This product is part of the Pardee RAND Graduate School (PRGS) dissertation series. PRGS dissertations are produced by graduate fellows of the Pardee RAND Graduate School, the world's leading producer of Ph.D.'s in policy analysis. The dissertation has been supervised, reviewed, and approved by the graduate fellow's faculty committee.

DISSERTATION

Planning for an Aging Nation

New Estimates to Inform Policy Analysis for Senior Health

Adam H. Gailey

This document was submitted as a dissertation in March 2012 in partial fulfillment of the requirements of the doctoral degree in public policy analysis at the Pardee RAND Graduate School. The faculty committee that supervised and approved the dissertation consisted of Nicole Maestas (Chair), Pierre-Carl Michaud, and Michael Hurd.

PARDEE RAND GRADUATE SCHOOL

The Pardee RAND Graduate School dissertation series reproduces dissertations that have been approved by the student's dissertation committee.

The RAND Corporation is a nonprofit institution that helps improve policy and decisionmaking through research and analysis. RAND's publications do not necessarily reflect the opinions of its research clients and sponsors.

Published 2012 by the RAND Corporation
1776 Main Street, P.O. Box 2138, Santa Monica, CA 90407-2138
1200 South Hayes Street, Arlington, VA 22202-5050
4570 Fifth Avenue, Suite 600, Pittsburgh, PA 15213-2665
RAND URL: http://www.rand.org
To order RAND documents or to obtain additional information, contact
Distribution Services: Telephone: (310) 451-7002;
Fax: (310) 451-6915; Email: order@rand.org

Planning for an Aging Nation: New Estimates to Inform Policy Analysis for Senior Health[1]

Contains:

Gailey, Adam. *A Structural Analysis of the Role of Restaurants and Exercise in Obesity among the Elderly.*

Michaud, PC, D. Goldman, D. Lakdawalla, A. Gailey, and Y. Zheng*. 2011. Differences in health between Americans and Western Europeans: Effects on longevity and public finance. Social science & medicine.*

Gailey, Adam H., Darius Lakdawalla, and Neeraj Sood*. 2010. Patents, Innovation, and the Welfare Effects of Medicare Part D. In Pharmaceutical Markets and Insurance Worldwide, edited by A. Dor: Emerald Group Publishing Limited.*

Dissertation Committee:

Nicole Maestas (chair)
Pierre-Carl Michaud
Michael Hurd

[1] Each sub-paper contains its own acknowledgements though for overall support, I am thankful to PRGS and Anne and James Rothenberg for the Anne and James Rothenberg Dissertation Award.

Abstract

This dissertation contains three papers on the health and welfare of the elderly population. Overall, these papers provide insights into the costs and challenges of providing health care to the elderly population. These papers help us understand the effects of obesity on longevity and health care, as well as better understand the benefits of social insurance. The first paper uses a micro-simulation model to estimate the longevity effects of poor health trends among younger Americans, and finds that difference in these trends can explain 92% of the difference between US and European longevity. The second paper estimates the welfare effects of Medicare Part-D from gains in market efficiency and dynamic incentives for pharmaceutical companies. It finds that these gains alone nearly cover the welfare cost of funding Medicare Part-D. The last paper presents and estimates a structural model of health, exercise, and restaurant consumption. It provides estimates for future welfare analyses of programs targeting obesity through restaurants and exercise in the elderly population. It also estimates the long run effects of making policies which make restaurant food healthier. It finds only minor effects of restaurant policies on health for the elderly. Overall, these papers further our understanding of the challenging objective of improving senior health while containing costs.

Paper 1: A Structural Analysis of the Role of Restaurants and Exercise in Obesity among the Elderly[1]

Adam H. Gailey
Pardee RAND Graduate School

Abstract

This research creates and estimates a structural model of consumption, savings, obesity, eating habits, health, and exercise for the elderly population. Individuals face tradeoffs between consuming unhealthy goods today, which they enjoy, and having increased disease risk in the future. Welfare analysis then must include the potential loss in utility of the policies which alter obesity from the implied socially optimal level. Using the Health and Retirement Study data I estimate strong preferences for eating outside of the home and large penalties for gaining weight. I find that unhealthy food at restaurants may account for as much as a 1.88% increase in the average BMI of elderly Americans and a 3.2% increase in the prevalence of hypertension. Over time, then, policies targeting the quality of food at restaurants may have a substantial impact on the health of Americans. Furthermore, I find that policies increasing the cost of health care for the elderly will do little to incentivize behavioral changes that affect obesity.

[1] I am thankful for support from PRGS and Anne and James Rothenberg through the Anne and James Rothenberg Dissertation Award and his publication was made possible by Grant Number R01AG030824 from NIA. Its contents are solely the responsibility of the author and do not necessarily represent the official views of the (NIA or NIH).

1. Introduction

In choosing their eating and exercise behaviors, many individuals sacrifice long-term health benefits for short term pleasure. These choices have led to a large increase in the obesity of elderly Americans, while at the same time reducing obesity has become a key public health goal due to its link to numerous diseases (NIH, 2011). Much is known about the costs of obesity through its links to chronic diseases like diabees, hypertension, heart disease, and stroke, as well as gallstones (Sturm, 2002; Field *et al*., 2001; Must *et al*., 1999) and mortality Calle *et al.* (1999). However, we know little about the benefits to individuals of the activities that lead to obesity. I develop a model for the elderly with incentives for risky behaviors (like eating out at restaurants or avoiding exercise) and savings in a world with uncertainty, which allows me to look at the welfare implications of policies. Some policies aimed at reducing obesity (such as increasing the relative cost of restaurant-prepared food) may not be welfare enhancing as they may reduce the pleasure individuals derive from the consumption of health-inhibiting goods.

The paper proceeds in the following manner: Section 2 provides background on obesity and structural models of health. Section 3 presents the model for this paper. Section 4 provides an estimation of the model. Section 5 assesses the performance of the model in fitting observed patterns. Section 6 presents policy simulations based on improved quality of food in restaurants and increased cost of health care. Section 7 concludes.

2. Background

2.1 Obesity, restaurants, exercise and the elderly

As the association between obesity and chronic disease was established, several studies noted that obesity is on the rise (Ogden *et al.*, 2006; Flegal *et al.*, 1998) and that the rise accounts for some of the increases in per capita health care costs (Duchovny and Baker, 2010). Hence many policymakers are looking for ways to reduce this epidemic. These concerns have increased the pressure to find ways to reduce obesity.

One established method of reducing individuals' risk of the diseases mentioned above is weight loss. Sjöström *et al.* (2000) find that individuals who have lost weight have reductions in their likelihood of getting diabetes and hypertension. Tuomilehto *et al.* (2001) use a randomized controlled trial to find large reductions in the risk of diabetes from behavioral changes to reduce weight. Two mechanisms to reduce weight are increased exercise and reduced caloric intake.

The link between exercise and obesity has been long established, but the reductions in weight tend to be small unless there is a substantial increase in the amount of exercise (Bouchard *et al.*, 1993; Slentz *et al.* 2004; Jakicic *et al*. 2003, Fontana *et al*. 2007). While a relatively weak method for weight loss, exercise still does have a direct effect on diseases such as heart disease and hypertension (NIH, 1998). Due to the relatively weak relationship between exercise and weight loss, the NIH guidelines on treating obesity call for the reduction of calories as the

primary method for weight-loss treatment (NIH, 1998). As a result, an increasing number of studies have begun to look at reducing caloric intake (e.g. Redman *et al*. 2007 or Fontana and Klein, 2007).

Guthrie *et al.* (2002) find that over the past 30 years, the proportion of food consumption that occurs in restaurants has gone up significantly and suggest that this may be related to the increase in obesity over the same time period. This, along with new policies targeting restaurants (and in particular fast food restaurants), has lead researchers to focus on the difference between calories consumed in restaurants and those at home. Nielsen and Popkin (2005) use the Nationwide Food Consumption Survey and the Continuing Survey of Food Intake to find that for most types of meals, with the exception of pizza, portion sizes at fast food restaurants have increased faster than for meals at home.

While these studies seem to implicate restaurants as a key area of concern, there is conflicting evidence on the relationship between eating in restaurants and obesity. Several studies have found no evidence that increased restaurant availability results in increased obesity (Davis and Carpenter, 2009; Anderson and Matsa, 2009). Yet, others have found an association between those who visit restaurants more often (McCrory *et al.*, 1999) and those who live in higher restaurant density neighborhoods (Chou *et al.*, 2002) and body fat or obesity, directly conflicting with the others' results[2]. Currie *et al.* (2010), in perhaps one of the best studies, use detailed location data on restaurants and students to determine that students and their mothers in locations close to fast food restaurants have an increased risk of obesity.

[2] Differences in data quality and populations likely explain the opposing findings.

Even as researchers debate the role of restaurants in the obesity problem the federal government and many states and municipalities have implemented new policies to improve the health quality of restaurant food. These polices have taken many forms, from bans on trans-fatty acids in New York City or policies that allow the consumer to choose healthier options through the calorie labeling requirements in the federal "Patient Protection and Affordable Care Act" (2010). These efforts have all been to improve the health quality of food people eat when they are at restaurants. However, it is unknown what effect these regulations will have on obesity, diseases, and longevity, because, for example, people may substitute other unhealthy behaviors (such as reducing exercise) in response to eating healthier foods.

While this paper focuses on the role of restaurants and exercise on obesity and health, researchers posit other explanations for the determinants of obesity, including technological change (Lakdawalla and Philipson, 2009), social networks (Christakis and Fowler (2007)) and socio-economic status (see Sobal and Stunkard, 1989, and McLaren, 2007 for a review of this literature)

This paper focuses on the elderly population, which is of particular interest to policy makers for a number of reasons. Summers (2009) in a CRS Report for Congress identifies the growing trend of obesity even among the elderly, showing that for those aged 65 to 74 the prevalence of obesity has increased for males from 13% in the period 1976 to 1980 to 33% in 2005 to 2006. For the same time period females had an increase in obesity from 22% to 37%. This increase, in combination with the substantial government expenditures (e.g. Medicare and Social Security) on the elderly, which are contingent on their health, has created great policy interest. Keeler *et al*.

(1989) establish the high cost of obesity. Goldman *et al*. (2010) show the importance of the trend in obesity (and its related diseases) particularly among the elderly and near elderly on the fiscal position of government programs.

There is some concern that weight loss in the elderly may be detrimental to health; however treatment guidance from the NIH (2010) clearly indicates that obesity treatments should not be withheld due to age for those up to the age of 80. The guidance indicates the large benefits even to the elderly of obesity reduction. Indeed, even the share of bariatric surgeries for the elderly population has gone up from 1.9% in 2005 to 4.8% in 2009 (Dorman *et al*., 2011), showing that the elderly and their doctors are increasingly taking substantial steps to avoid deleterious effects of obesity.

