza (2015)). My estimates suggest that, the

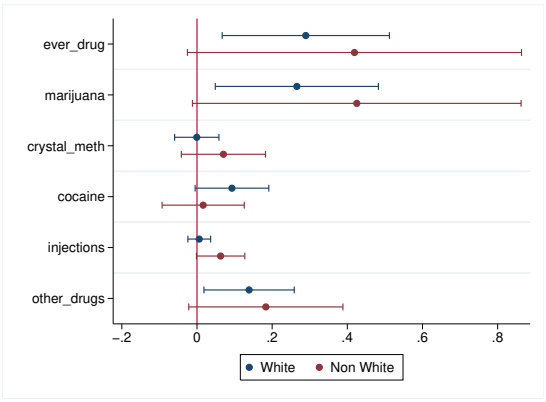
Figure 4.4: Heterogeneous Effects of Depression on Drug Consumption



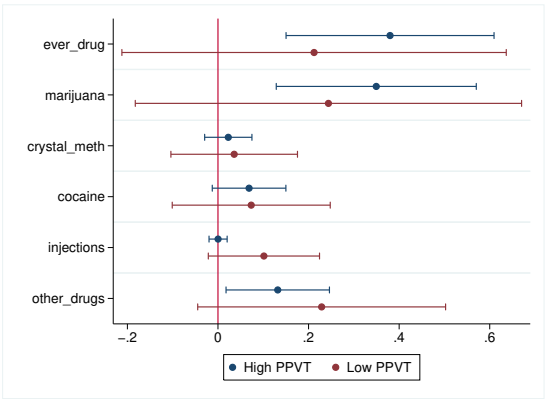
(a) Gender



(b) Age



(c) Race



(d) Cognitive Abilities

effects of depression are more concentrated in older adults. For younger adults, the treatment effects are only suggestive and statistically insignificant at any convenient levels, while for older adults the effects are even larger than the baseline average estimates in Table 4.3. Comparing across different drug types, depression mainly induces the use of marijuana among older adults while it increases the frequency of binge drinking among young adults. This is suggestive of a dynamic behavioral change, with age increasing the likelihood that individuals, as they get older substitute

alcohol with drug as a coping mechanism to counter the effects of depression.

Race

Past literature have shown that, frequency of alcohol use disorder is more common among whites, while marijuana use disorder is more common among non-whites especially African Americans (Chen and Kandel (2002); Anthony *et al.* (1994)). Panel (c) in Figure 4.4 illustrates the heterogeneous responses to depressive symptoms by racial groups. Although whites appear to be more affected by the terrorist attack on 9/11 in terms of depressive symptoms, the likelihood of illicit drug use is approximately double for non-whites for a standard deviation increase in depression. The results suggest, that whites tend to be more perturbed by traumatic events although the coping mechanism in terms of drug use may be different from nonwhites. On the contrary, whites are more likely to use cocaine compared to nonwhites, which could be explained by the fact that whites tend to have more income to purchase expensive drugs like cocaine.

4.6.2 Heterogeneity by Cognition and Academic Status

Cognitive Ability

Illicit drug use tends to impair cognitive function which negatively impacts the acquisition of new knowledge and has been linked to long-term drug use and abuse. In this section, I investigate whether individuals with greater cognitive abilities are likely to use illicit drug following the onset of depressive symptoms. Panel (d) in Figure 4.4 suggests individuals with high PPVT scores are more vulnerable to illicit drug use due to experiencing depressive symptoms. Individuals with high cognitive ability may be more likely to suffer cognitive impairment (problems with attention, memory, decision making) as a result of depression, which makes them more vul-

nerable to initiate drugs as a short-term solution to depression. Higher cognition also tends to be increase vulnerability to depression following a traumatic attack. As shown in Appendix Table B.20, individuals with higher PPVT scores are more likely to suffer depressive symptoms from the 9/11 terror attack. The most preferred form of drug initiation to combat depression appears to be marijuana, which is more affordable and more accessible for the age-group in consideration. There is only suggestive evidence that individuals with higher cognition are more likely to use other drugs. There appears to be no significant impacts of depression on people with lower cognitive abilities.

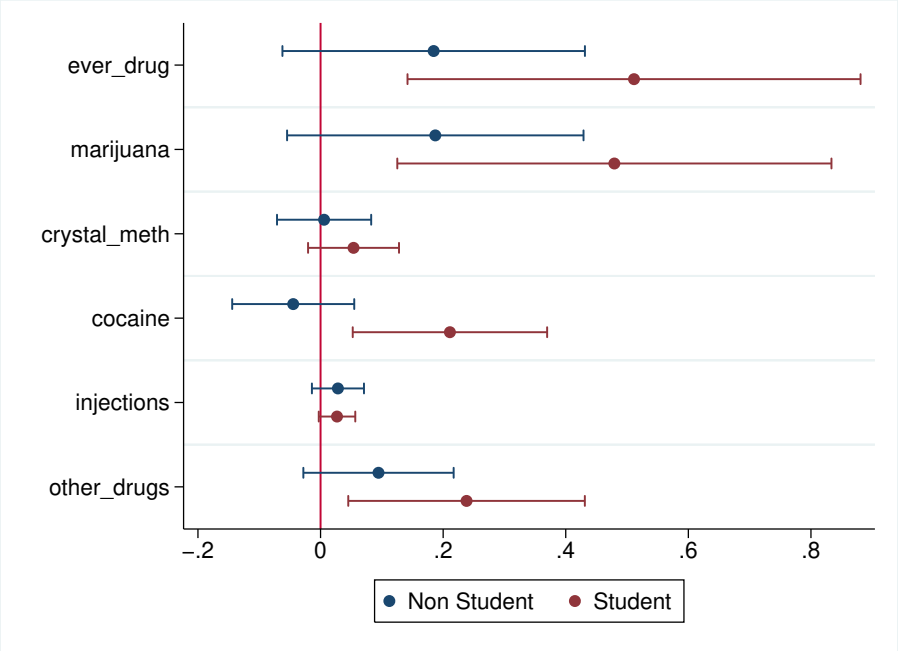
College Students VS Non-College Students

Depression is common among college students. According to the American College Health Association (2018), students cited anxiety, depression and stress as factors contributing to their poor academic performance. Around 32 % and 17 % reported of having stress and depression respectively. Also, around 17 % of these students report of using marijuana in the past month or so. Studies have reported that students with depression are more likely to smoke compared to students without (Cranford *et al.* (2009)). I focus on heterogeneity in the effects of depression between students enrolled in school/college and non-students.

Figure 4.5 illustrates the effects of depression triggered by 9/11 terrorist attack on the likelihood of using illicit drugs. My results suggest students are much more prone to drug use following depression compared to non-students. Moving to college often presents students with academic pressure which can deepen depression and heighten anxiety. Further, drugs and alcohol may be more readily available to college students which makes them more vulnerable to drugs as a tool to cope with the mental stress. This is a cause for concern as college students using alcohol or drugs to alleviate

depression are in far greater danger of developing problems relating to alcohol or drug related abuse than individuals who use drugs for recreation (Baer (2002)).

Figure 4.5: Effects of Depression on Illicit Drug Use for Students vs Non-students



On average, a standard deviation increase in depression increases drug consumption by more than 50 pp among students while there is only modest suggestive effects on non-students. First-stage estimates suggest that traumatic events like the 9/11 have much stronger effects in terms of depression on students. Consistent with the fact that, marijuana is the most commonly abused drug by college student and is more widely available, this is the most preferred option for students based on my estimates. Depression also induces an increase in the consumption of stimulants like cocaine but the effects are modest compared to marijuana. This may reflects the fact that cocaine is one of the most expensive illegal drugs in the US, and college students are often budget constrained. The results also suggest the composite category

of heroin, LSD, ecstasy and others is also popular among college students. On the other hand, young adults not attending school/college are unlikely to be induced into drugs due to depression.

4.7 Economic Consequences of Depression

In the preceding analysis, I have discussed the effects of depressive symptoms triggered by an unexpected event on illicit drug use. The heterogeneity analysis have shown young adults enrolled in schools/college and those with higher cognitive abilities, are disproportionately more affected by depression. From a policy perspective, it is also important to understand how consumption of drugs might affect labor market outcomes in terms of wages, employment and earnings. Empirical evidence estimating the relationship between wages and illicit drug use is mixed. While some studies have documented a positive association between illicit drug use and wages (Kaestner (1991); Register and Williams (1992); Buchmueller and Zuvekas (1998)), other find no such relationship (Kaestner (994a)). Evidence on drug use and employment generally points toward a negative association (Gill and Michaels (1992); French *et al.* (2001)). Use of drugs in adolescence might also have consequences in later stages. For instance, illicit drug use in early adulthood, might affect cognitive skills and impact decisions concerning school attainment and labor markets that could have negative effects on earnings. Ringel *et al.* (2006) documents negative association between late life earnings and drug use while Burgess and Propper (1998) find no such effect for soft drugs. [13]

With respect to education attainment, findings in previous literature generally point towards a negative association. Chatterji (2006) documents reductions in school

[13]According to the National Institute on Drug Abuse, soft drugs are drugs that may not lead to addiction, but users may become psychologically dependent. Examples include hallucinogens like LSD, cannabis, DMT etc.

years completed due to cocaine and marijuana use in high school. Ellickson *et al.* (1998) and Fergusson and Horwood (1997) also report negative associations. However, these studies are vulnerable to identification issues in the instruments they use to solve the endogeneity problem.McCaffrey *et al.* (2010) In this section, I discuss how 9/11 terrorist attack instrument can be used to compute bounds for the effect of depression and drug consumption on outcomes related to labor market productivity. Since, depression also impacts productivity, with one one instrument and two endogenous variables (depression and drug use) point identification is not possible. [14] In essence, I compute bounds for the impacts of depression and drug use on productivity outcomes by imposing certain restrictions.