2.2 Structural models of health

To assess long-run outcomes which incorporate welfare analysis where individuals may react to policy changes, researchers often use dynamic structural lifecycle models. Khwaja (2010) presents a dynamic life cycle model with choices of smoking, exercise, and alcohol consumption to estimate the willingness to pay for Medicare, estimated using the Health and Retirement Study data. This modeling is done in the context of evaluating the willingness to pay for Medicare among the elderly. Rashad (2006) develops a semi-structural model of obesity using data from the National Health and Nutrition Examination Surveys to estimate the effects of restaurant prices and exercise on obesity. Rashad finds that while caloric intake and exercise appear to have

an effect on obesity, when one controls for their endogeneity the effects are no longer significant. He does however find that restaurant prices have a statistically significant effect on obesity.

Fonseca *et al*. (2009) develop a dynamic retirement model in which individuals choose health expenditures as an investment in their health. De Nardi *et al*. (2010) and Palumbo (1999) also produce structural models accounting for health care expenditures and savings of the elderly. They use differential health risk to explain heterogeneity in savings behavior. Adda and Lechene (2004) estimate a model of smoking decisions and health. Similarly, Hurd (1989) estimates a structural retirement model to identify the magnitude of a bequest motive.

2.3 Contributions

This paper combines elements of all the approaches, but the key additional nuance to this model is that obesity is modeled as an implicit choice which is a result of behavior. Rather than choosing health expenditures, individuals choose health-inducing activities, such as exercise or consumption of food in the home, which have immediate utility costs. However, these decisions affect future obesity and disease, and thus healthcare expenditures and mortality risk. This paper also employs a refined treatment of diseases. Rather than using self-reported health or indices developed from diseases, I model numerous diseases independently. This is a similar treatment to that used in the Future Elderly Model developed by Goldman *et al*. (2004) and used in numerous studies over the past seven years to estimate the effects of health trends on the elderly (e.g. Joyce *et* al., 2004, and Michaud *et al*., 2011). These papers also argue for the importance of incorporating specific diseases.

The structural modeling approach allows me to determine the behavioral responses to changes in the health quality of restaurant food and cost of medical care. It models the effects of these changes on obesity, consumption, health, and longevity. I find that obesity levels can be reduced through changes in eating behavior (in this case restaurant versus in-home meals) and exercise. Simulating healthier restaurants results in a reduction of body mass index for males of 1.88% and for females of 1.25% by the time they are 80. This model focuses on the elderly, but as the effects are cumulative, if I were to consider younger individuals a larger effect might be possible over time. The paper also suggests that monetary incentives will not be as effective at increasing exercise as policies aimed at reducing the burden of exercise on the elderly.

3. Model

This project develops a structural model of health, wealth, and behavior for the elderly population, to understand the effect of restaurant policy changes. The model I present is philosophically similar to Khwaja (2010): Individuals elect to consume goods and activities that provide utility today but the effects of this consumption reduce utility in the future through increased medical expenditures and reduced health. This paper adds to Khwaja's work by incorporation restaurant decisions and a more detailed treatment of health. When people choose to consume fattening goods at restaurants rather than eating at home or choose to refrain from exercise, they increase their risk of obesity and disease but get direct utility from the consumption. This increased risk has two effects: mortality risk and increased healthcare costs that otherwise would have been used for consumption or savings.

The model examines elderly decisions after the age of 65 regarding consumption, restaurant dining, and exercise. [3] Based upon these decisions the individual faces obesity risk and health risks, including mortality. Health is made endogenous (with uncertainty) through choices of health inducing activities, mainly eating decisions and exercise. The model implements a robust treatment of health, as food consumption and exercise have different effects on obesity and diseases (see Katzel *et al*., 1995, and Dengel *et al*., 1996).

3.1 Utility from consumption, exercise, obesity, and restaurants

Individuals derive pleasure from consumption not related to restaurants or healthcare (c_t) and eating outside the home (e_t), and they face disutility from exercise or physical activity (v_t). They also derive disutility from having a higher weight (O_t), as measured by body mass index (BMI) at the beginning of every period.

$$U(c_t, e_t, v_t, O_t) = \frac{{c_t}^{1-\sigma}}{1-\sigma} + \varphi * O_t + \vartheta * e_t + \omega(v_t)$$

Here, σ represents the relative risk aversion of an individual. Utility is assumed to be additively separable in obesity status, restaurant consumption, and exercise. The parameter φ is the degree to which the individual dislikes being overweight. ϑ is the value to the individual of eating out in restaurants, while ω represents the disutility the individual derives from physical activity.

[3] See Section 2.1 for a discussion of the importance of reducing obesity even among the elderly. Computation available to the author at this time does not allow for estimation of a model with this level of detail on health and one that would incorporate the complications of retirement, employment and health insurance. As the focus of the paper is on obesity, a detailed treatment of diseases is important. Including just the choice to retire doubles the size of the problem.

When the person dies he or she gets an additional utility benefit which is a function of the level of assets he or she has at the time of death. [4] In order to match the data on the savings behavior of the elderly, this paper assumes a logarithmic function of assets at death where δ determines the relative utility value of post-mortem assets and a_t represents the amount of assets at time *t*.[5]

$$b(a_t) = \delta * \log(a_t)$$

3.2 Choice variables

The individual makes decisions every year $t = t_0, t_1, \ldots, T$. If the individual has survived to age *T*, he or she dies with certainty at that time. At every age the individual makes three choices, $z(t) = (c_t, e_t, v_t)$. These are consumption not related to restaurants or healthcare (c_t), whether or not to eat in restaurants or at home (e_t)[6], and how much exercise or physical activity to pursue (v_t).

3.3 Stochastic health process influenced by choices and state

In this paper, individuals face a risk of developing a number of different diseases: heart disease, hypertension, diabetes, cancer, stroke, and lung disease. Each disease has an effect on health care

[4] This is similar to a bequest motive, though this terminology is not quite accurate as we do not control for whether or not the assets after death are an inter or intra-generational transfer. As female are more likely to be single when they die and males more likely to be married, in the context of this model, it may be reasonable to have a sex specific motive, as the male is more likely to be pass wealth to his spouse, whereas a female is more likely to be bequeathing to children. Computational limits do not allow for a full analysis of how couples interact in these decisions.

[5] This is the same functional form as used in Fonseca *et al.* (2009).

[6] Eating in restaurants is treated as a discrete choice between eating at any point outside the home versus eating only at home. Continuous models of expenditures at restaurants on obesity indicated that the only statistically significant effect on BMI was from the discrete jump from no consumption outside the home to some consumption outside the home.

expenditures, mortality, and future disease risk (though no direct effect on utility). I model each disease separately as a function of current health, age, sex, BMI, and exercise. Thus, when calculating the likelihood of each disease in the next period, the probability of disease is as follows:

$$\Pr(H_{t+1}|H_t, v_t, O_t, \mu) = \Phi(X\alpha)$$

where H_t is a vector of all of the above diseases in period *t,* and μ is a vector of individual characteristics including age and sex. Thus, X includes the current set of diseases, exercise, obesity, and individual characteristics, where Φ is a standard normal distribution. α is a vector of estimated parameters. In this way increased obesity levels create an increased likelihood of acquiring diseases in the future, while physical activity reduces that risk, as has been noted in the literature. Lastly, out-of-pocket expenses are different for different diseases. All individuals in our sample are eligible for Medicare, and I do not model insurance choices, though a more complete model would model enrollment in subsections of Medicare and supplemental insurance plans.

3.4 Obesity transition specification

Prior to engaging in a physical activity or a diet, an individual does not have precise knowledge over the effects of such an endeavor, nor does the individual have complete knowledge of his or her future weight absent these activities. Thus, our model includes uncertainty over future obesity levels. The likelihood of being in a particular obesity level in the future depends on current levels of obesity, decisions about restaurant consumption and exercise, and individual

characteristics (including age). The only disease allowed to have an effect on obesity is cancer, as cancer and its treatments have a known effect on weight (Huhmann and Cunningham, 2005), while this is not the case for the other diseases modeled. A person's level of obesity is a categorization of a continuous body mass index, and thus we formulate the transition for obesity as an ordered probit.

3.5 Assets and consumption

At each time period, a person may forgo consumption and invest savings in an asset, a_t, which receives an annual rate of return of θ. Thus, at each time period, the cash-on-hand to consume or save (x_t) pension income (y_t) plus assets brought forward to this period (1+θ) a_t , minus out-of-pocket medical expenditures (oop_t), which are deterministic based on the health state.

Consumption in a period then consists of non-medical and non-restaurant consumption (c_t) and restaurant costs (p_t*e_t where p_t is the price of restaurant consumption relative to non-medical, non-restaurant consumption). Lastly, government transfers are imposed to ensure a minimum consumption floor. $Tr_t=\max(C_{min} - x_t, 0)$[7]. This consumption floor captures the principle of Medicaid and other social programs for the poor, wherein individuals with low assets receive government transfers to pay for their health care and other consumption. Individuals are not allowed to borrow against future revenues, and thus assets at the end of any period must be at least zero. This yields the following budget constraints:

$$x_t=(1+\theta)\, a_t + y_t - oop_t$$

[7] Note that government transfers may be used for restaurant consumption.

$$a_{t+1}=x_t+ Tr_t –C_t – p_t*e_t$$

$$a_t \geq 0$$

A person's public and private pension are aggregated and are assumed to be fixed throughout the individual's retirement. This does not account for survivor benefits changing an individual's pension income. In this way the results may over- or understate an individual's pension income when his or her spouse dies. Individuals are not allowed to enter the workforce and thus the only income available to an individual is from previous assets and pension income.

3.6 Lifetime utility maximization

The individual maximizes the discounted sum of utility from all future periods with a discount factor of β, which accounts for the individual's time preference. At each period, the individual chooses z_t from the set of feasible choices for a given state in the state space. The state space (s_t) includes assets, pension income, race and ethnicity, sex, disease status, and obesity status.[8] The problem is solved through a dynamic process as follows:

$$V_t(s_t) = Max_{z(t)}U(C_t, O_t, e_t, v_t) + I(Death) * b(a_t)$$

$$+\beta * E[\rho_{t+1}V_{t+1}(s_{t+1})|t, H_t, O_t, \kappa, v_t]$$

The above equation is subject to all constraints and formulations outlined in this section. The technique for solving V_t is described in Section 4.

4. Data and Estimation

[8] The state space defines the different possible states an individual might be in. The model must solve for the optimal decision at each element of the state space in each time period.