Define y to represent the outcome of interest. I assume y is a negative measure of productivity:

$$y_i = \Gamma_0 + \Gamma_1 Dep_i + \Gamma_2 Drug_i + X_i'\Gamma_3 + \eta_i \tag{4.6}$$

$$Drug_i = \alpha_0 + \alpha_1 Dep_i + X_i'\alpha_2 + \epsilon_i \tag{4.7}$$

$$Dep_i = \gamma_0 + \gamma_1\, 9/11 + X_i'\gamma_2 + \nu_i \tag{4.8}$$

Next, through sequential substitution of equations (4.8) into (4.7) and (4.6), the reduced form equation of the model can be written as:

$$y_i = \beta_0 + \beta_1 9/11 + X_i'\beta_2 + \eta_i \tag{4.9}$$

[14] Studies like Chatterji *et al.* (2011) have also documented negative impacts of mental illness like psychotic disorder on labor force participation and employment. Breslau *et al.* (2008) also suggest mental illness to be an important factor contributing to termination of schooling which might later impact labor market outcomes.

where $\beta_1 = \gamma_1 \Gamma_1 + \gamma_1 \alpha_1 \Gamma_2$. From equation 4.9, it can be seen that there are two unknowns (Γ_1, Γ_2) with one equation to estimate. In other words, the problem is under-identified because I have only one (excluded) exogenous source of variation (9/11). Hence, I partially identify the parameters of interest, Γ_1 and Γ_2 by imposing two restrictions. First, I impose the restriction that both drug consumption and depression negatively affects productivity (in this case, positively affects our outcome variable y). Second, I assume that the OLS estimates are biased upwards due to unobserved health. Therefore, the two restrictions imply: $0 < \Gamma_1 < \Gamma_1^{OLS}$, $0 < \Gamma_2 < \Gamma_2^{OLS}$). Using these assumptions it is easy to show that:

$$\Gamma_1 \in \left[\frac{\beta_1}{\gamma_1} - \alpha_1 \Gamma_2^{OLS}, \frac{\beta_1}{\gamma_1}\right] \tag{4.10}$$

$$\Gamma_2 \in \left[\frac{\beta_1 - \gamma_1 \Gamma_1^{OLS}}{\gamma_1 \alpha_1}, \frac{\beta_1}{\gamma_1 \alpha_1}\right] \tag{4.11}$$

Using the restrictions therefore the parameters of interest Γ_1 and Γ_2 are bounded. I focus on two productivity outcomes for this analysis: ii) frequency of falling asleep in the past week at school/work i) frequency of skipping school/work in the past month due to health problems. While very few studies exclusively focus on the effect of drugs on productivity, there is evidence that initiation of drugs might be associated with dropping out of school (Bray *et al.* (2000)) and truancy (Roebuck *et al.* (2004)). The National Drug Intelligence Centre (2011) estimates the total annual loss in market productivity due to illicit drugs to be $ 42 billion (2015 US$). I additionally, provide estimates of the effect of depression on these productivity outcomes.

Table 4.4 shows the main results of this analysis. Panel B presents the estimates of the direct effect of the 9/11 shock. While the estimates suggest, that the traumatic experiences of the 9/11 events had no significant impact on school or work related

absenteeism, they however did affect the number of productive hours because of sleep. The 9/11 events increased the frequency of sleeping during work/study hours by about 2.4 percentage points. As outlined earlier, from this, I estimate the bounds of the parameters determining the impact of depression and drug consumption on the outcomes of interest. Drug use can make it difficult to sleep because of the influence of extreme stimulants like cocaine or methamphetamine, and therefore individuals taking these drug may have trouble staying up during school/work. Also, illicit drug use can hamper the motivation and cognitive ability to use school or work hours productively.

My main estimates are presented in Panel A. For the outcome variable, sleep, the coefficient on depression is in the range [0.21,0.22], and for drug use it is in the range [0.458,0.672]. These estimates suggest that, drug use has a larger effect on the likelihood of falling asleep during school/work compared to depression. On the other hand, the estimates in Table 4.4 only provides suggestive evidence of the effects of drug use on school/ work absence, Overall, these results suggest that, both depression and drug use may lead to sleeping disorders and disturbances and therefore affects productivity at school or students and at workplaces for workers.

I also investigate whether these effects are strongly concentrated in certain groups. Particularly, I focus on college going students and working young adults. My estimates show that the 9/11 shock has a much bigger impact on college students' productivity compared to the average (around 4 times). As discussed above they are also more likely to consume drugs than individuals not enrolled in college. On average one standard deviation increase in depression can increase the frequency of sleep during work/school hours by 0.70 times. Drug use on the other hand has effects that are twice as large as the average estimates. Similar results are observed for absenteeism from school. On the other hand, I do not observe any statistically significant impacts

Table 4.4: Partially Identified Estimates of the Effect of Depression and Drug Use on Sleeping or Absenteeism at School/Work

	Outcomes: Productivity at School or Work					
	Sleeping	Skipping	Sleeping	Skipping	Sleeping	Skipping
			If enrolled in School/College		If working	
	Panel A: Partially Identified Bounds					
CES-D	[0.209, 0.217]	[0.044, 0.050]	[0.702,0.716]	[0.404,0.435]	[-0.148,-0.144]	[-0.291,-0.287]
95 % CI	(0.039,0.432)	(-0.146, 0.212)	(0.291,1.317)	(0.149, 0.932)	(-0.584,0.133)	(-0.873,-0.002)
Drug	[0.458, 0.672]	[-0.011, 0.159]	[1.099,1.265]	[0.703,0.812]	[-0.551,0.264]	[-0.561,-0.436]
95 % CI	(-0.064, 1.271)	(-0.584, 0.695)	(0.503,2.347)	(0.232,1.684)	(-3.461,3.13)	(-4.524,4.294)
	Panel C: 9/11 on productivity outcomes					
9/11	0.024***	0.006	0.077***	0.049***	-0.011	-0.025
	(0.011)	(0.011)	(0.019)	(0.017)	(0.014)	(0.015)
N Obs	4,650	4,593	1,868	1,864	2,777	2,724
	Panel D: First-Stage Effects on Depression					
9/11	0.118***	0.120***	0.128***	0.132***	0.108***	0.108***
	(0.031)	(0.031)	(0.047)	(0.047)	(0.042)	(0.042)
N Obs	4,629	4,573	1863	1859	2762	710
	Panel E: Second-Stage Effects on Drug Use					
CES-D	0.370***	0.353***	0.597***	0.561***	0.267	0.242
	(0.154)	(0.149)	(0.293)	(0.250)	(0.226)	(0.232)
N Obs	4,615	4,559	1,858	1,854	2,753	2,701

Notes The productivity outcomes variables include frequency of falling asleep at school/work in the past week and skipping school/work in the past month. Panel A reports the partially identified effects of both depressive symptoms and drug use on the productivity outcomes. 95% confidence intervals are estimated following Imbens and Manski (2004). Panel B reports the reduced form-estimates of 9/11 on these outcomes, while Panel C and D reports the first and second stage estimates as in Table 4.3. Robust standard errors are reported in parenthesis in Panel B - D.

of depression and drug use on sleeping during work or absenteeism from work. From a policy perspective, it is important to target policies that reduce depression and drug use because this can facilitate improvement of educational outcome for young adults.

4.8 Conclusion

This study provides empirical evidence of the short-term effects of depression on drug consumption. The identification strategy uses the exogenous variation of an unexpected event– the effects of 9/11 attack on individual's depression as measured by

the CES-D Scales. Comparing the individuals interviewed before and after 9/11, I find a significant jump in the measured depression levels. With no significant observable differences between the treatment and control groups, I am able to use the occurrence of 9/11 as an instrument for depressive symptoms to identify the causal effect on drug consumption.

I focused my analysis on young adults, aged 18-28 years. This is important because observed statistics suggest that illicit drug use is the largest for these population. My results show that depressive symptoms cause individuals to use drugs. The effects are heterogeneous with respect to the type of drug with the largest and most persistent effects observed for marijuana. Heterogeneity analysis provided suggestive evidence that certain sub-groups are more affected than others. In particular, the effects are higher for whites, females, adults above the age of 21 and college going students with higher cognitive abilities.

I supplement my main analysis with an attempt to identify the impact of drug use on productivity in the school/workplace. My identification strategy makes use of the 9/11 event to partially identify the impact of drug consumption and also depression on sleep during work/school hours and truancy. The results suggest that, drug consumption can significantly reduce productivity by reducing sleep hours and worsening health condition of the affected individuals. Finally the results, provides added justification for subsidizing medical treatment for depression in a effort to reduce the cost of curbing drug consumption.

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APPENDIX A

SUPPLEMENTARY INFORMATION ON DATA AND METHODS

A.1 Hierarchical condition categories risk adjustment scores

CMS uses the Hierarchical Condition Categories (HCC) risk adjustment score to adjust capitation payments to Medicare Advantage plans based on their enrollees' health expenditure risk. The HCC score is designed to synthesize information about individuals' chronic illnesses and demographics from CMS administrative records [1] The index is a function of age, gender, indicators for numerous chronic illnesses and the initial reason for Medicare eligibility.

Raw HCC scores may embed some measurement error. In particular, there is evidence that some of the spatial and temporal variation in diagnosis rates for the chronic illnesses used to compute HCC scores actually reflects differences in medical care providers' diagnostic and treatment decisions rather than differences in patients' health (Song *et al.* (2010); Welch *et al.* (2011)). [2] I reduce the scope for such errors by adjusting HCC scores using the procedure from Finkelstein *et al.* (2016). This involves regressing HCC score on dummies for year and geographic area, individual fixed effects, and a vector of covariates used to proxy for latent health.

I follow Finkelstein *et al.* (2016) in defining geographic areas as $j = 1, ..., 306$ Dartmouth Atlas of medical care hospital referral regions (HRR) and in defining the vector of covariates, x_{it}, to include dummies for five-year age bins and relative-year fixed effects, ρ_{it}, for people who change their residential location, where $\rho_{it} = t - t^*$, t^* denotes the year of move, and $\rho_{it} = 0$ for people who do not move at any point during the study period. [3] Including these relative–year migration dummies in

[1] Additional background information on the risk adjustment model can be found at `http://www.nber.org/data/cms-risk-adjustment.html`.

[2] For example, Song *et al.* (2010) uses movers to examine how diagnosis rates change as people move across quintiles of the distribution of spending. Results showed a significantly larger increase in diagnosis rates for those who moved to higher intensity regions compared to those who moved to lower intensity regions and those who did not move at all.

[3] "Hospital Referral Regions" (HRRs) represent regional medical care markets for tertiary medical care as determined by the Dartmouth Atlas. Each HRR contains at least one hospital that performs

the covariate vector recognizes that migration late in life may coincide with negative health shocks that induce people to move closer to caregivers. I use the resulting predicted health index as a measure of objective health in the survival function.

A.2 Additional tables and figures

Figure C.1 provides a graphical representation of our first stage results. The histogram shows the density of the instrument constructed from the HRR fixed effects in (2.12). The solid line shows the conditional variation in medical expenditures predicted by the HRR–based instrument. Specifically, it shows the expenditure levels predicted by regressing residual medical spending on residual variation in the instrument, after controlling for the HCC index, presence of ADL restrictions, presence of IADL restrictions, self-reported health categories, smoking history, sex, an age spline that allows the marginal effect of age to vary by sex and by whether people are under or over 90, marital status, living children, underweight BMI, indicators for race, and indicators for educational attainment. The dashed lines represent a 95% confidence interval on the prediction. Intuitively, the slope suggests that a one dollar increase in the instrument is associated with approximately a one dollar increase in medical expenditure.