4.1 Data

The primary data for this paper are from the Health and Retirement Study. The Health and Retirement Study (HRS) provides a repeated sample of elderly households in the United States. As the HRS is panel data, these data are ideal both for estimation of the structural model and for estimating the parameters used in the model. The HRS includes information on health conditions, wealth, activity levels, pension income, and eating behaviors. I use the 2004, 2006, and 2008 waves of the HRS with individuals at least 65 years old. This paper models the behavior of nearly all people eligible for Medicare - that is, individuals at least 65 years old. This age range is chosen because it represents a large proportion of health care expenditures, and particular of government health care expenditures. Additionally, by using only elderly people, the modeling is simplified by not considering choices more common earlier in life (such as child birth). This population is still of interest for their health behaviors as there is still scope for individuals to reduce their obesity levels and diminish disease and mortality risk (see NIH, 2010). Only three waves are used, as these waves provide consistent definitions of the key exercise variables used in our model.[9]

Two key variables in this study are exercise and eating in restaurants. For exercise, the respondents are asked how often they take part in vigorous activities and are given options for more than once a week, once a week, one to three times a month, and hardly ever or never. I use

[9] For some estimates in the transition matrix, a larger population is used to avoid selection bias issues in the healthy elderly.

these four levels of activity directly.[10] For dining in restaurants the question is "About how much do you (and other family members living there) spend eating out in a typical week, not counting meals at work or at school?" While this provides a continuous variable on food consumption, I find little difference in estimation between the level of dining outside the home and whether or not one eats outside the home at all. Thus for simplicity I categorize people as having eaten outside the home or not.

While the HRS is ideal for most estimates there is concern that, because of self-reporting, it does not estimate medical expenditures for the elderly well (Hurd and Rohwedder 2011). Thus, for estimating medical costs conditional on diseases this paper uses the Medicare Current Beneficiary Survey (MCBS). The MCBS provides a nationally representative sample of elderly people which tracks the costs of medical expenditures and health states for Medicare beneficiaries over time. Given that our population is all at least 65 years old, this is an ideal data set for estimating medical expenditures.

4.2 Estimation of parameters

Several parameters are estimated outside of the dynamic program and those govern the dynamics of the model. The most important is the estimate of the effects of exercise and dining out on obesity. Rather than tracking continuous body mass index or highly disaggregated categories of obesity, I split body mass index into ten even categories ranging from 20 to 45. Using the HRS

[10] The question is "We would like to know the type and amount of physical activity involved in your daily life. How often do you take part in sports or activities that are vigorous, such as running or jogging, swimming, cycling, aerobics or gym workout, tennis, or digging with a spade or shovel: more than once a week, once a week, one to three times a month, or hardly ever or never?

data, I then estimate an ordered probit of the change in the probability of being in any given BMI category conditional on previous body mass index categories, individual characteristics, exercise level, and restaurant consumption. The effect of individual decisions on future obesity levels is directly estimated. This panel data approach is better than a cross-sectional analysis as it allows us to estimate the change associated with a particular behavior, though it does not deal with unobserved heterogeneity. Table 1 below provides the parameter estimates for the ordered probit with clustered standard errors.

I find that activity/exercise has a statistically significant negative effect on body mass index, while eating in restaurants has a statistically significant positive effect on body mass index. For the average individual, eating out at a restaurant increases the probability of being obese (BMI > 30) in the next period by 1.94%. I find a surprisingly large effect of exercise on obesity: the average person reduces his or her chance of being obese by 11% if he or she exercises at least twice a week.

I also calculate the probability of contracting each disease using a probit and the corresponding health state.[11] As an example, reducing one's body mass index from a range of 30 to 32.5 down to 25 to 27.5 has a statistically significant reduction in the prevalence of hypertension of 3.9% and diabetes of 2.5% (both statistically significant at the 99% level) in the next period. There is no statistically significant effect for this change on heart disease (though increasing diabetes and hypertension have statistically significant effects on future heart disease prevalence). Cost

[11] Complete parameter estimates are available upon request from the author.

estimates for diseases are done using a simple linear regression of out-of-pocket medical expenditures controlling for individual characteristics.[12]

[12] Complete parameter estimates are available upon request from the author.

Table 1: Ordered Probit of Next Period BMI - 16,932 observations

Characteristic	Coefficient	Standard Error	P-Value
BMI 20 to 22.5	-1.064	0.037	<0.001
BMI 22.5 to 25	-0.573	0.027	<0.001
BMI 25 to 27.5	-0.205	0.023	<0.001
BMI 27.5 to 30			Comparison Group
BMI 30 to 32.5	0.297	0.026	<0.001
BMI 32.5 to 35	0.535	0.033	<0.001
BMI 35 to 37.5	0.606	0.043	<0.001
BMI 37.5 to 40	0.855	0.055	<0.001
BMI 40 to 42.5	1.013	0.072	<0.001
BMI 42.5 to 50	1.321	0.079	<0.001
Eats Out	**0.055**	**0.023**	**0.016**
Age	0.019	0.004	<0.001
Less than High School	0.001	0.033	0.968
High School			Comparison Group
College	-0.090	0.024	<0.001
Male	0.104	0.021	<0.001
Wealth ($s)	0.000	0.000	<0.001
Hispanic	-0.027	0.032	0.41
African American	0.217	0.031	<0.001
Smoke Ever	0.053	0.022	0.003
Never Smoke			Comparison Group
Current Smoker	-0.486	0.029	<0.001
Cancer	0.010	0.036	0.784
Employed	-0.061	0.020	0.002
Married	0.024	0.025	0.339
High Activity	**-0.324**	0.022	<0.001
Moderate Activity	**-0.221**	0.028	<0.001
Low Activity	**-0.159**	0.029	<0.001
No Activity			Comparison Group
Cohort	-0.022	0.004	<0.001
Cut Point 1	-1.841	0.148	
Cut Point 2	-1.192	0.148	
Cut Point 3	-0.521	0.147	
Cut Point 4	-0.005	0.147	
Cut Point 5	0.462	0.147	
Cut Point 6	0.844	0.147	
Cut Point 7	1.176	0.147	
Cut Point 8	1.484	0.148	
Cut Point 9	1.775	0.148	

4.3 Implementation and estimation

4.3.1 Formulation of the state and decision spaces

The state space has three continuous variables: pension income, wealth, and BMI. I limit the state space by allowing for 20 categories of wealth, 15 of pension income, [13] and 10 categories of body mass index. The state space also includes 33 health states, 2 sexes, and 4 races and ethnicities. This creates a state space with 792,000 elements solved in each time period.[14] The model is solved and decision rules identified for each time period starting with the terminal time period (wherein death is a certainty) and proceeding backwards through time until the age of 65, t_0.[15]

The only continuous decision variable is consumption, which determines assets in the next period. Unfortunately, twenty categories of wealth are too sparse to accurately estimate the consumption behavior of individuals. Thus, for each category of wealth in the state space, the model searches across 30 points of feasible consumption levels and chooses the optimal one. It then identifies the implied level of assets for the next period. When decisions would result in a state not on the grid interpolation across the closest states is done to determine the value in the next period. This provides a more refined grid of potential assets for those of low wealth and a

[13] As retirement is not a decision in this model, this project does not require a particularly fine a grid on pension income.

[14] The model is implemented in FORTRAN and the parallel message parsing interface (MPICH) on a 48 processor core cluster. The model takes approximately 30 minutes to solve once.

[15] The estimation strategy for the structural model has many moving parts and requires simplifications in order to successfully estimate the parameters. Every increase in one dimension of the state space makes the problem substantially larger. For example, were I to model an additional variable like marital status, with four categories, this would make the problem four times larger.

less refined grid for states with higher wealth. The maximum wealth is set to $500,000, while minimum wealth is $0, as borrowing is not allowed in this model. A discount rate of 0.98 and an asset rate of return of 0.03 are used.

4.3.2 Estimation

Once decision rules have been computed individuals are simulated forward. Rather than using synthetic individuals, this paper uses actual individuals from the HRS and determines their initial location in the state space. When individuals choose levels of consumption off the grid of assets, they are randomly assigned to either the higher or lower asset category based on the relative distance from the asset categories closest to their ideal. In this way the model is able to maintain estimates of the wealth profile without needing a more refined asset grid.

The model is estimated using a Method of Simulated Moments Estimator (see Pakes and Pollard, 1989, and Newey and McFadden, 1994). Once the path of each individual is simulated, moments are computed and compared with empirical moments derived from the HRS sample. Parameter values for the utility function are chosen to minimize the sum of the square of the distance (in standard deviations) between the simulated moments and the actual ones. As the criteria function is not differentiable in many regions, a Nelder-Mead search algorithm is used (Nelder and Mead, 1965).

4.3.3 Moments

For identification, I choose and estimate a refined set of moments. This paper attempts to fit moments by sex and age. It calculates moments for the proportion of individuals exercising, eating out in restaurants, median and mean assets, average body mass index, and probability of death. It is important, especially when estimating the moments for body mass index, to account for cohort affects. Without accounting for these effects it appears as though individuals lose weight with age. However, when one accounts for these effects individuals tend to increase in BMI with time (not controlling for other factors).[16]

4.4 Results

Using the methods described in Section 4.3, I estimate the structural model. The parameter estimates are found in Table 2. The model estimates the appropriate signs for each variable; it estimates a parameter for risk aversion of 2.772, which is consistent with other estimates and calibrations (e.g. Fonseca et al, 2009). The estimated post-mortem asset motive is larger than has been typically found[17]. As anticipated, individuals prefer to have lower body mass index and enjoy eating out at restaurants, while increased levels of physical activity reduces utility.

[16] Section 5 contains both the empirical moments and moments estimated from the model.
[17] This is likely due to inclusion of intra- and intergenerational transfers.

Table 2: Parameter Estimates for the Structural Model[18]

Parameter	Value Estimate
Consumption (σ)	2.772
BMI (φ)	-0.061
Post-Mortem Assets (δ)	0.356
Eating Out (ω)	0.032
Low Activity (ϑ_2)	-0.041
Medium Activity (ϑ_1)	-0.054
High Activity (ϑ_3)	-0.105

Given the parameter for consumption, the estimates of the disutility of exercise, BMI, and the utility of restaurants are high (as the equivalent change in consumption for a change in these activities is quite large for average levels of baseline consumption). This indicates that any policy aimed at increasing exercise or reducing restaurant consumption among the elderly through taxes or cash incentives will likely be unsuccessful in increasing activity or decreasing food consumption outside the home. Thus, policies should focus on reducing the utility burden of exercise and changing the health effects of exercise and dining out.

5. Model Fit

Table 3 shows that the model predicts that males and females have generally increasing wealth over time. While in the actual data this is generally true for males, it is not the case for females. The difference here is likely due to not separating post-mortem asset motivations across inter- and intra-generational transfers. Intra-generational transfers are likely to induce more savings

[18] Standard Errors are yet to be calculated. Recall the utility function is: $U(c_t, e_t, v_t, O_t) = \frac{{c_t}^{1-\sigma}}{1-\sigma} + \varphi * O_t + \vartheta * e_t + \omega(v_t)$

and are more common for males than for females (as males are more likely to die before their spouse). I intend to separate these two effects in the future.

Table 3: Actual and Simulated Moments for the State Space

Age	Sex	Average Wealth (000s)		BMI		Mortality Rate	
		Actual	Simulated	Actual	Simulated	Actual	Simulated
66	Male	162	160	27.78	27.32	4.14%	1.13%
70	Male	163	179	27.83	28.23	3.66%	0.83%
75	Male	167	193	27.9	29.15	6.11%	2.96%
80	Male	171	190	27.97	30.93	12.00%	2.93%
66	Female	143	139	28.06	27.6	2.09%	0.44%
70	Female	140	146	28.11	27.15	2.31%	0.22%
75	Female	134	157	28.18	28.11	4.35%	0.46%
80	Female	94	163	28.25	28.75	5.13%	0.83%

Table 3 also shows that the model successfully estimates the increase in body mass index as people age and the increasing mortality rate for individuals over time. That said, the model systematically under-predicts mortality. This is likely because, as can be seen in Table 4, the model over-predicts high and moderate levels of activity (which have a substantial, direct effect on health).[19]

[19] The over-prediction of high activity levels is likely due to being in a saddle point, despite using a Nelder-Mead optimization routine which neither requires a concave objective function nor relies on derivatives. Because of the simplex nature of Nelder-Mead, the model has a difficulty realizing that both high and moderate activity parameters must be decreased simultaneously to improve the model.

Table 4: Actual and Simulated Moments for Choice Variables

Age	Sex	Eats Out		High Activity		Moderate Activity		Low Activity	
		Actual	Simulated	Actual	Simulated	Actual	Simulated	Actual	Simulated
66	Male	70.57%	71.08%	31.10%	73.86%	11.24%	26.14%	6.94%	0.00%
70	Male	70.29%	62.46%	28.57%	66.16%	7.56%	33.50%	7.35%	0.00%
75	Male	71.24%	61.22%	21.94%	46.50%	9.09%	49.91%	8.15%	0.00%
80	Male	67.35%	68.41%	20.39%	22.94%	4.85%	64.19%	1.94%	0.00%
66	Female	68.35%	74.15%	19.88%	77.55%	5.08%	22.45%	6.01%	0.00%
70	Female	61.29%	69.46%	18.89%	72.93%	4.64%	27.07%	5.42%	0.00%
75	Female	58.54%	57.34%	13.51%	75.11%	7.21%	24.77%	3.90%	0.00%
80	Female	50.00%	54.73%	11.11%	61.20%	0.00%	37.96%	11.11%	0.00%

For eating outside the home, the model predicts that between 60 and 70 percent of people will eat outside the home and that the proportion of people eating out decreases with age, with the only exception being for 80 year old men.[20] This matches the trend quite well for females while there remains some gap for males.

6. Policy Simulation

Numerous governments have implemented policies aimed at making the food that people eat outside the home healthier. The federal "Patient Protection and Affordable Care Act" (2010) includes a requirement that restaurants of a certain size provide calorie information on the dishes

[20] Moments are generally more precisely estimated for younger ages as there is a larger population for estimation.

that they serve to customers. Some states, such as California, have had similar policies for the last couple years. Many cities, including New York, Boston, Chicago, Philadelphia, and San Francisco have introduced restrictions on the use of particular oils which contain trans-fatty acids, which are high in calories and linked to obesity. Both of these policies are aimed at people eating fewer obesity inducing foods when outside the home. The model is uniquely able to assess the potential long-term impact of such policies on the health and welfare of the elderly. To simulate these policies I use the preference parameters estimated above, but I constrain the health effect of restaurants to be zero. That is, restaurants become as healthy as eating at home while keeping the quality and cost of the food constant.[21] This simulation allows for behavioral responses across exercise and changes in consumption habits. Table 5 provides the results of this simulation. Given the strong incentives estimated above, it is not surprising that when restaurants are as healthy as eating at home, all individuals choose to at some point consume outside of the home (recall that restaurant consumption is defined as ever eating outside the home).[22]

Table 5: Policy Simulation: Restaurants as Healthy as Home

		Eats Out		BMI		
Sex	Age	Status Quo	Healthy Restaurants	Status Quo	Healthy Restaurants	Percent Change
Male	66	71.08%	100.00%	27.32	27.15	-0.62%
Male	70	62.46%	100.00%	28.23	27.95	-0.99%
Male	75	61.22%	100.00%	29.15	28.64	-1.75%
Male	80	68.41%	100.00%	30.93	30.35	-1.88%
Female	66	74.15%	100.00%	27.60	27.43	-0.62%
Female	70	69.46%	100.00%	27.15	26.82	-1.22%
Female	75	57.34%	100.00%	28.11	27.76	-1.25%
Female	80	54.73%	100.00%	28.75	28.42	-1.15%

[21] It may be that calorie labeling in restaurants is more effective because information about calories may be more apparent to consumers than when eating at home. If this is the case, I will under-estimate the effects of such policies.

[22] It is NOT the case that all meals are eaten outside the home.

For males, this results in a reduction of the average BMI for the entire population of 1.88% by the age of 80. For females BMI goes down by 1.15%. Since the effect of restaurant food is cumulative, if one were to start simulation from earlier in a person's life, one would expect to see even larger reductions in the average BMI for the population. This reduction in BMI has corresponding improvements in health. Table 6 shows that this reduction is associated with a 6.44% reduction in hypertension among males living to age 80 and a smaller 0.5% reduction for females. Across the entire population there is a reduction of 3.21%.

Table 6: Effect of Healthy Restaurants on Hypertension

Sex	Age	Status Quo	Healthy Restaurants	Percent Change
Male	66	28.27%	28.27%	0.00%
Male	70	33.12%	32.96%	-0.48%
Male	75	34.41%	33.76%	-1.89%
Male	80	32.63%	30.53%	-6.44%
Female	66	32.82%	32.82%	0.00%
Female	70	34.57%	34.46%	-0.32%
Female	75	37.51%	36.75%	-2.03%
Female	80	35.06%	34.90%	-0.46%

In my second policy simulation we will look at the extent to which obesity can be reduced through financial incentives for seniors. I simulate such a policy by increasing out-of-pocket expenses for seniors by 20%. Seniors do not pay for all of their medical expenditures, and thus this policy would have the added benefit of removing a negative externality of excess food consumption. Such a policy may be attractive to policy makers interested in stemming the

increasing costs of Medicare and rising US Government debts[23]. As the cost of treating diseases increases, the negative health effects of restaurants become more costly (in dollars) and we thus expect seniors to reduce their use of restaurants. Table 7 shows that there is a general trend that individuals will reduce their dining out behavior by 1.47% for males and 0.12% for females at age 80 when faced with increased responsibility for medical costs.

In the same simulation I look to see if there is an effect of increasing medical expenditures on exercise. As exercise reduces your likelihood of disease, individuals now have a greater incentive to increase their levels of activity. Table 7 suggests that females may be switching from moderate levels of activity to high levels. For females at age 75 high levels of activity have gone up by 0.16%, while moderate activity has decreased by 0.46%. Males see little change until age 80. I have yet to calculate the confidence intervals for the estimate, and these results may not prove to be statistically significant. For the parameters estimated, it is not surprising that the effect of increases in medical costs is small. As there has been a substantial increase in the cost of health care while obesity has been rising, finding a large response to health care costs would not fit the macro trend.

[23] Similarly the Defense Departments 2013 proposed budget increases the share of medical expenditures military retirees must pay.

Table 7: Effect of an Increase of Treatment Costs of 20% on Behavior

		Eats Out			High Activity			Moderate Activity		
Sex	Age	Status Quo	High Health Costs	Percent Change	Status Quo	High Health Costs	Percent Change	Status Quo	High Health Costs	Percent Change
Male	66	71.08%	71.24%	0.23%	73.86%	74.02%	0.22%	26.14%	25.98%	-0.61%
Male	70	62.46%	61.95%	-0.81%	66.16%	66.16%	0.00%	33.50%	33.50%	0.01%
Male	75	61.22%	60.50%	-1.17%	46.50%	46.50%	0.00%	49.91%	50.09%	0.36%
Male	80	68.41%	67.40%	-1.47%	22.94%	22.54%	-1.76%	64.19%	64.59%	0.62%
Female	66	74.15%	74.15%	0.00%	77.55%	77.44%	-0.15%	22.45%	22.56%	0.50%
Female	70	69.46%	69.35%	-0.16%	72.93%	72.82%	-0.15%	27.07%	27.18%	0.41%
Female	75	57.34%	57.22%	-0.20%	75.11%	75.23%	0.16%	24.77%	24.66%	-0.46%
Female	80	54.73%	54.67%	-0.12%	61.20%	61.36%	0.27%	37.96%	37.80%	-0.42%

7. Conclusions

This research has formulated and estimated a complex model of health, obesity, restaurants, and exercise for the elderly population. It treats obesity as the result of individual choices, and allows for an optimal level of obesity for each individual. The model estimates provide useful parameters for welfare analysis of policies aimed at reducing obesity. I policy simulations to find that were restaurants able to improve the healthiness of their food while maintaining the same quality and cost, there would be a roughly 2% reduction in the average BMI of elderly Americans. While the model does not fit the data perfectly, it can generally predict the behavior of elderly Americans, especially in their BMI, savings, and eating habits. It finds strong incentives for individuals to eat out and substantial penalties for gaining weight. It implies that over time policies targeting the quality of food at restaurants may have a substantial impact on

the health of Americans, while those policies which increase the cost of health care will likely have little effect on the behavior and thus obesity of individuals.

Bibliography

Adda, J., and V. Lechene. 2004. On the identification of the effect of smoking on mortality.

Anderson, M., and D.A. Matsa. 2009. Are Restaurants Really Supersizing America? *Available at the SSRN eLibrary Paper*.

Bouchard, C., JP Depres, and A. Tremblay. 1993. Exercise and obesity. *Obesity Research* 1 (2):133.

Calle, E.E., M.J. Thun, J.M. Petrelli, C. Rodriguez, and C.W. Heath Jr. 1999. Body-mass index and mortality in a prospective cohort of US adults. *New England Journal of Medicine* 341 (15):1097-1105.

Chou, S.Y., M. Grossman, and H. Saffer. 2002. An economic analysis of adult obesity: results from the Behavioral Risk Factor Surveillance System. National Bureau of Economic Research Cambridge, Mass., USA.