A.3 Solution Method for the Dynamic Optimization Problem

I solve the optimization problem by backward induction. At the terminal age, $T = 95$, death is certain in the following period. All remaining resources are assumed to be consumed by the individual. Given this and $a_{i,T+1} = 0$, utility in the final period

major cardiovascular procedures and neurosurgery. HRRs were defined by assigning Hospital Service Areas to the region where the greatest proportion of major cardiovascular procedures were performed, with minor modifications to achieve geographic contiguity, a minimum population size of 120,000, and a high localization index. The Dartmouth Atlas defines a Hospital Service Area as a collection of ZIP Codes whose residents receive most of their hospitalizations from hospitals in the area. For further details see: `http://www.dartmouthatlas.org/downloads/methods/geogappdx.pdf`.

can be expressed as, $V_{i,T}(a_{i,T}, h_{i,T}, I_i)$. Specifically, I use a polynomial grid, which is preferred to a grid of equispaced points because the observed distribution of assets in data is skewed to the right. Moving backward, I discretize the state space with disproportionately more points in the lower end of the asset domain. I allocate 40 points for assets and 10 points for health and permanent income. Using grid search, I determine the optimal level of consumption and medical expenditures. Conditioning first on the choice of consumption c, the optimal medical expenditures, $m^*(c)$ is determined. Then, I determine optimal consumption using $m^*(c)$. To calculate the values between grid points for the next period's value function, I used bi-linear interpolation. I use a simulation-based approach to calculate transition probabilities between health states using reduced-form estimates from the health production function. Specifically, for each level of medical expenditure, I calculate non-parametric transition probabilities of moving between each pair of health states. This solution method yields reasonable decision rules. After solving for optimal decision rules, I simulate the life paths of 1000 individuals using initial conditions drawn from the data.

A.4 Measurement System & Estimation Algorithm for the Health Technology

$$Z_{i,t,k} = \mu_{t,k} + \lambda_{t,k} \ln h_{it} + \epsilon_{i,t,k} \qquad \forall k = 1, ..., K$$

$Z_{i,t,k}$ are the observed measures of health, $\mu_{t,k}$ is the location and $\lambda_{t,k}$ is the factor loading.$E(\epsilon_{t,k}) = 0 \; \forall \; t \; \epsilon \; \{1, ..., T\}$

A.4.1 Normalization

1. $E(\ln h_{66}) = c, \quad c > 0$
2. $\lambda_{t,1} = 1 \forall t$

That is, the mean of the latent factor at age 66 is assumed to be a constant, c which I set to 1. The second normalization relates to setting the scale for one of the measurements.

I make the following standard assumption relating to the measurement errors:

$(i) \; \epsilon_{t,k} \perp \epsilon_{t,k'} \;\; \forall \; t \; \& \; k \neq k'$

$(ii) \; \epsilon_{t,k} \perp \epsilon_{t',k'} \;\; \forall \; t \neq t' \; \& \; k \; \& \; k'$

$(iii) \; \epsilon_{t,k} \perp h_{t'} \;\; \forall \; t \; \& \; t' \; \& \; k$

Assumption (i) and (ii) states that measurement errors are independent across measures and across age. Assumption (iii) states that the measurement errors for any period and measures are independent of the true latent health stock.

A.4.2 Estimating Initial Conditions and Measurement Parameters

My approach to estimating initial conditions and parameters of the measurement equations follows Cunha and Heckman (2008) Cunha *et al.* (2010). First, factor loadings, $\lambda_{66,k} \forall k \neq 1$ are computed based on assumptions (i)-(iv) [4] using:

$$\lambda_{66,k} = \frac{Cov(Z_{66,k}, Z_{66,k'})}{Cov(Z_{66,1}, Z_{66,k'})}, \text{ where } k \neq k'$$

Now, given the normalization in age 66 for mean health the intercepts of the measurement equations can be easily estimated by :

$$\mu_{66,k} = E(Z_{66,k}) - c \; \forall k \qquad [E(\ln h_{66}) = c \;\; \& \;\; E(\epsilon_{66,k}) = 0]$$

The initial health distribution is computed from the covariance in measures of health in the following way:

$$\begin{aligned} \text{Cov}(Z_{66,k}, Z_{66,k'}) &= \text{Var}(\ln h_{66}) \lambda_{66,k} \lambda_{66,k'} \\ \sigma_h^2 = Var(\ln h_{66}) &= \frac{\text{Cov}(Z_{66,k}, Z_{66,k'})}{\lambda_{66,k} \lambda_{66,k'}} \end{aligned}$$

A.4.3 Steps to Estimate Health Technology

1. **Obtain the residual measure of health**

   I use the estimates for the measurement parameters to calculate the residual measures of health is computed as:

$$\tilde{Z}_{66,k} = \frac{Z_{66,k} - \mu_{66,k}}{\lambda_{66,k}} = \ln h_{66} + \tilde{\epsilon}_{66,k} \;\; \forall k$$

$$\tilde{\epsilon}_{66,k} = \frac{\epsilon_{66,k}}{\lambda_{66,k}}$$

2. **Estimating the health production function**

   I estimate the health production function without imposing any restrictions on

[4] Assumption (i) is redundant when more than 2 measures are available

the parameters of the health production function. Next rewriting the health production function 3.1:

$$\frac{Z_{67,k} - \mu_{67,k}}{\lambda_{67,k}} - \frac{\epsilon_{67,k}}{\lambda_{67,k}} = \alpha_{0,66} + \alpha_{1,66}(\tilde{Z^h}_{66,k} - \tilde{\epsilon}^h_{66,k}) + \alpha_{2,66} m_{66} + \nu_{66}$$

Again, rearranging:

$$\tilde{Z}_{67,k} = \alpha_{0,66} + \alpha_{1,66}\tilde{Z}_{66,k} + \alpha_{2,66} m_{66} + \pi_{66,k} \tag{A.1}$$

where,

$$\pi_{66,k} = \tilde{\epsilon}_{67,k} + \alpha_{1,66}\tilde{\epsilon}_{66,k} + \nu_{66}$$

Identification of the parameters α's requires the value of the residual measure $\tilde{Z}_{67,k}$, which inturn requires the identification of $\mu_{67,k}$, $\lambda_{67,k}$. Under the normalization and assumptions in A.4.1, $\lambda_{67,k}$ is normalized to 1 for $k = 1$. Identification requires setting the mean of the latent health factor to c for all ages. The scale and the factor loading for all measures can be identified using the same methodology in A.4.

$\tilde{Z}_{66,k}$ is endogenous because it correlates with $\pi_{66,k}$ through the $\tilde{\epsilon}_{66,k}$ term (measurement equation). To account for endogeneity of $\tilde{Z}_{66,k}$, I use $Z_{66,k'}$, $k \neq k'$ as IVs to consistently estimate the α's. The validity of the IVs comes from assumptions (i)-(iii) i.e the measurement errors for any measure is independent across measures and age. Therefore, $[Corr(Z_{66,k'}, \tilde{\epsilon}_{67,k}), Corr(Z_{66,k'}, \tilde{\epsilon}_{66,k}) = 0]$. To identify the coefficient on m_{66}, I use the supply-side variation in medical expenditures across geographic regions.

3. **Estimate the variance of the health shock**

To estimate the variance of the health shock, $\sigma^2_{\nu,66}$, I use the covariance between

the residual in A.1 and an alternative residual measure of health in period, $t = 67$, $\tilde{Z}_{67,k'}$:

$$\mathrm{Cov}\big(\pi_{66,k}, \tilde{Z}_{67,k'}\big) = \mathrm{Var}(\nu_{66}) = \sigma^2_{\nu,66}$$

4. **Estimate the parameters for the remaining periods**

   Steps (2) - (4) is then repeated to estimate the parameters of the health evolution for the remaining periods.

APPROVAL:CONTINUATION

Jonathan Ketcham
WPC: Economics
480/965-5507
ketcham@asu.edu

Dear Jonathan Ketcham:

On 10/23/2020 the ASU IRB reviewed the following protocol:

Type of Review:	Continuing Review
Title:	The Effects of Pollution on Health, Cognitive Function and Financial Decisions of Older Adults
Investigator:	Jonathan Ketcham
IRB ID:	STUDY00001990
Category of review:	
Funding:	None
Grant Title:	None
Grant ID:	None
Documents Reviewed:	None

The IRB approved the protocol from 10/23/2020 to 10/22/2021 inclusive. Three weeks before 10/22/2021 you are to submit a completed Continuing Review application and required attachments to request continuing approval or closure.

If continuing review approval is not granted before the expiration date of 10/22/2021 approval of this protocol expires on that date. When consent is appropriate, you must use final, watermarked versions available under the "Documents" tab in ERA-IRB.

In conducting this protocol you are required to follow the requirements listed in the INVESTIGATOR MANUAL (HRP-103).

Sincerely,

IRB Administrator

cc:

Nirman Saha
Kelly Bishop
Sophie-Marie Mathes
Nicolai Kuminoff

APPENDIX B

SUPPLEMENTARY TABLES

Table B.1: First Stage Results from Survival Function Estimation

	(2)	(3)	(4)	(5)	(6)
instrument	0.868	0.961	0.796	0.646	0.670
	(0.166)	(0.171)	(0.160)	(0.195)	(0.293)
HCC index	14.926	14.849	14.857	15.098	15.105
	(0.414)	(0.396)	(0.397)	(0.398)	(0.400)
one or more ADL restrictions	2.556	2.644	2.613	2.552	2.562
	(0.195)	(0.199)	(0.196)	(0.196)	(0.196)
one or more IADL restrictions	1.493	1.449	1.482	1.490	1.493
	(0.214)	(0.211)	(0.212)	(0.209)	(0.207)
health = poor	7.633	7.812	7.825	7.868	7.850
	(0.461)	(0.466)	(0.469)	(0.467)	(0.469)
health = fair	2.353	2.470	2.475	2.486	2.487
	(0.241)	(0.242)	(0.241)	(0.240)	(0.240)
health = very good	-1.599	-1.635	-1.643	-1.625	-1.631
	(0.149)	(0.143)	(0.143)	(0.146)	(0.147)
health = excellent	-2.641	-2.657	-2.670	-2.656	-2.670
	(0.186)	(0.185)	(0.183)	(0.184)	(0.184)
ever smoked	0.195	0.220	0.193	0.145	0.139
	(0.147)	(0.143)	(0.143)	(0.143)	(0.141)
male	-0.553	-1.128	-1.030	-0.812	-0.855
	(1.934)	(1.869)	(1.850)	(1.840)	(1.823)
age x male x under 90	-0.369	-0.378	-0.382	-0.394	-0.393
	(0.024)	(0.022)	(0.022)	(0.022)	(0.022)
age x female x under 90	-0.353	-0.369	-0.372	-0.381	-0.381
	(0.019)	(0.018)	(0.018)	(0.019)	(0.018)
age x male x over 90	-0.361	-0.372	-0.376	-0.387	-0.387
	(0.022)	(0.021)	(0.021)	(0.021)	(0.021)
age x female x over 90	-0.337	-0.353	-0.356	-0.364	-0.364
	(0.018)	(0.017)	(0.017)	(0.018)	(0.018)
insurance type covariates		x	x	x	x
health care quality covariates			x	x	x
environmental covariates				x	x
state dummies					x
number of person-years	44,697	44,697	44,697	44,697	44,697
number of people	22,206	22,206	22,206	22,206	22,206

Notes: The table reports coefficients and bootstrapped standard errors clustered by hospital referral region from the first stage regressions corresponding to Table 2.2 columns (2) through (6). The dependent variable is annual gross medical expenditures, measured in $1,000. Continued on the next page.