Christakis, N.A., and J.H. Fowler. 2007. The spread of obesity in a large social network over 32 years. *New England Journal of Medicine* 357 (4):370-379.

Currie, J., S. DellaVigna, E. Moretti, and V. Pathania. 2010. The effect of fast food restaurants on obesity and weight gain. *American Economic Journal: Economic Policy* 2 (3):32-63.

Davis, B., and C. Carpenter. 2009. Proximity of fast-food restaurants to schools and adolescent obesity. *American Journal of Public Health* 99 (3):505.

De Nardi, M., E. French, and J. Jones. 2010. Why do the elderly save? the role of medical expenses. *Journal of Political Economy* 118 (1):39-75.

Dengel, D.R., R.E. Pratley, J.M. Hagberg, E.M. Rogus, and A.P. Goldberg. 1996. Distinct effects of aerobic exercise training and weight loss on glucose homeostasis in obese sedentary men. *Journal of Applied Physiology* 81 (1):318.

Duchovny, Noelia, and Colin Baker. 2010. How Does Obesity in Adults Affect Spending on Health Care? In *Economic and Budget Issue Brief*: Congressional Budget Office.

Elia, M. 2001. Obesity in the elderly. *Obesity* 9:244S-248S.

Field, A.E., E.H. Coakley, A. Must, J.L. Spadano, N. Laird, W.H. Dietz, E. Rimm, and G.A. Colditz. 2001. Impact of overweight on the risk of developing common chronic diseases during a 10-year period. *Archives of Internal Medicine* 161 (13):1581.

Flegal, K.M., M.D. Carroll, R.J. Kuczmarski, and C.L. Johnson. 1998. Overweight and obesity in the United States: prevalence and trends, 1960-1994. *International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity* 22 (1):39.

Fonseca, R., P.C. Michaud, T. Galama, and A. Kapteyn. 2009. On the rise of health spending and longevity. RAND Working Paper.

Fontana, L., and S. Klein. 2007. Aging, adiposity, and calorie restriction. *JAMA: the journal of the American Medical Association* 297 (9):986.

Fontana, L., D.T. Villareal, E.P. Weiss, S.B. Racette, K. Steger-May, S. Klein, and J.O. Holloszy. 2007. Calorie restriction or exercise: effects on coronary heart disease risk factors. A randomized, controlled trial. *American Journal of Physiology-Endocrinology And Metabolism* 293 (1):E197.

Goldman, D. P., P. G. Shekelle, J. Bhattacharya, M. D. Hurd, G. F. Joyce, D. N. Lakdawalla, D. Matsui, S. Newberry, C. Panis, and B. Shang. 2004. *Health status and medical treatment of the future elderly: Final Report*. Vol. 169: Rand Corporation.

Goldman, D.P., P.C. Michaud, D. Lakdawalla, Y. Zheng, A. Gailey, and I. Vaynman. 2010. The Fiscal Consequences of Trends in Population Health. *National Tax Journal* 63 (2):307-330.

Gourinchas, P.O., and J.A. Parker. 2002. Consumption over the life cycle. *Econometrica* 70 (1):47-89.

Guthrie, J.F., B.H. Lin, and E. Frazao. 2002. Role of food prepared away from home in the American diet, 1977-78 versus 1994-96: changes and consequences. *Journal of Nutrition Education and Behavior* 34 (3):140-150.

Heckman, J.J., and T.E. MaCurdy. 1980. A life cycle model of female labour supply. *The Review of Economic Studies* 47 (1):47-74.

Hopkins, M., N.A. King, and J.E. Blundell. 2010. Acute and long-term effects of exercise on appetite control: is there any benefit for weight control? *Current Opinion in Clinical Nutrition & Metabolic Care* 13 (6):635.

Huhmann, M.B., and R.S. Cunningham. 2005. Importance of nutritional screening in treatment of cancer-related weight loss. *The lancet oncology* 6 (5):334-343.

Hurd, M.D. 1989. Mortality risk and bequests. *Econometrica: Journal of the Econometric Society*:779-813.

Hurd, M.D., and S. Rohwedder. 2011. The Effect of the Risk of Out-of-Pocket Spending for Health Care on Economic Preparation for Retirement. University of Chicago Press.

Jakicic, J.M. 2003. Exercise in the treatment of obesity. *Endocrinology and metabolism clinics of North America* 32 (4):967.

Joyce, G.F., E.B. Keeler, B. Shang, and D.P. Goldman. 2005. The lifetime burden of chronic disease among the elderly. *Health Affairs*.

Kant, A.K., and B.I. Graubard. 2004. Eating out in America, 1987-2000: trends and nutritional correlates. *Preventive Medicine* 38 (2):243-249.

Katzel, L.I., E.R. Bleecker, E.G. Colman, E.M. Rogus, J.D. Sorkin, and A.P. Goldberg. 1995. Effects of weight loss vs aerobic exercise training on risk factors for coronary disease in healthy, obese, middle-aged and older men. *JAMA: the journal of the American Medical Association* 274 (24):1915.

Keeler, E.B., W.G. Manning, J.P. Newhouse, E.M. Sloss, and J. Wasserman. 1989. The external costs of a sedentary life-style. *American Journal of Public Health* 79 (8):975.

Khwaja, A. 2010. Estimating willingness to pay for medicare using a dynamic life-cycle model of demand for health insurance. *Journal of Econometrics* 156 (1):130-147.

Lakdawalla, D., and T. Philipson. 2009. The growth of obesity and technological change. *Economics & Human Biology* 7 (3):283-293.

McCrory, M.A., P.J. Fuss, N.P. Hays, A.G. Vinken, A.S. Greenberg, and S.B. Roberts. 1999. Overeating in America: association between restaurant food consumption and body fatness in healthy adult men and women ages 19 to 80. *Obesity Research* 7 (6):564.

McLaren, L. 2007. Socioeconomic status and obesity. *Epidemiologic reviews* 29 (1):29.

Michaud, PC, D. Goldman, D. Lakdawalla, A. Gailey, and Y. Zheng. 2011. Differences in health between Americans and Western Europeans: Effects on longevity and public finance. *Social science & medicine*.

Must, A., J. Spadano, E.H. Coakley, A.E. Field, G. Colditz, and W.H. Dietz. 1999. The disease burden associated with overweight and obesity. *JAMA: the journal of the American Medical Association* 282 (16):1523.
Nelder, J.A., and R. Mead. 1965. A simplex method for function minimization. *The computer journal* 7 (4):308.
Newey, W.K., and D. McFadden. 1994. Large sample estimation and hypothesis testing. *Handbook of econometrics* 4:2111-2245.
Nielsen, S.J., and B.M. Popkin. 2003. Patterns and trends in food portion sizes, 1977-1998. *JAMA: the journal of the American Medical Association* 289 (4):450.
NIH. 1998. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. In *The Evidence Report*: National Institutes of Health.
———. 2010. The Practical Guide: Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. National Institutes of Health.
———. 2011. Strategic Plan for NIH Obesity Research. NIH Obesity Research Task Force.
Ogden, C.L., M.D. Carroll, L.R. Curtin, M.A. McDowell, C.J. Tabak, and K.M. Flegal. 2006. Prevalence of overweight and obesity in the United States, 1999-2004. *JAMA: the journal of the American Medical Association* 295 (13):1549.
Pakes, A., and D. Pollard. 1989. Simulation and the asymptotics of optimization estimators. *Econometrica: Journal of the Econometric Society*:1027-1057.
Palumbo, M.G. 1999. Uncertain medical expenses and precautionary saving near the end of the life cycle. *The Review of Economic Studies* 66 (2):395.
Rashad, I. 2006. Structural estimation of caloric intake, exercise, smoking, and obesity. *The Quarterly Review of Economics and Finance* 46 (2):268-283.
Redman, L.M., L.K. Heilbronn, C.K. Martin, A. Alfonso, S.R. Smith, and E. Ravussin. 2007. Effect of calorie restriction with or without exercise on body composition and fat distribution. *Journal of Clinical Endocrinology & Metabolism* 92 (3):865.
Sjostrom, C.D., M. Peltonen, H. Wedel, and L. Sjostrom. 2000. Differentiated long-term effects of intentional weight loss on diabetes and hypertension. *Hypertension* 36 (1):20.
Slentz, C.A., B.D. Duscha, J.L. Johnson, K. Ketchum, L.B. Aiken, G.P. Samsa, J.A. Houmard, C.W. Bales, and W.E. Kraus. 2004. Effects of the amount of exercise on body weight, body composition, and measures of central obesity: STRRIDE--a randomized controlled study. *Archives of Internal Medicine* 164 (1):31.
Sturm, R. 2002. The effects of obesity, smoking, and drinking on medical problems and costs. *Health Affairs* 21 (2):245.
Sobal, J., and A.J. Stunkard. 1989. Socioeconomic status and obesity: a review of the literature. *Psychological bulletin* 105 (2):260.
Tuomilehto, J., J. Lindström, J.G. Eriksson, T.T. Valle, H. Hämäläinen, P. Ilanne-Parikka, S. Keinänen-Kiukaanniemi, M. Laakso, A. Louheranta, and M. Rastas. 2001. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *New England Journal of Medicine* 344 (18):1343-1350.

Paper 2: Differences in Health between Americans and Western Europeans: Effects on Longevity and Public Finance[24]

As previously seen in: Michaud, PC, D. Goldman, D. Lakdawalla, A. Gailey, and Y. Zheng. 2011. Differences in health between Americans and Western Europeans: Effects on longevity and public finance. *Social science & medicine*.

Abstract

In 1975, 50 year-old Americans could expect to live slightly longer than most of their Western European counterparts. By 2005, American life expectancy had fallen behind that of most Western European countries. We find that this growing longevity gap is primarily due to real declines in the health of near-elderly Americans, relative to their Western European peers. We use a microsimulation approach to project what US longevity would look like, if US health trends approximated those in Western Europe. The model implies that differences in health can explain most of the growing gap in remaining life expectancy. In addition, we quantify the public finance consequences of this deterioration in health. The model predicts that gradually moving American cohorts to the health status enjoyed by Western Europeans could save up to $1.1 trillion in discounted total health expenditures from 2004 to 2050.

[24] We are grateful to the U.S. Department of Labor, the National Institute on Aging (RC4G039036 and 7P30AG024968) and the MacArthur Research Network on an Aging Society for financial support. We also thank Michael Hurd, James Smith, Maggie Weden, Samuel Preston, James Banks and Arthur van Soest as well as seminar participants at Tilburg University and the Michigan RRC.