Table B.1: (continued) First Stage Results from Survival Function Estimation

	(2)	(3)	(4)	(5)	(6)
married	0.412	0.114	0.127	0.135	0.121
	(0.153)	(0.150)	(0.150)	(0.151)	(0.151)
has living children	0.634	0.617	0.638	0.597	0.572
	(0.264)	(0.255)	(0.255)	(0.254)	(0.254)
underweight BMI	-0.741	-0.778	-0.754	-0.692	-0.682
	(0.361)	(0.356)	(0.355)	(0.356)	(0.356)
African-American	-2.136	-1.150	-1.102	-1.039	-1.079
	(0.332)	(0.307)	(0.303)	(0.309)	(0.310)
Hispanic	-1.579	0.010	-0.031	-0.026	-0.067
	(0.649)	(0.679)	(0.704)	(0.679)	(0.664)
race = other	-2.647	-1.602	-1.719	-1.548	-1.494
	(0.527)	(0.509)	(0.492)	(0.496)	(0.501)
eduction = less than high school	-1.493	-1.057	-1.031	-0.933	-0.924
	(0.205)	(0.204)	(0.202)	(0.203)	(0.205)
education = some college	0.631	0.576	0.553	0.575	0.557
	(0.193)	(0.191)	(0.192)	(0.193)	(0.194)
education = college	1.810	1.485	1.402	1.396	1.377
	(0.215)	(0.204)	(0.203)	(0.202)	(0.204)
Medicare advantage coverage		-3.234	-3.271	-3.326	-3.284
		(0.252)	(0.246)	(0.230)	(0.234)
Medigap coverage		1.519	1.525	1.544	1.571
		(0.184)	(0.183)	(0.184)	(0.187)
Medicaid coverage		-1.622	-1.564	-1.628	-1.652
		(0.261)	(0.257)	(0.261)	(0.261)
insurance type covariates		x	x	x	x
health care quality covariates			x	x	x
environmental covariates				x	x
state dummies					x
number of person-years	44,697	44,697	44,697	44,697	44,697
number of people	22,206	22,206	22,206	22,206	22,206

Notes: The table reports coefficients and bootstrapped standard errors clustered by hospital referral region from the first stage regressions corresponding to Table 2.2 columns (2) through (6). The dependent variable is annual gross medical expenditures, measured in $1,000. Continued on the next page.

Table B.1: (continued) First Stage Results from Survival Function Estimation

	(2)	(3)	(4)	(5)	(6)
hospital compare index			-0.249	-6.643	-0.544
			(2.523)	(3.177)	(5.175)
hospital beds / capita			-0.906	-0.528	-0.769
			(0.274)	(0.372)	(0.512)
primary care physicians / capita			-0.006	-0.022	-0.027
			(0.012)	(0.017)	(0.025)
medical care specialists / capita			0.017	0.007	0.014
			(0.006)	(0.009)	(0.013)
ambulatory discharges / capita			0.023	0.026	0.032
			(0.010)	(0.011)	(0.018)
automobile mortality				-0.180	-0.187
				(0.027)	(0.032)
homicide mortality				-0.038	-0.078
				(0.036)	(0.049)
fine particulate matter				-0.173	-0.142
				(0.050)	(0.054)
mean summer high temperature				0.030	0.036
				(0.020)	(0.023)
mean winter low temperature				-0.017	-0.013
				(0.021)	(0.032)
share urban				0.093	-1.489
				(1.230)	(1.791)
median household income				0.000	0.000
				(0.000)	(0.000)
high school graduation rate				6.045	4.026
				(3.766)	(5.351)
insurance type covariates		x	x	x	x
health care quality covariates			x	x	x
environmental covariates				x	x
state dummies					x
number of person-years	44,697	44,697	44,697	44,697	44,697
number of people	22,206	22,206	22,206	22,206	22,206

Notes: The table reports coefficients and bootstrapped standard errors clustered by hospital referral region from the first stage regressions corresponding to Table 2.2 columns (2) through (6). The dependent variable is annual gross medical expenditures, measured in $1,000.

Table B.2: IV Survival Functions, Full Results

	(2)	(3)	(4)	(5)	(6)
$1,000 in medical spending	-0.471	-0.424	-0.638	-0.884	-0.815
	(0.201)	(0.178)	(0.263)	(0.628)	(5.804)
1st stage residual morbidity	0.537	0.493	0.707	0.954	0.885
	(0.203)	(0.180)	(0.264)	(0.629)	(5.804)
HCC index	11.114	10.718	13.910	17.771	16.726
	(3.014)	(2.673)	(3.864)	(9.418)	(86.501)
one or more ADL restrictions	3.049	2.958	3.514	4.128	4.021
	(0.558)	(0.518)	(0.698)	(1.572)	(14.305)
one or more IADL restrictions	1.281	1.216	1.544	1.919	1.860
	(0.396)	(0.367)	(0.487)	(0.993)	(8.739)
health = poor	7.190	6.854	8.518	10.462	9.845
	(1.670)	(1.516)	(2.231)	(5.020)	(46.173)
health = fair	2.756	2.666	3.187	3.810	3.613
	(0.544)	(0.520)	(0.733)	(1.613)	(14.962)
health = very good	-2.390	-2.343	-2.694	-3.080	-2.953
	(0.453)	(0.426)	(0.542)	(1.043)	(9.442)
health = excellent	-3.810	-3.699	-4.269	-4.910	-4.696
	(0.673)	(0.618)	(0.786)	(1.643)	(14.832)
ever smoked	1.302	1.271	1.307	1.337	1.327
	(0.250)	(0.244)	(0.257)	(0.299)	(1.322)
male	6.761	6.646	6.436	6.567	6.454
	(3.366)	(3.322)	(3.558)	(4.279)	(16.835)
age x male x under 90	0.019	0.029	-0.053	-0.159	-0.130
	(0.077)	(0.069)	(0.099)	(0.239)	(2.197)
age x female x under 90	0.090	0.098	0.018	-0.080	-0.054
	(0.072)	(0.066)	(0.102)	(0.247)	(2.204)
age x male x over 90	0.044	0.053	-0.028	-0.132	-0.103
	(0.074)	(0.068)	(0.098)	(0.236)	(2.158)
insurance type covariates		x	x	x	x
health care quality covariates			x	x	x
environmental covariates				x	x
state dummies					x
number of person-years	44,697	44,697	44,697	44,697	44,697
number of people	22,206	22,206	22,206	22,206	22,206

Notes: The table reports coefficients and bootstrapped standard errors clustered by hospital referral region from the survival functions in Table 2.2 columns (2) through (6). The table reports average marginal effects expressed as percentage point changes in the one-year probability of death. Continued on the next page.

Table B.2: (continued) IV Survival Functions, Full Results

	(2)	(3)	(4)	(5)	(6)
age x female x over 90	0.112	0.119	0.042	-0.052	-0.027
	(0.068)	(0.063)	(0.098)	(0.235)	(2.122)
married	-0.394	-0.520	-0.491	-0.430	-0.441
	(0.232)	(0.212)	(0.230)	(0.293)	(0.520)
has living children	0.594	0.554	0.701	0.837	0.759
	(0.411)	(0.406)	(0.456)	(0.591)	(3.851)
underweight BMI	2.258	2.307	2.155	1.994	2.006
	(0.425)	(0.417)	(0.487)	(0.709)	(3.743)
African-American	-0.799	-0.427	-0.661	-0.817	-0.798
	(0.530)	(0.438)	(0.528)	(0.740)	(7.508)
Hispanic	-1.966	-1.362	-1.334	-1.436	-1.294
	(0.668)	(0.601)	(0.736)	(0.972)	(5.946)
race = other	-2.873	-2.372	-2.716	-2.958	-2.957
	(1.178)	(1.007)	(1.111)	(1.447)	(7.807)
eduction = less than high school	-0.752	-0.508	-0.740	-0.902	-0.835
	(0.432)	(0.349)	(0.408)	(0.663)	(4.487)
education = some college	0.238	0.175	0.293	0.455	0.409
	(0.277)	(0.267)	(0.313)	(0.434)	(3.498)
education = college	-0.123	-0.280	-0.004	0.351	0.248
	(0.473)	(0.399)	(0.478)	(0.910)	(8.898)
Medicare advantage coverage		-0.878	-1.564	-2.430	-2.190
		(0.578)	(0.818)	(1.970)	(18.238)
Medigap coverage		-0.227	0.118	0.542	0.442
		(0.432)	(0.559)	(1.112)	(9.634)
Medicaid coverage		-1.501	-1.811	-2.208	-2.128
		(0.479)	(0.564)	(1.105)	(8.932)
insurance type covariates		x	x	x	x
health care quality covariates			x	x	x
environmental covariates				x	x
number of person-years	44,697	44,697	44,697	44,697	44,697
number of people	22,206	22,206	22,206	22,206	22,206

Notes: The table reports coefficients and bootstrapped standard errors clustered by hospital referral region from the survival functions in Table 2.2 columns (2) through (6). The table reports average marginal effects expressed as percentage point changes in the one-year probability of death. Continued on the next page.

Table B.2: (continued) IV Survival Functions, Full Results

	(2)	(3)	(4)	(5)	(6)
hospital compare index			-1.888	-5.581	-2.347
			(3.224)	(7.205)	(20.157)
hospital beds / capita			-0.694	-0.255	-0.385
			(0.497)	(0.693)	(3.274)
primary care physicians / capita			-0.017	-0.020	-0.011
			(0.017)	(0.031)	(0.126)
medical care specialists / capita			0.019	0.006	0.008
			(0.012)	(0.017)	(0.127)
ambulatory discharges / capita			0.026	0.037	0.026
			(0.016)	(0.025)	(0.187)
automobile mortality				-0.101	-0.080
				(0.115)	(0.960)
homicide mortality				-0.108	-0.117
				(0.061)	(0.557)
fine particulate matter				-0.109	-0.107
				(0.150)	(0.910)
mean summer high temperature				0.033	0.042
				(0.042)	(0.221)
mean winter low temperature				-0.040	-0.037
				(0.030)	(0.288)
share urban				3.527	2.495
				(1.822)	(10.999)
median household income				0.000	0.000
				(0.000)	(0.000)
high school graduation rate				6.359	7.006
				(6.425)	(58.410)
insurance type covariates		x	x	x	x
health care quality covariates			x	x	x
environmental covariates				x	x
state dummies					x
number of person-years	44,697	44,697	44,697	44,697	44,697
number of people	22,206	22,206	22,206	22,206	22,206

Notes: The table reports coefficients and bootstrapped standard errors clustered by hospital referral region from the survival functions in Table 2.2 columns (2) through (6). The table reports average marginal effects expressed as percentage point changes in the one-year probability of death.

Table B.3: Internal Meta-Analysis of Sensitivity to Analytic Decisions

	ln(mean VSL) (all ages)	ln(mean VSL) (age 67)	ln(mean VSL) (age 77)	ln(mean VSL) (age 87)
Gompertz specification	-0.088***	-0.134***	-0.055**	0.011
	(0.026)	(0.026)	(0.026)	(0.026)
claims-based spending measure	0.261***	0.226***	0.299***	0.382***
	(0.026)	(0.026)	(0.026)	(0.026)
include workers in estimation sample	0.214***	0.243***	0.124***	0.084***
	(0.026)	(0.026)	(0.026)	(0.026)
covariates: Table 2, Column 2	-0.119***	-0.129***	-0.110***	-0.101**
	(0.042)	(0.042)	(0.042)	(0.042)
covariates: Table 2, Column 4	-0.324***	-0.322***	-0.327***	-0.324***
	(0.042)	(0.042)	(0.042)	(0.042)
covariates: Table 2, Column 5	-0.427***	-0.420***	-0.429***	-0.426***
	(0.042)	(0.042)	(0.042)	(0.042)
covariates: Table 2, Column 6	-0.552***	-0.542***	-0.554***	-0.553***
	(0.042)	(0.042)	(0.042)	(0.042)
instrument: FGW + integer age	-0.002	-0.002	-0.002	-0.002
	(0.042)	(0.042)	(0.042)	(0.042)
instrument: FGW + integer age x gender	-0.003	-0.003	-0.003	-0.003
	(0.042)	(0.042)	(0.042)	(0.042)
instrument: FGW, including never-movers	-0.129***	-0.130***	-0.129***	-0.128***
	(0.042)	(0.042)	(0.042)	(0.042)
instrument: end of life spending	0.328***	0.327***	0.327***	0.328***
	(0.042)	(0.042)	(0.042)	(0.042)
R-squared	0.74	0.74	0.74	0.76
number of models	200	200	200	200

Note: The table reports coefficients and standard errors from a regression of VSL estimates on indicators for features of 200 alternative specifications of the survival function. In column (1) the dependent variable is the log of mean VSL. In columns (2), (3) and (4) the dependent variables are the logs of mean VSL for the subsets of people aged 67, 77, and 87. The excluded indicators define the reference model as the one summarized in Table 2 column (3). It uses a Gompit specification for mortality with MCBS spending data, workers excluded, and the instrument based on Finkelstein, Gentzkow and Williams (2016). Coefficients define the conditional effects of deviating from those analytic decisions as explained in the main text. Because all covariates are binary the Halvorsen-Palmquist formula can be used to convert any coefficient shown in the table, β, into its percentage point effect on the VSL: $100 * (e^{\beta} - 1)$. Asterisks indicate the coefficients are statistically distinguishable from zero at the 10% (*), 5% (**) and 1% (***) levels.

Table B.4: VSL and VSLY Estimates by Age for Males and Females

	male			female		
	VSL	VSLY		VSL	VSLY	
Age		3% discount	7% discount		3% discount	7% discount
67	260,904	19,837	26,742	528,917	36,221	50,532
68	246,966	19,410	25,907	519,140	36,677	50,625
69	232,789	18,937	25,024	490,645	35,674	48,776
70	205,763	17,349	22,699	403,258	30,354	41,048
71	176,659	15,462	20,030	362,179	28,150	37,697
72	174,812	15,907	20,404	317,122	25,624	33,926
73	156,340	14,816	18,818	292,854	24,531	32,154
74	144,655	14,301	17,986	265,266	23,085	29,951
75	120,096	12,406	15,453	213,372	19,338	24,830
76	109,743	11,865	14,639	191,382	18,110	23,010
77	95,056	10,776	13,171	180,766	17,912	22,517
78	93,301	11,112	13,457	174,803	18,197	22,630
79	86,224	10,809	12,971	157,665	17,172	21,156
80	72,024	9,522	11,325	129,189	14,766	18,019
81	68,922	9,628	11,352	118,230	14,229	17,198
82	63,562	9,401	10,991	109,484	13,927	16,670
83	54,333	8,523	9,883	99,651	13,456	15,950
84	52,276	8,717	10,027	88,994	12,678	14,904
85	51,609	9,171	10,469	80,811	12,340	14,362
86	44,077	8,353	9,465	71,298	11,593	13,380
87	40,113	8,116	9,133	61,120	10,490	12,024
88	35,795	7,739	8,652	62,148	11,472	13,038
89	40,245	9,306	10,340	62,104	12,213	13,784
90	31,254	7,733	8,543	47,550	10,012	11,222
91	21,504	5,696	6,258	38,784	8,796	9,790
92	22,956	6,510	7,116	35,824	8,629	9,554
93	16,924	5,137	5,590	37,818	9,940	10,928
94	19,586	6,362	6,892	34,704	9,799	10,715
95	14,500	5,037	5,434	26,508	7,878	8,584
96	14,929	5,540	5,956	23,409	7,557	8,189
97	11,419	4,522	4,845	29,332	10,050	10,852

Note: All measures are reported in constant year 2010 dollars. See the main text for explanation of the underlying calculations.

Table B.5: Sample Selection

	Observations Left
Total person years in MCBS 2005-2011	101,703
Drop if employed	90,423
Keep observations for the ages 66-95	72,795
Drop if medical expenditures = 0 or > $ 100,000 or missing	53,384
Drop if missing information on health measures	35,426

Table B.6: Summary Statistics of Key Variables

Measures	Mean	Std. Dev.	N
Health Measures			
Raw HCC Scores	1.10	0.73	35,426
Self-reported Health Status (1 : Poor, 5 : Excellent)	3.43	1.06	35,426
$\mathbb{1}\{18.5 \leq BMI \leq 25\}$	0.39	0.49	35,426
Number of ADL conditions (0 : None ; Max : 6)	0.49	1.07	35,426
Number of IADL conditions (0 : None; Max: 5)	0.54	1.03	35,426
Measures of Unhealthy Behavior			
Smoking	0.09	0.29	35,426
Ever Smoked	0.56	0.50	35,426
Health Expenditure			
Medical Spending (2010 $)	8,012	13,494	35,426
Out-of-Pocket Cost (2010 $)	1,310	1,787	35,426
Demographics			
Age	76.83	6.92	35,426
Male	0.40	0.49	35,426
Non-Hispanic White	0.88	0.33	35,237
African-American / Black	0.07	0.25	35,237
Hispanic	0.04	0.19	35,237

Table B.7: Estimates of Measurement Parameters and Percent of Total Variation in Measurements Captured by Health Factor

		Estimates		Percent	
Age Group		λ	μ	Signal	Noise
	HCC Score	1.000	-1.897	76.51	23.49
	Health Status (5: excellent, 1: poor)	0.607	2.760	12.81	87.19
Age 66-70	ADL Conditions (6 : No ADL)	0.401	5.150	6.40	93.60
	IADL Conditions (5 : No IADL)	0.353	4.202	7.08	92.92
	BMI ([18.5,25] = 1)	0.052	0.265	0.55	99.45
	HCC Score	1.000	-2.025	82.43	17.57
	Health Status (5 : excellent, 1 :poor)	0.546	2.854	12.43	87.57
Age 71-75	ADL Conditions (6 :No ADL)	0.368	5.162	5.99	94.01
	IADL Conditions (5 :No IADL)	0.336	4.191	6.79	93.21
	BMI ([18.5,25] = 1)	0.038	0.302	0.32	99.68
	HCC Score	1.000	-2.203	81.94	18.06
	Health Status [5 : excellent, 1: poor]	0.485	2.871	10.25	89.75
Age 76-80	ADL Conditions (6 : No ADL)	0.359	5.083	4.90	95.10
	IADL Conditions (5 : No IADL)	0.312	4.126	5.23	94.77
	BMI ([18.5,25] = 1)	0.039	0.346	0.32	99.68
	HCC Score	1.000	-2.326	79.45	20.55
	Health Status [5 : excellent, 1 : poor]	0.471	2.813	8.66	91.34
Age 81-85	ADL Conditions (6 : No ADL)	0.577	4.588	7.81	92.19
	IADL Conditions (5 : No IADL)	0.457	3.737	7.09	92.91
	BMI ([18.5,25] = 1)	0.076	0.355	1.07	98.93
	HCC Score	1.000	-2.405	83.82	16.18
	Health Status (5 : excellent, 1 : poor)	0.367	2.976	4.92	95.08
Age 86-95	ADL Conditions (6 : No ADL)	0.398	4.548	2.94	97.06
	IADL Conditions (5 : No IADL)	0.342	3.592	2.80	97.20
	BMI ([18.5,25] = 1)	0.046	0.468	0.36	99.64

Table B.8: Elasticity of VSL with Respect to Permanent and Non-asset Annual Income

	(1)	(2)	(3)	(4)
	Everyone	Age 66-75	Age 76-85	Age 86-95
Elasticity of VSL	0.698 ***	0.814 ***	0.708 ***	0.569 ***
wrt permanent income	(0.008)	(0.0081)	(0.011)	(0.020)
Elasticity of VSL	0.952 ***	1.115 ***	0.979 ***	0.849 ***
wrt non-asset annual income	(0.013)	(0.011)	(0.016)	(0.028)

Note: All regression are estimate using simulated data. Each coefficient are estimates from separate regressions. All specifications control for current health and asset holdings.

Table B.9: Avoided Cases and Estimated PM 2.5 Benefits for Clean Air Act in 2010

Health Endpoints	Age Group	Cases Avoided in 2010	Benefits (million $ 2010)	
			EPA	This study
	< 65	41,000	331,000	331,000
	$65 - 74$	31,000	250,000	**140,000**
Mortality	$75 - 84$	42,000	343,000	**96,000**
	85+	47,000	380,000	**42,000**
	Total	160,000	1,300,000	**610,000**
Morbidity :				
	< 65	43,000	21,000	21,000
	$65 - 74$	6,000	2,700	**3,100**
Chronic Bronchitis	$75 - 84$	3,500	1,600	**1,300**
	85+	1,500	690	**410**
	Total	54,000	26,000	**25,000**
	< 65	48,000	6,500	6,500
Non-fatal	$65 - 74$	44,000	4,500	**4,000**
Myocardial	$75 - 84$	27,000	2,500	**2,000**
Infarction	85+	11,000	1,000	**690**
	Total	130,000	15,000	**13,000**
Other Morbidity Benefits*			9,000	9,000
Total Morbidity Benefits			50,000	47,000

* These benefits are estimated by EPA for individuals under the age of 65.
Notes: All the numbers are rounded up to 2 significant figures. The results in bold are obtained using the WTP for mortality and morbidity results from this study. The age-group distribution for each health end-points are obtained using EPA's concentration response functions and the reported incidences in US EPA (2011). Age-group estimates for individuals under 65 are directly obtained from US EPA (2011) and for population over 65, age-bin averages from this study are applied. All results for benefits are expressed in 2010 US million $.