Introduction

The populations of the United States and Western Europe have experienced large gains in life-expectancy over the last century. U.S. life expectancy at birth increased from 61 years in 1933 to 78 years in 2004. In many other developed countries, age-specific death rates have declined exponentially over this period (Tuljapurkar et al., 2000). During the first half of the 20th century, it was large declines in infectious diseases that drove down these mortality rates, particularly for the young. But in the second half of the 20th century, it was reductions in mortality among the elderly, rather than the young, that propelled increases in life expectancy (Olshansky and Carnes, 2001).

During the first half of the 20th century, when infectious diseases were on the decline, life expectancy across developed countries converged (White, 2002). The second half, however, witnessed divergence, as the US began to fall behind other developed countries in terms of life expectancy (Oeppen and Vaupel, 2002). So far, little is known about the causes and consequences of this widening gap (Lee, 2003).

The U.S. allocates the highest share of national income to health expenditures, yet does not lead the world in life expectancy. This has been used by some to suggest the inefficiency of the U.S. health care system. However, a recent study by Preston and Ho (2009) questions this conclusion by demonstrating that the U.S. ranks high in terms of life expectancy for people already diagnosed with chronic or terminal illness. They conclude that the health care system, at least in terms of curative treatment, is unlikely to be responsible for the deterioration in life expectancy. Instead, these findings point toward poor health behaviors and prevention strategies in the US population.

Indeed, many studies have shown that the health of middle-aged Americans, and health behaviors such as smoking and obesity, are much worse than those of Western Europeans (Banks et al., 2006; Andreyeva et al., 2007; Thorpe et al., 2007). This raises the question as to whether health behaviors have contributed to the divergence in life expectancy, and the question of where in the life-cycle the deterioration in U.S. life expectancy originates. Understanding both the fact and the source of deteriorations in health is a prerequisite for intervening against such trends.

In this paper, we argue that the worsening health of middle-aged Americans relative to their Western European counterparts is responsible for this disparity. Furthermore, we quantify the fiscal consequences of this gap. We use a dynamic microsimulation model calibrated to match historical U.S. health and longevity dynamics over the life course. We use the model to simulate the total longevity, disability, and financial costs to the US population of its poorer health status. We also quantify the gains that could be realized over time by gradually transitioning US cohorts to the health levels enjoyed by their Western European counterparts.

For the balance of the paper, the term "European" refers to the population of a sub-group of Western European countries (Denmark, France, Germany, Greece, Italy, The Netherlands, Spain and Sweden). This group of countries is quite representative of heterogeneity in health and socio-economic conditions within Western Europe, and growth in life expectancy has been higher than in the U.S. both for this group of countries and for an enlarged group of Western European countries (EU-15).

The paper is structured as follows. We first describe the data on mortality and health in the U.S. and Europe. Then, we describe the model that is used to describe the long-term economic consequences of these trends. Next, we use the model to quantify the effect of differences in health on longevity and government expenditures/revenues, and finally we discuss the results.

Mortality, Health Behaviors, and Health in the US and Western Europe

Cross-Country Differences in Mortality

In 1975, 50 year-old Americans could expect to live 0.6 years longer than their counterparts residing in a group of 15 Western European countries. 50 year-old Americans lived on average 27.3 years, compared to 26.6 years for the 15 countries originally forming the European Union (Austria, Belgium, Denmark, Finland, France, Ireland, Italy, The Netherlands, Norway, Portugal, Spain and Sweden, United Kingdom and West Germany). These estimates were obtained from the Human Mortality Database project (www.mortality.org). Over the ensuing decades, however, Western European life expectancy grew more quickly. As Figure 1 shows, a 50 year-old American in 2005 could expect to live for 31 years, compared to 32 years in Europe (32.8 in France and Italy). From 1975 to 2005, life expectancy grew by 5.37 years in Europe compared to just 3.75 years in the U.S. Only Denmark experienced a lower growth in life expectancy over this period (2.9 years).

The 1.6 year life expectancy gap between the U.S. and the EU-15 countries implies a non-trivial welfare loss. For example, using $100,000 as a lower-bound estimate of the value of a statistical life year (Viscusi and Aldy, 2003), this would represent at least a $700 million dollar disadvantage for the current generation of 50 year-olds. While these differences are not as large as within-country differences in health (across race for example), it is worth noting that these cross-country differences have emerged in spite of similar levels of economic development across countries.

Cross-Country Differences in Health Behaviors

While US life expectancy was deteriorating in relative terms, chronic illnesses associated with more sedentary lifestyles were spreading (Goldman et al., 2005; Lakdawalla et al., 2005). Due to data limitations, it is hard to assess whether trends in chronic disease have spread *more rapidly* in the US, but historical data do exist on obesity and smoking, two important health behaviors that contribute to chronic disease.

Both the levels of obesity and growth in obesity are higher in the U.S. than in Europe (based on OECD Health Data, at http://www.ecosante.fr). In 1975, 15% of Americans were obese, while obesity rates in European countries such as France, the Netherlands and Spain were less than 8% as recently as the 1980s. By 2005, the obesity rate in the U.S. was well over 30%, while the European average remains close to 12%.

Reductions in the costs of food consumption and technological innovations that led to more sedentary work are two key explanations for the U.S. trend (Lakdawalla and Philipson, 2009). Cutler et al. (2003) argue that these changes may have taken place more slowly in Europe due in particular to stricter food regulation. Obesity elevates the risk of various health conditions such as hypertension, diabetes and heart disease (Colditz, 1995; Willett, 1995). In that sense, it has the potential to explain part of the difference in life expectancy emerging over time.

Tobacco consumption trends are somewhat harder to interpret definitively. On the one hand, tobacco consumption has fallen by more in the U.S. than in Europe. Today, based on data from OECD Health Database (http://www.ecosante.fr), tobacco consumption is higher in Europe (1750 grams per capita vs. 1315 for the U.S.), but in 1975, it was much higher in the US (3506 grams per capita vs. 2540 grams in Europe). This means that the near-elderly Americans are less likely to be smoking now than their European counterparts. On the other hand, these American cohorts are much more likely to have *ever smoked*, which may have independent effects on

health. One plausible explanation for this rapid decrease is that smoking cessation programs have been more effective in the U.S. than in Europe. For example, Cutler and Glaeser (2006) argue that 50% of the gap in current smoking status is due to differences in beliefs about the health effects of smoking.

The health consequences of smoking depend in part on the length of exposure to tobacco, or lifetime consumption, rather than consumption at a point in time. For example, Rogers and Powell-Griner (1991) estimated that for males (females), compared to current smokers, former smokers could expect to live 3.7 (5.2) additional years and those who never smoked an additional 2.4 (1) years. Based on the OECD data, it is not clear whether lifetime exposure to tobacco – in terms of cigarettes smoked -- is greater among Americans or Europeans. In addition, the consequences of smoking for life expectancy will also depend on changes over time in the age composition of smokers; this may vary across countries. In sum, it is unclear whether trends in tobacco use have contributed to worsening or improving health for Americans, compared to Europeans.

Cross-Country Differences in Health

Figure 2 displays the possible health consequences of these divergent trends in health behavior: The US prevalence of different types of chronic disease and risky behavior is much higher than in a selected but representative group of European countries (Banks et al., 2006; Andreyeva et al., 2007). The figure displays these data among the 50-55 year old population using internationally comparable survey micro-data in Denmark, France, Germany, Greece, Italy, the Netherlands, Spain and Sweden. We used the 2004 waves of the Health and Retirement Study in the U.S. and the Survey of Health Ageing and Retirement in Europe (http://www.share-project.org/) to produce these numbers. Properly weighted, both surveys are representative of the

age 50+ population in each country. Questions on health conditions are very similar. The text of those questions is reproduced in Table A.1 of the online appendix. Data from Switzerland are not used because of small sample sizes and low response rates (lower than 50%) and data from Austria are not used for lack of sampling weights. Except for the behavioral measure of current smoking status, Americans look worse along all dimensions of health. Americans are about twice as likely to have hypertension, twice as likely to be obese, and twice as likely to have diabetes. As we demonstrate later, these differences are unlikely to be explained by differences in diagnosis or reporting. For example, the prevalence of stroke—a condition that rarely goes undiagnosed—is twice as low in Europe as in the U.S. On the other hand, the prevalence of cancer may be higher in the U.S. because of higher screening rates (Howard et al., 2009).

Clearly, the apparent gaps in observed health status will contribute to a gap in longevity, but it is not obvious how big the contribution will be. For instance, there are well-documented longevity gaps across racial and socioeconomic lines within countries. These are not fully explained by differences in observed health status. The question is whether "being American" is an independent mortality risk factor, in the same way that being poor or being black increase risk above and beyond observed health.

Microsimulation Model of Health and Economic Dynamics

Background

Compared to Europe, the US has enjoyed smaller longevity increases, and worsening health along many, but not all dimensions. Understanding the fiscal consequences of these trends requires that we account for the varied nature and magnitudes of health and mortality trends. To do this, we construct a transition model that relates current health to the future risk of mortality.

Given our interest in fiscal consequences, we also need a model linking health and economic outcomes. Both the epidemiological and economic literatures contain complex models of each, but few integrate both.

The current social science literature features several well-known and complementary approaches for measuring population health and projecting future disease burden and mortality—including models by Manton and co-authors (Manton, Singer, and Suzman, 1993), Lee (Lee, 2000), and Hayward (Hayward and Warner, 2005). Across these models, there is an underlying trade-off between the complexity of the data required, and the broad applicability of the model.

We use a model that considers the dynamic interplay among a large number of individual outcomes, including health status, and economic behavior. The model is an extension of the Future Elderly Model (FEM) (Goldman et al., 2004). The FEM consists of a transition model across health states that allows for unobserved heterogeneity (frailty) and dynamic population simulations. In that sense, it is well-equipped to analyze the effect of health differences on longevity and public financial liabilities, as it allows for complex interactions between multi-dimensional measures of health and economic outcomes.

Functioning of the Dynamic Model

Overview

The Future Elderly Model (FEM) was developed to examine health and health care costs among the elderly Medicare population (age 65+) (Goldman et al., 2004). The most recent version now projects these outcomes for all Americans aged 50+ using data from the Health and Retirement Study. The defining characteristic of the model is its use of real individuals, rather than synthetic

cohorts. This allows for more heterogeneity in behavior than would be allowed by a cell-based approach. The model has three core components:

The initial cohort module predicts the health and socio-economic outcomes of new cohorts of 50 year-olds. This module calibrates the Health and Retirement Study (HRS) to reflect population trends observed in younger populations from the National Health Interview Study (NHIS). It allows us to generate new cohorts as the simulation proceeds, so that we can measure outcomes for the age 50+ population in any given year.

The transition module calculates the probabilities of entering and exiting various health states, and the likelihood of various financial outcomes. The module takes as inputs risk factors such as smoking, weight, age, and education, along with lagged health and financial states. This allows for a great deal of heterogeneity and fairly general feedback effects. The transition probabilities are estimated from the longitudinal data in the Health and Retirement Study (HRS). These probabilities are then used to simulate the path of individuals in the simulation.