Table B.10: Avoided Cases and Estimated Ozone Benefits for Clean Air Act in 2010

Health Endpoints	Age Group	Cases Avoided in 2010	Benefits (million $ 2010)	
			EPA	This study
Mortality	< 65	1,100	8,600	8,600
	$65 - 74$	810	6,600	**3,600**
	$75 - 84$	1,100	9,200	**2,600**
	85+	1,300	10,000	**1,100**
	Total	4,300	35,000	**16,000**
Total Morbidity Benefits*			1,300	1,300

* These benefits are estimated by EPA for individuals under the age of 65.
All the numbers are rounded up to 2 significant figures. The results in bold are obtained using the WTP for mortality and morbidity results from this study. The age-group distribution for each health end-points are obtained using EPA's concentration response functions and the reported incidences in US EPA (2011). Age-group estimates for individuals under 65 are directly obtained from US EPA (2011) and for population over 65, age-bin averages from this study are applied. All results for benefits are expressed in 2010 US $.

Table B.11: Comparison of Key Variables Between the Estimating Sample and Full Sample in Wave III

Variable	Estimating Sample	Everyone in Wave III
Demographics:		
Age (Years)	21.7	22.0
% Male	41.3	47.2
% White	71.5	68.7
% Black	21.3	23.3
% Native American	7.3	7.7
% Asian	10.7	8.9
Years of Education	13.3	13.2
Personal Income	12,450	14,054
Depression & Substance Use:		
CES-D Score	4.7	4.6
% with CES-D > 0	90.0	88.0
% using illicit drug	23.1	21.2
% of binge drinkers	32.4	31.6
% interviewed after 9/11	47.0	78.0

Note: The table provides a comparison of selected statistics for the estimating sample and the full Wave III sample. The estimating sample includes all interviews conducted 30 days before and after 9/11.

Table B.12: Difference in Means of Outcome Variables Between Those Interviewed Before and After 9/11

Variable	Difference in Means 30 days before and after 9/11	Std. Error
Outcome Variables		
Any illicit drug	0.038***	0.012
Marijuana	0.037***	0.013
Cocaine	0.009*	0.049
Methamphetamine	0.004	0.004
Injections	0.003	0.002
Other (LSD, Ecstasy, Heroin etc.)	0.017***	0.006
Bi-weekly Frequency of Binge Drinking	0.165***	0.055

Note: The table reports the difference in means of outcomes variables for interviews conducted before and after 9/11. The difference in mean is estimated using a regression of the variables on a dummy variable, 9/11 which takes the value of 1 if the interview is conducted on or after 9/11. The table reports the estimated coefficients on the dummy, "9/11"

Table B.13: Difference in Means of Components of CES-D Index Before and After 9/11

	Control Group Mean	Difference in Means (Before and After 9/11)	Std. Error	CI (95 %)
Bothered by things	0.524	0.064	0.021	[0.024,0.105]
Couldn't Shake of Blues	0.334	0.025	0.019	[-0.012,0.063]
Felt as good as others	0.730	0.031	0.027	[-0.022, 0.084]
Trouble Concentrating	0.596	0.067	0.021	[0.025, 0.109]
Depressed	0.333	0.046	0.019	[0.009, 0.084]
Too Tired to do things	0.655	0.004	0.021	[-0.038,0.045]
Enjoyed Life	0.630	0.058	0.024	[0.011,0.105]
Sad	0.485	0.151	0.020	[0.110,0.191]
People Dislike you	0.240	0.012	0.015	[-0.017,0.043]

Note: The table reports the difference in means of different items on the CES-D index for interviews conducted before and after 9/11. The difference in mean is estimated using a regression of the variables on a dummy variable, 9/11 which takes the value of 1 if the interview is conducted on or after 9/11. The table reports the estimated coefficients on the dummy, "9/11"

Table B.14: Unabridged OLS Estimates of the Effects of Depressive Symptoms on Illicit Drug Use

	Outcomes: Drug Consumption last 30 days						
	Ever Consumed Any	Marijuana	Cocaine	Methamphetamine	Inject	Others	Alcohol
CES-D	0.035***	0.034***	0.006**	0.004*	0.003*	0.005	0.099***
	(0.006)	(0.006)	(0.003)	(0.002)	(0.001)	(0.003)	(0.030)
Male	0.080***	0.075***	0.009*	0.000	0.000	0.028***	0.687***
	(0.013)	(0.012)	(0.005)	(0.000)	(0.000)	(0.006)	(0.057)
PPVT	0.031***	0.031***	-0.001	-0.004	-0.003	0.009***	-0.020
	(0.006)	(0.006)	(0.003)	(0.003)	(0.002)	(0.003)	(0.033)
Years of Education	-0.011***	-0.010***	-0.001	-0.002*	-0.000	-0.001	0.009
	(0.003)	(0.003)	(0.002)	(0.001)	(0.001)	(0.002)	(0.015)
Religiosity	-0.080***	-0.077***	-0.014***	-0.005***	-0.000	-0.018***	-0.236***
	(0.005)	(0.005)	(0.002)	(0.002)	(0.001)	(0.003)	(0.023)
N Obs	4,634	4,635	4,646	4,648	4,648	4,646	4,625

Notes: CES-D denotes the continuous index of depressive symptoms used in this study and the measure is standardized. The outcome variables include: whether the individual consumes any illicit drug, any specific type of drug like marijuana, cocaine or methamphetamine etc, and biweekly frequency of 5+ alcoholic drinks per day. In each columns, I control for: gender, race, cognitive abilities (PPVT test score), years of education and an index of religiosity. The others variable is a composite category indicating whether the individual consumes LSD, ecstasy, Heroin etc. Asterisks indicate statistical significance at the 1% (***), 5% (**) and 10% (*) levels. Robust standard errors are in parenthesis.

Table B.15: Unabridged 2SLS estimates of the Effects of Depressive Symptoms on Illicit Drug Use

	Outcomes: Drug Consumption last 30 days						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Ever Consumed Any	Marijuana	Cocaine	Methamphetamine	Inject	Others	Alcohol
	Panel A: First Stage						
Terror Attack 9/11	0.119	0.119	0.118	0.118	0.118	0.119	0.119
	(0.028)	(0.028)	(0.028)	(0.028)	(0.028)	(0.028)	(0.028)
F-Stat	17.500	17.529	17.390	17.390	17.402	17.615	17.504
	Panel B: Second Stage						
CES-D	0.321***	0.309**	0.066	0.026	0.027*	0.154**	1.166**
	(0.122)	(0.119)	(0.045)	(0.032)	(0.016)	(0.063)	(0.524)
Male	0.146***	0.139***	0.023*	0.015**	0.011**	0.063***	0.937***
	(0.032)	(0.031)	(0.012)	(0.008)	(0.005)	(0.017)	(0.147)
PPVT	0.068***	0.066***	0.006	-0.002	0.000	0.028***	0.119
	(0.018)	(0.018)	(0.007)	(0.005)	(0.002)	(0.009)	(0.078)
Years of Education	0.006	0.007	0.002	-0.001	0.001	0.008*	0.073**
	(0.009)	(0.008)	(0.003)	(0.002)	(0.001)	(0.004)	(0.034)
Religiosity	-0.064***	-0.062***	-0.011***	-0.003	0.001	-0.010**	-0.178***
	(0.009)	(0.009)	(0.003)	(0.002)	(0.001)	(0.005)	(0.041)
N Obs	4,634	4,635	4,646	4,648	4,648	4,646	4,625

Notes: The table reports the 2SLS estimates of effects of depressive symptoms on illicit drug use for individuals interviewed 30 days before and after 9/11 attack. In each columns, I control for: gender, race, cognitive abilities (PPVT test score), years of education and an index of religiosity. The others variable is a composite category indicating whether the individual consumes LSD, ecstasy, Heroin etc. Asterisks indicate statistical significance at the 1% (***), 5% (**) and 10% (*) levels. Robust standard errors are in parenthesis.

Table B.16: Placebo Effects: 30 days Before and After September 11, 2008

	Outcomes: Drug Consumption last 30 days	
	Marijuana	Favorite Drug
	Panel A: First Stage	
Terror Attack 9/11	0.127	0.138
	(0.146)	(0.147)
F-Stat	0.758	0.882
	Panel B: Second Stage	
CES-D	-0.060	0.001
	(0.125)	(0.177)
N Obs	1,178	1,174

Notes: Data used to replicate the 2SLS estimates comes from Wave IV interviews. The cut-off date chosen is September 11, 2008

Table B.17: 2SLS Estimates of the Effects of Depressive Symptoms on Illicit Drug Use for Alternative Time Boundary

	Outcomes: Drug Consumption last 30 days						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Ever Consumed Any	Marijuana	Cocaine	Methamphetamine	Inject	Others	Alcohol
	Panel A: First Stage						
Terror Attack 9/11	0.117	0.116	0.115	0.115	0.115	0.116	0.116
	(0.030)	(0.030)	(0.030)	(0.030)	(0.030)	(0.030)	(0.030)
F-Stat	15.237	14.980	14.859	14.859	14.853	15.054	14.940
	Panel B: Second Stage						
CES-D	0.367***	0.350***	0.059	0.030	0.035*	0.164**	1.296 **
	(0.140)	(0.136)	(0.048)	(0.035)	(0.020)	(0.070)	(0.578)
N Obs	4306	4307	4318	4320	4320	4318	4299

Notes: The table reports the 2SLS estimates of effects of depressive symptoms on illicit drug use for an alternative time boundary using individuals interviewed 30 days before and after 9/11 attack excluding first week after the attack. In each columns, I control for: gender, race, cognitive abilities (PPVT test score), years of education and an index of religiosity. The others variable is a composite category indicating whether the individual consumes LSD, ecstasy, Heroin etc. Asterisks indicate statistical significance at the 1% (***), 5% (**) and 10% (*) levels. Robust standard errors are in parenthesis.

Table B.18: 2SLS Estimates of the Effects of Depressive Symptoms on Illicit Drug Use for Alternative CES-D Index

	Outcomes: Drug Consumption last 30 days						
	Ever Consumed Any	Marijuana	Cocaine	Methamphetamine	Inject	Others	Alcohol
	Panel A: First Stage						
Terror Attack 9/11	0.139***	0.140***	0.140***	0.140***	0.140***	0.141***	0.141***
	(0.029)	(0.029)	(0.029)	(0.029)	(0.029)	(0.029)	(0.029)
F-Stat	22.524	23.051	22.904	22.904	22.979	23.099	23.018
	Panel B: Second Stage						
CES-D	0.267***	0.257***	0.055	0.022	0.022*	0.127**	0.996**
	(0.098)	(0.096)	(0.037)	(0.027)	(0.014)	(0.051)	(0.431)
Male	0.136***	0.129***	0.021*	0.014*	0.010**	0.058***	0.902***
	(0.027)	(0.027)	(0.011)	(0.007)	(0.004)	(0.015)	(0.128)
PPVT	0.045***	0.044***	0.002	-0.003	-0.002	0.017***	0.037
	(0.011)	(0.011)	(0.004)	(0.003)	(0.002)	(0.006)	(0.047)
Years of Education	-0.000	0.000	0.001	-0.001	0.001	0.005	0.051*
	(0.006)	(0.006)	(0.002)	(0.002)	(0.001)	(0.003)	(0.025)
Religiosity	-0.074***	-0.071***	-0.013***	-0.004**	0.000	-0.015***	-0.211***
	(0.007)	(0.007)	(0.003)	(0.002)	(0.001)	(0.004)	(0.030)
N Obs	4,648	4,649	4,660	4,662	4,662	4,660	4,639

Notes: The table reports the 2SLS estimates of effects of depressive symptoms on illicit drug use for an alternative depression index. Here, I consider a subset of questions as used by Wang and Yang (2013) in their study of depressive symptoms on dietary and physical intake among young adults. In each columns, I control for: gender, race, cognitive abilities (PPVT test score), years of education and an index of religiosity. The others variable is a composite category indicating whether the individual consumes LSD, ecstasy, Heroin etc. Asterisks indicate statistical significance at the 1% (***), 5% (**) and 10% (*) levels. Robust standard errors are in parenthesis.