The policy outcomes module aggregates projections about individual-level outcomes into policy outcomes such as taxes, medical care costs, pension benefits paid, and disability benefits. This component takes account of public and private program rules to the extent allowed by the available outcomes. Because we have access to HRS-linked restricted data from Social Security records and employer pension plans, we are able to realistically model receipt of retirement benefits.

Figure 3 provides a schematic overview of the model. We start in 2004 with an initial population aged 50+ taken from the HRS. We then predict outcomes using our estimated transition probabilities. Those who survive make it to the end of that year, at which point we calculate policy outcomes for the year. We then move to the following year, when a new cohort

of 50 year-olds enters (with a different health profile). These entrants, along with the survivors from the last period, constitute the new age 50+ population, which then proceeds through the transition model as before. This process is repeated until we reach the final year of the simulation. In what follows, we give an overview of each component of the model. The online technical appendix accompanying this paper contains more details on the implementation.

Initial Cohort Module

We need to characterize outcomes for the age 50+ population. Hence, we need to predict the characteristics of the current and future 50 year-old population, in terms of health, demographics, and economic outcomes. Unfortunately, the HRS does not include respondents younger than age 50; therefore, the characteristics of tomorrow's 50 year-olds must be modeled using data on people who are younger than age 50 today.

We estimate trends in the health of 50 year-olds using two methods. First, we use the method described in Goldman, Hurd et al. (2004) to calculate trends in disease prevalence from the National Health Interview Surveys (NHIS). This method adopts a synthetic cohort approach and uses historical age-year prevalence estimates to "age-forward" prevalence rates, taking account of cures and mortality. The trends we estimate from this procedure are relatively close to other independent estimates, as documented in the online appendix. For outcomes other than disease prevalence, we use existing estimates, all of which are documented in the appendix.

Two important trend assumptions are made in the status-quo for obesity and smoking. The trend for obesity comes from Ruhm (2007), who predicts prevalence in different classes of obesity up to 2030. In particular, using his predictions, obesity prevalence in 2050 would near 50%. Although this is likely a "doomsday" prediction, it reveals a worst-case scenario which

serves as a useful benchmark. Similarly, Levy (2006) uses a simulation model to predict smoking prevalence. We extended those projections up to 2050. The prevalence of current smokers is predicted to decrease among age 50-54 individuals from 25% to less than 10% by 2050.

Second, we use the 50 year-old HRS respondents from 2004 as a template for future cohorts of 50 year-olds. Due to sample size consideration, we consider individuals aged 50-53 as our initial cohort of "50 year-olds." We adjust their health to match the levels of health predicted for future 50 year-olds, according to the methods discussed earlier. For example, if obesity is projected to rise in 2020, we increase the rate of obesity within the cohort of 50 year-olds, by reassigning enough non-obese individuals to obesity status. Since obesity is correlated with other outcomes such as hypertension and diabetes, we reassign obesity status so that those at greatest risk are more likely to be designated as being obese.

The reassignment is governed by a latent health model with correlated unobservables. An individual's disease status is a function of the mean population probability of the disease, along with a random error term. For an individual, the error terms are correlated across diseases. This builds on the possibility that, for instance, the occurrence of diabetes and hypertension are correlated. The bottom panel of Table 1 lists all the outcomes that we consider in this latent health model. There are seven binary outcomes: hypertension, heart disease, diabetes, fair or poor self-reported health, labor force participation, insurance status and positive wealth. There are three ordered outcomes: BMI status, smoking status and functional status in the transition model. Finally, there are five continuous outcomes measuring pension eligibility and savings.

Each of these outcomes depends on fixed characteristics such as race, education, gender and marital status. We also consider cancer, lung disease and stroke as fixed covariates, because

their prevalence is very low in this population (age 50-53). Estimates are presented in the online appendix.

Finally, the size of the entering cohort is adjusted to reflect population projections from Census by gender and race. We also adjust the size of the initial new cohort in 2004 to Census estimates by gender, race, and ethnicity.

Transition Model

The transition model tracks movement among states as a function of risk and demographic factors. The online technical appendix provides details on the parametric structure, estimation, and validation of the model. These consist of first-order Markovian limited-dependent variable models (probits, ordered probits, multinomial logits, censored regressions, etc). We enumerate and discuss all the key inputs and outputs of the model, and how they are measured.

The data come from the 1992 to 2004 biennial waves of the HRS. We consider both health and economic outcomes, all of which are listed in the top panel of Table 1. The table lists several groups of variables: diseases, risk factors, functional status, labor force and benefit status, financial resources, nursing home residence, and death. At a particular point in an individual's life, the model takes as inputs risk factors, along with the individual's lagged disease status, functional status, labor force and benefit status, financial resources, and nursing home status. The outputs are current disease status, functional status, labor force and benefit status, financial resources, and nursing home status. More detail on variable measurement is presented below.

Transition rates are allowed to differ across demographic and economic groups. In particular, we allow differences by gender, race and ethnicity, education, and marital status. Transition equations are estimated using 7 waves of HRS data. We assess the fit of the model by simulating 2004 outcomes for the 1992 HRS respondents; these are then compared with actual outcomes.

We use half the sample for estimation and the other half for simulation. In general, the model fits the data quite well, with a close correspondence between predicted and actual outcomes in most areas. Complete results can be found in Table A.9 of the online technical appendix.

Model Restrictions

We make several restrictions on the transition risks permitted in the model. First, we only allow feedback from diseases where clinical research supports such a link, based on consultation with several physicians from the Southern California Evidence-Based Practice Center. For example, we allow hypertensive patients to have higher risk of heart disease, but we do not allow hypertensive patients to have higher risk of cancer. These clinical restrictions are documented in the online technical appendix and elsewhere (Goldman et al., 2004) and generally do not affect the results of the simulations done here (many of the restrictions are in fact valid using statistical tests).

Another important restriction we impose is that economic outcomes do not feed back into health status. Although controversial across disciplines, this is consistent with the findings from recent studies looking at the elderly population (Adams et al., 2003). SES innovations (shocks) do not appear to have a causal effect on health outcomes *in this age range*. The correlation between SES and health appears to be generated by feedback effects from health to economic status, most notably through the effect of health shocks on labor supply and medical spending. Also playing a role are predetermined (earlier) events or common factors (genetics, etc) that induce a non-causal correlation between SES and health. Both these factors are accounted for in the estimation.

This assumption of no feedback from SES to health does not qualitatively affect our thought-experiment. Because we keep economic outcomes constant at U.S. levels when changing the health of Americans at baseline, failure of this assumption would lead us to underestimate the effects of improving US health. In earlier estimation stages of the transition equations, we found that very few of the economic variables were statistically significant. Hence, this assumption tends toward parsimony and tractability.

Policy Outcomes

The model simulates a number of relevant health and economic outcomes for individuals. First, we consider a set of health outcomes such as life expectancy, healthy life expectancy (no ADL limitations), and medical expenditures. Average medical expenditures by disease and demographic group are calculated from two sources. For those younger than age 65, we use the Medical Expenditure Panel Survey (MEPS) and include in medical expenditures the respondents' medical care costs and the cost of drugs. For those above age 65, we use the Medicare Current Beneficiary Survey (MCBS). Some adjustments are made such that aggregate expenditures match National Health Accounts estimates (see online appendix).

In addition to the individual outcomes, the model predicts tax revenues and medical expenditures by the Federal Government for the age 50+ population. As part of the predicted medical expenditures, we also predict expenditures by source, including those by Medicare and Medicaid. Next, we compute Social Security retirement benefits for those predicted to receive such benefits. We account for spouse and survivor benefits. We also compute disability insurance (DI) benefits and Supplemental Security Income (SSI). As Goldman et al. (2010) report, the simulation is able to replicate basic fiscal aggregates for 2004. Where deviations exist, we adjust our predictions from the model so that we match national aggregates in 2004.

Simulation Methods

For population scenarios, the simulation starts with the existing age 50+ population in the 2004 wave of the HRS. The microsimulation is stochastic, meaning that transitions are randomly drawn from the joint distribution of state variables, which is estimated from the HRS. This process is repeated a number of times to ensure independence from any particular sequence of random numbers. We average more than 100 replications.

These simulations are bound to be imprecise due to two sources of uncertainty. First, there is simulation noise due to the use of pseudo-random draws in drawing state-variables. However, since we average over many simulated individuals and replications, this type of noise tends to be minimal. A more important source of noise comes from the sampling error in estimating the parameters of the transition and initial condition model. We have not incorporated this type of uncertainty in these calculations. Hence, our estimates should be interpreted as point estimates which illustrate magnitudes rather than precise estimates of the effect of the scenarios we consider.

Results

As noted earlier, our objectives are: (1) to assess the extent to which health differences explain the longevity gap; and (2) to quantify the fiscal consequences of gradually closing the health gap. To meet the first objective, we examine the 2004 cohort of 50 year-olds, and consider the counterfactual in which Americans have the same health as Europeans. We compare the resulting longevity estimate to longevity predicted using the baseline health status of Americans. We also assess the total differences in public spending generated by these underlying differences

in health. The second objective requires that we analyze the consequences of gradually moving American health levels to those enjoyed by Europeans.

Explaining Differences in Longevity

We use the prevalence rates presented in Figure 2 to construct the first counterfactual cohort. We simulate the baseline outcomes of the cohort using the adjusted prevalence rates from European data. Using the methodology outlined, we preserve the correlation between health and other outcomes in the model. We keep other socio-economic characteristics constant at American levels, while varying the health status of the cohort. We then simulate transitions until everyone dies in the simulation. We compare this counterfactual with the status quo case where American health is unchanged.

Our baseline projection of remaining life-expectancy at age 50 is 31 years. This is very close to the 30.98 years estimated from life tables, as shown in Figure 1. Assigning European health status levels to Americans increases healthy life expectancy (years without ADLs) by 1.3 years, and decreases unhealthy life expectancy (years with 1+ ADLs) by a tenth of a year (virtually zero). The overall effect is to increase life expectancy by 1.2 years, which is 92% of the difference in life expectancy reported in World Health Organization data. In other words, differences in health status explain nearly all the longevity difference across the US and Europe. Moreover, these findings indicate that worse health in the U.S. is associated with a loss of *healthy* life expectancy, rather than an increase in *unhealthy* life expectancy.