Table B.19: 2SLS Estimates of the Effects of Depressive Symptoms on Illicit Drug Use for both Alternative Time and CES-D Index

	Outcomes: Drug Consumption last 30 days						
	Ever Consumed Any	Marijuana	Cocaine	Methamphetamine	Inject	Others	Alcohol
	Panel A: First Stage						
Terror Attack 9/11	0.128***	0.128***	0.127***	0.127***	0.127***	0.128***	0.128***
	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)
F-Stat	17.264	17.303	17.182	17.182	17.232	17.350	17.272
	Panel B: Second Stage						
CES-D	0.326***	0.311***	0.053	0.027	0.032*	0.144**	1.179**
	(0.122)	(0.118)	(0.043)	(0.031)	(0.017)	(0.061)	(0.513)
Male	0.148***	0.139***	0.018	0.015*	0.013**	0.062***	0.942***
	(0.034)	(0.033)	(0.012)	(0.009)	(0.006)	(0.018)	(0.151)
PPVT	0.053***	0.052***	0.000	-0.003	-0.001	0.020***	0.046
	(0.014)	(0.013)	(0.004)	(0.004)	(0.002)	(0.007)	(0.056)
Years of Education	0.003	0.003	0.001	-0.001	0.001	0.005	0.071**
	(0.008)	(0.007)	(0.003)	(0.002)	(0.001)	(0.004)	(0.030)
Religiosity	-0.074***	-0.071***	-0.013***	-0.004**	0.000	-0.015***	-0.226***
	(0.008)	(0.008)	(0.003)	(0.002)	(0.001)	(0.004)	(0.033)
N Obs	4,320	4,321	4,332	4,334	4,334	4,332	4,313

Notes: The table reports the 2SLS estimates of effects of depressive symptoms on illicit drug use considering both the alternative time window and CES-D index. In each columns, I control for: gender, race, cognitive abilities (PPVT test score), years of education and an index of religiosity. The others variable is a composite category indicating whether the individual consumes LSD, ecstasy, Heroin etc. Asterisks indicate statistical significance at the 1% (***), 5% (**) and 10% (*) levels. Robust standard errors are in parenthesis.

Table B.20: 2SLS Estimates of the Effects of Depressive Symptoms by Demographic Groups

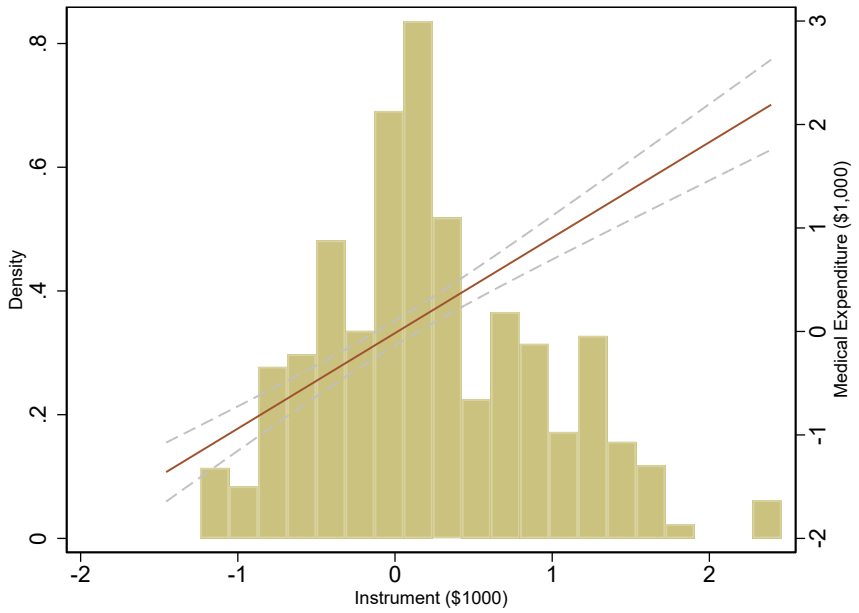
	First-Stage	Outcomes: Drug Consumption last 30 days						
		Ever Consumed Any	Marijuana	Cocaine	Methamphetamine	Inject	Others	Alcohol
				Panel (a): Gender Differences				
Female	0.1064***	0.360**	0.352**	0.012	0.033	0.002	0.130	0.468
	(0.040)	(0.185)	(0.182)	(0.057)	(0.041)	(0.014)	(0.080)	(0.539)
Male	0.134***	0.266*	0.247	0.120*	0.016	0.053*	0.182*	1.817**
	(0.040)	(0.160)	(0.155)	(0.073)	(0.049)	(0.030)	(0.097)	(0.915)
				Panel (b): Age Differences				
(Age≤21)		0.233	0.220	0.141	0.027	0.050	0.205*	2.174**
		(0.174)	(0.171)	(0.086)	(0.046)	(0.032)	(0.114)	(1.043)
(Age>21)		0.399**	0.388**	0.003	0.024	0.008	0.116	0.315
		(0.180)	(0.175)	(0.054)	(0.045)	(0.018)	(0.073)	(0.608)
				Panel (c): Race Differences				
White	0.141***	0.289**	0.266**	0.093	-0.001	0.006	0.139*	1.234**
	(0.028)	(0.135)	(0.132)	(0.060)	(0.036)	(0.018)	(0.073)	(0.614)
Non-White	0.091***	0.469	0.481	0.012	0.074	0.067	0.194	1.227
	(0.038)	(0.310)	(0.308)	(0.071)	(0.074)	(0.043)	(0.137)	(1.083)
				Panel (d): PPVT Differences				
Low PPVT	0.080***	0.212	0.244	0.073	0.036	0.101	0.229	2.919
	(0.047)	(0.258)	(0.259)	(0.106)	(0.085)	(0.075)	(0.166)	(2.009)
High PPVT	0.140***	0.380***	0.349***	0.069	0.023	0.000	0.132*	0.568
	(0.035)	(0.140)	(0.134)	(0.049)	(0.032)	(0.012)	(0.069)	(0.493)

Notes: Column (1) reports the first-stage coefficient of 9/11 terrorist attack on the CES-D index and Columns (2)-(7) report the second-stage coefficient on different types of drugs and alcohol. Each panel reports the coefficients estimates for each group. The others variable is a composite category indicating whether the individual consumes LSD, ecstasy, Heroin etc. Asterisks indicate statistical significance at the 1% (***), 5% (**) and 10% (*) levels. Robust standard errors are in parenthesis.

APPENDIX C

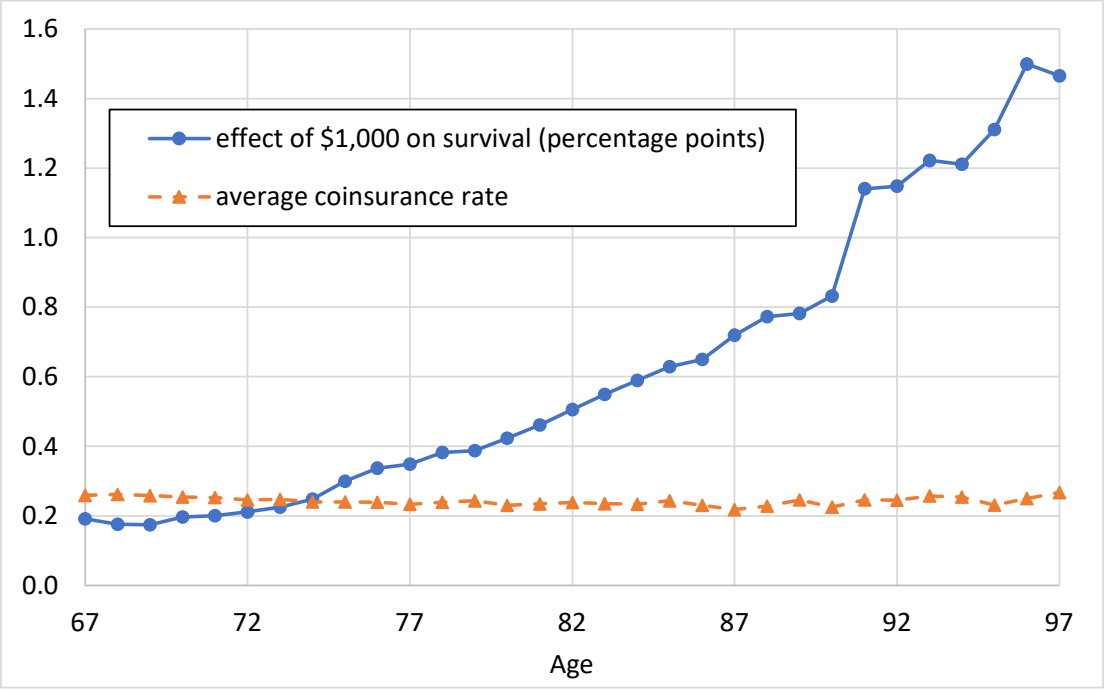
SUPPLEMENTARY FIGURES

Figure C.1: Identifying Variation in the Instrument



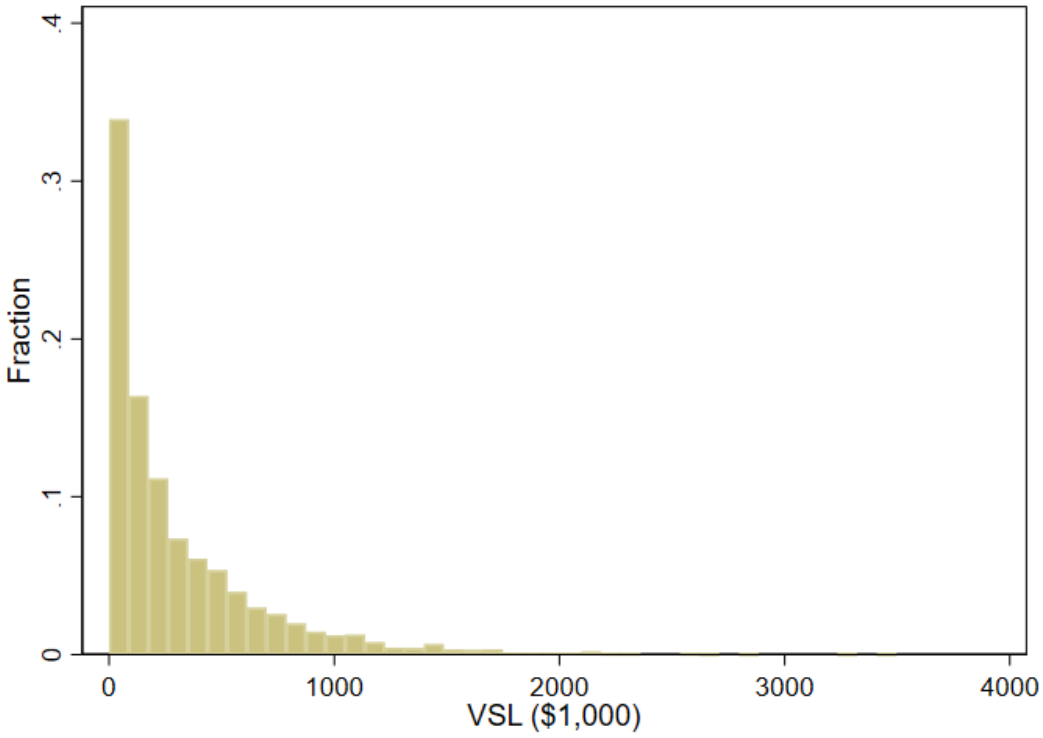
Notes: The histogram shows the variation in medical spending due to place effects estimated for 306 hospital referral regions for 2005–2011. The right vertical axis plots conditional variation in medical spending against conditional variation in the instrument after removing the variation in each that is explained by individual measures of health and age. Dashed lines show 95% confidence intervals on predicted values.

Figure C.2: Coinsurance Rate and Return to Spending: Age 67 to Age 97



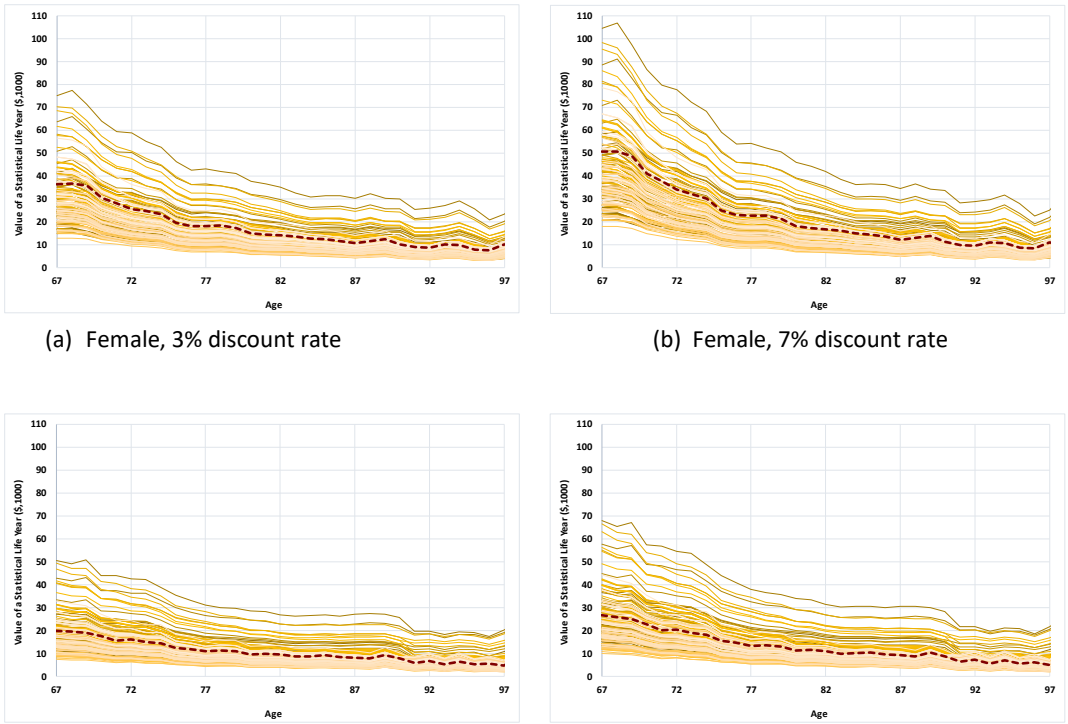
Notes: The dashed line shows the average coinsurance rate from the data, i.e., the ratio of out-of-pocket to total medical expenditures. The solid line shows the average marginal effect of a $1,000 increase in medical spending on the probability of surviving to the end of the following year measured in percentage points and calculated from the model shown in col (3) of Table 2.2.

Figure C.3: Heterogeneity in the VSL at Age 70



Notes: The histogram shows the variation in VSL estimates based on 2,698 people who we observe at age 70. Conditional on age, the VSL differs across person–types due to differences in their health and demographics..

Figure C.4: Sensitivity of VSLY Estimates to Model Features



(a) Female, 3% discount rate

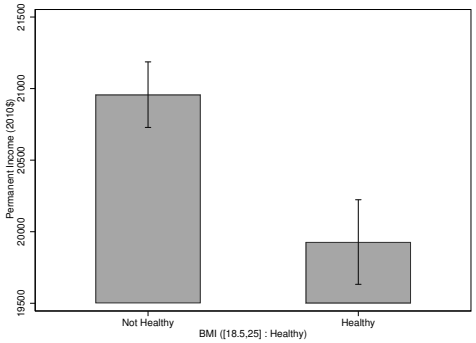
(b) Female, 7% discount rate

(c) Male, 3% discount rate

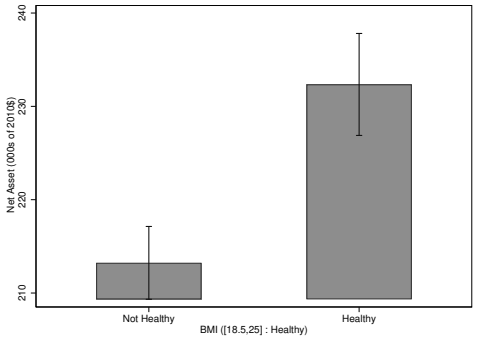
(d) Male, 7% discount rate

Notes: The figures show the estimated mean VSLY by age, gender, and discount rate for each of 200 different specifications of the survival function. Each line corresponds to a different combination of modeling decisions as described in the main text. The large dashed line is our main specification from col (3) of Table 2.2.

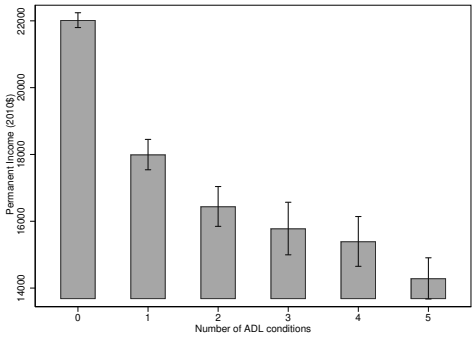
Figure C.5: Permanent Income and Net Asset Distribution by Other Health Measures



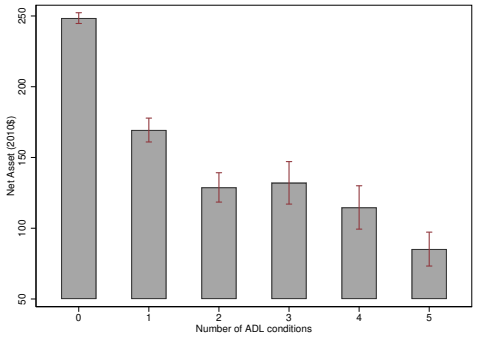
(a) Permanent Income and BMI



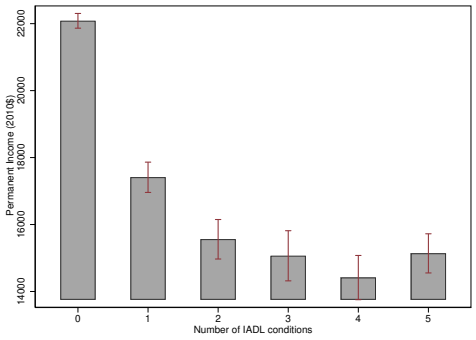
(b) Net Asset and BMI



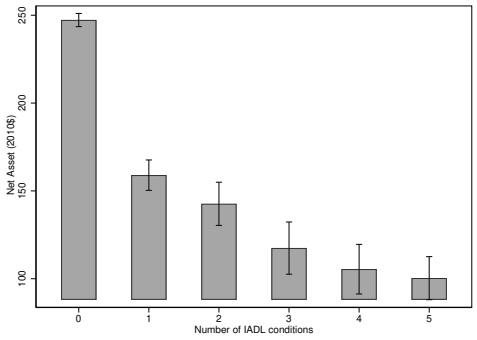
(c) Permanent Income and ADL Count



(d) Net Asset and ADL Count



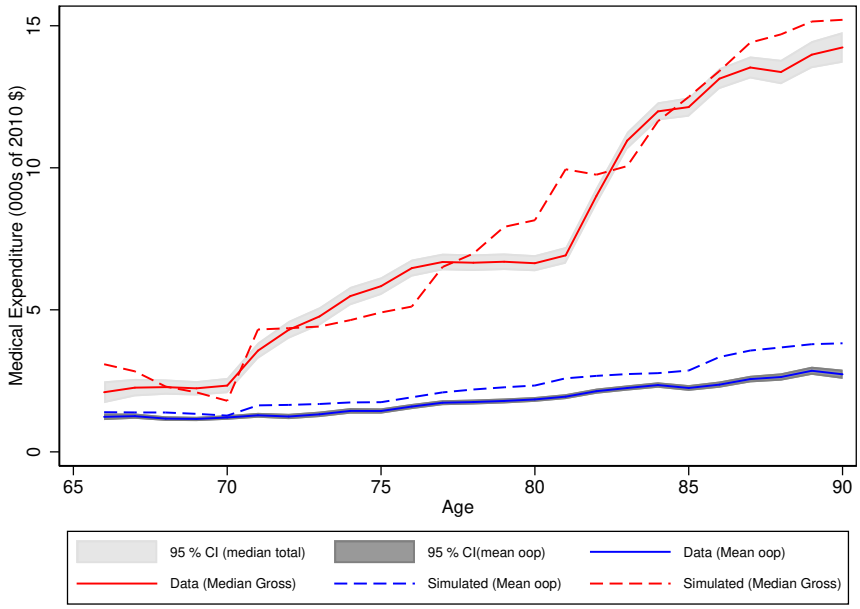
(e) Permanent Income and IADL Count



(f) Net Asset and IADL Count

Notes: All figures use the health measures and financial variables reported in the HRS. The BMI variable is a dummy variable for having BMI in the healthy range of 18. 5 to 25.

Figure C.6: Non-targeted Moment



Notes: The solid and dashed line represents the data and simulated profiles respectively. The red lines show the median medical expenditures over the life-cycle and the blues line represent the mean out-of-pocket costs over the life-cycle. The data values are adjusted to 2010 US $ using the CPI.

Figure C.7: Distribution of VSL at Age 70