The Fiscal Consequences of Differences in Health

The differences in health across the US and Europe have important fiscal consequences. Table 2 computes the overall fiscal effects on a per capita basis. Revenue would increase by $2,425 per capita, partly due to the increase in life expectancy and increase in earnings/labor

force participation. On the expenditure side, there are two effects. First, old age pension benefits would increase by a substantial amount ($6,593 per capita). This is roughly the size of the average annual Social Security benefit payment. But, there would be a larger decrease in Medicare, Medicaid, and Disability insurance (DI) benefit payments: Total lifetime health-care expenditures would decrease by a stunning $17,791. This represents an 8.5% reduction in lifetime medical expenditures. The average reduction in lifetime payments is $4,717 for Medicare and $3,687 for Medicaid. Adding the reduction in DI costs, the net effect on government expenditures would be $2,477 per capita. Overall, the net fiscal impact of this scenario is an increase in per capita net revenue of $4,902 per capita for the government.

Long-Term Fiscal Consequences of US Health Improvements

The experiment we describe above computes the contribution of health differences between the US and Europe to differences in longevity and public spending. But we are also interested in the more practical question concerning the consequences of gradually moving cohorts of near-elderly Americans towards the health status of their European counterparts.

To implement a gradual scenario, we allow prevalence rates in the entering cohort to reach European levels by 2030. This is compared to a status quo scenario in which trends currently observed and projected among the new elderly persist. This scenario can be interpreted as a transition from the current steady-state where nothing is done, to a new one. Figure 4 provides the time path for various conditions among entering cohorts in the baseline and counterfactual scenarios. Each year, the population alive is representative of the age 50+ population alive in the U.S.

We report the results for the status quo in Table 3. Given current trends, we project the size of the population aged 50+ will increase by nearly 75% from 80.7 million in 2004 to 145 million

in 2050. As a test of validity, we found that the forecast of 81.4 million 65+ year-olds in 2050 is very close to that of the Social Security Administration, which predicts 80.8 million. Life expectancy for people at age 50 is projected to increase from 31 years in 2004, to 31.6 years in 2050, a very modest increase.

We compare our baseline projections to the scenario described in Figure 6—gradual movement towards European prevalence levels. Table 4 reports the results. Gradual health improvements would result in an aged 50+ population that is 4%, or 5.75 million, larger in 2050, than it would have been in the absence of those improvements. The population is also much healthier in 2030 and 2050 than under the status quo. For example, the obesity rate falls by 24 percentage points, while the prevalence of lifetime smoking and of diabetes both fall by roughly 10 percentage points each.

Government revenue rises by 10%, or $30 billion, by 2050, as a result of the longevity gains. As before, there are two offsetting expenditure effects. On one hand, longer lives imply larger annuity burdens: OASI benefits go up by $70.4 billion. On the other hand, medical costs decrease by $124 billion, or 6.7%, in 2050. Medicare saves $36.4 billion, despite the increase in longevity. The overall effect on expenditures is initially negative, but turns positive by 2050. As found in Michaud et al. (2009), a transition to better health first decreases expenditures, but gains in longevity eventually exert upward pressure on spending.

Figure 5 shows that the gains in health expenditure materialize quickly, while the annuity burden takes longer to emerge. This is essentially a timing issue: cost savings due to lower disability appear before cost increases due to extensions in life expectancy. The total effect on expenditures is largest around 2030 and goes to zero by 2050. Since revenue rises as well, the net fiscal effect is positive in 2050 but slowly converges to zero. Hence, the transition to better

health involves important fiscal effects, but these largely vanish once the new equilibrium is reached.

Figure 6 shows that, in present value terms from the 2004 perspective, the increase in tax revenue almost entirely offsets the annuity burden. The effect on health expenditures remains. The present discounted value of Medicare and Medicaid savings combined is $632 billion, or 1.6 years of combined 2004 spending on the two programs. In terms of total medical spending, the present value of those savings is $1.1 trillion dollars. The fact that these are such large amounts illustrates the potential for savings by improving population health.

Robustness to Cross-Country Differences in Diagnosis

An alternative interpretation of cross-country differences in health focuses on differences in rates of diagnosis, rather than real differences in health status. The literature documents under-diagnosis of diseases like diabetes and hypertension in the US (Smith, 2007b), but it is difficult to find comparable European studies. However, one direct analysis of this question suggests that differences in diagnosis are relatively modest. Banks et al. (2006) compare objective clinical diagnosis among men, using commonly used thresholds on biomarkers, to self-reported measures of whether respondents have previously been diagnosed. For diabetes among those aged 40-70, they find a clinical prevalence of 4.8% in the UK and 8.9% in the US, but self-reported prevalence of 4.4% and 8.6%, respectively. The cross-country difference is similar using self-reports or clinical measurements (4.2% versus 4.1%). They find a similar result for hypertension. These discrepancies are not large relative to the differences we observe in the data.

Of course, the Banks et al. evidence is somewhat narrow in its focus on diabetes and hypertension, and on the US-UK difference specifically. Differences in screening and diagnosis might be more important for other diseases like cancer, as documented in Howard et al. (2009).

To test the sensitivity of our results to diagnostic differences, we ran the cohort analysis under various assumptions about differential diagnosis. We allowed the "diagnosis effect" to account for between 0% and 100% of the total difference in measured health across the US and Europe, in 6 alternative sensitivity analyses. In these sensitivity analyses, we kept differences in obesity and smoking constant, as differential reporting across countries seems less clearly linked to systematic institutional factors. We calculate that if all of the difference we observed was due to under-reporting of disease in Europe, US life expectancy would increase by 0.25 years, as a result of differences in baseline obesity and smoking. On the other hand, if there are no differences in the rates at which diseases are diagnosed across countries, the effect is 1.2 years. If the Banks et al. result holds more generally, and there is at most a 5% difference in diagnosis, the effect drops to about 1.1 year. This suggests that the differences in health are likely to remain meaningful under reasonable assumptions about differential diagnosis.

Discussion

There is a growing longevity gap between the US and Europe with no settled interpretation. We have demonstrated that differences in observed disease prevalence can almost entirely account for this difference. In this sense, the international longevity gap appears much easier to explain than the racial or socioeconomic longevity gaps, which are not well explained by health differences. Internationally, there is no "American-specific" effect on longevity, above and beyond differences in disease at age 50. This suggests further that addressing the health gap across the Atlantic will likely erase the longevity gap, although the same cannot necessarily be said for analogous disparities within countries. The expansion of the gap in longevity and health coincided with relative increases in obesity among the US population. Policies that target

obesity and better management of chronic health conditions such as diabetes and hypertension could be effective in improving health. Moreover, near-elderly cohorts of Americans took up smoking at much higher rates than their European counterparts and while the prevalence of smoking is likely to continue decreasing, more could be done.

As we demonstrate, the gap in health and longevity has obvious private costs to the citizens suffering from disease. There are also significant public finance consequences, on the order of $17,800 in per capita medical costs, and net public finance costs of roughly five thousand dollars per capita. Gradual transitions of US cohorts towards European levels could generate large fiscal benefits. In the long-run, medical expenditures may fall by $1.1 trillion on a present value basis. Our results suggest that prevention, in the form of lowering health risks prior to age 50 may yield important benefits. This is in line with results we published on the private value of prevention for major risk factors (Goldman et al., 2009). In that research, we found that treating 50 year-old obese individuals could be worth approximately $50,000 while preventing diabetes would be worth close to $200,000. It is not clear which policies could help reach this goal. But the historical success of anti-smoking campaigns suggests that behavior change is possible. The costs of such policies will need to be weighed against the welfare and economic consequences we have analyzed in this paper.

References

Adams, P., M. D. Hurd, et al. (2003). "Healthy, wealthy, and wise? Tests for direct causal paths between health and socioeconomic status." Journal of Econometrics **112**(1): 3-56.

Andreyeva, T., P.-C. Michaud and A. van Soest: "Obesity and Health in Europeans aged 50 years and older", *Public Health*, 2007, 121, pp. 497-509.

Banks, J., M. Marmot, et al. (2006). "Disease and Disadvantage in the United States and in England." *JAMA* 295(17): 2037-2045.

Bhattacharya, J., B. Shang, et al. (2005). "Technological advances in cancer and future spending by the elderly." Health Affairs **24 Suppl 2**: W5R53-66.

Colditz, G. (1995). 'Weight Gain as a Risk Factor for Clinical Diabetes Mellitus in Women, *Annals of International Medicine',* 122, 7, 481-6

Cutler, D. M., Glaeser, E. L. and J. M. Shapiro (2003). 'Why Have Americans Become More Obese?', Journal of Economic Perspectives, 17(3), 93-118.

Cutler, D.M. and E.L. Glaeser (2006): "Why Do Europeans Smoke More than Americans?", NBER working paper 12124.

Goldman, D., M. Hurd, et al. (2004). Health status and medical treatment of the future elderly: Final Report. Santa Monica, CA, RAND Corporation.

Goldman, D. P., B. Shang, et al. (2005). "Consequences of health trends and medical innovation for the future elderly." Health Affairs **24 Suppl 2**: W5R5-17.

Goldman, D., P.-C. Michaud, D. Lakdawalla, Y. Zheng, A. Gailey (2010) : "The Fiscal Consequences of Trends in Population Health", *National Tax Journal*, 63:2, pp. 307-330.

Goldman, D., Y. Zheng, F. Girosi, P.-C. Michaud, S.J. Olshansky, D. Cutler and J. Rowe (2009): "The Benefits of Risk Factor Prevention in Americans Aged 51 and Older". *American Journal of Public Health* vol. 99 (11) pp. 2096-101.

Hayward, M. D. and D. F. Warner (2005). Demography of Population Health. The Handbook of Demography. D. L. Poston Jr and M. Micklin. New York, Springer**:** 809-825.

Howard, D., L. Richardson and K. Thorpe (2009): "Cancer Screening and Age in the United States and Europe", Health Affairs, 28:6, pp. 1838-1847.

Lakdawalla, D., and T. Philipson (in press). "The Growth of Obesity and Technological Change." *Economics and Human Biology*

Lee, R. (2003), "Mortality Forecasts and Linear Life Expectancy Trends" (March 25, 2003). Center for the Economics and Demography of Aging. CEDA Papers: Paper 2003-0003CL.http://repositories.cdlib.org/iber/ceda/papers/2003-0003CL

Lee, R. D. (2000). "The Lee-Carter Method for Forecasting Mortality, With Various Extensions and Applications." North American Actuarial Journal **4**: 80-91.

Levy, D. (2006). "Trends in Smoking Rates Under Different Tobacco Control Policies:Results from the SimSmoke Tobacco Policy Simulation Model", working paper University of Baltimore.

Manton, K., B. Singer, et al. (1993). Forecasting the Health of Elderly Populations. New York, Springer-Verlag.

Manton KG, Gu XL, and Lowrimore G. 2008. Cohort Changes in Active Life Expectancy in the U.S. Elderly Population: Experience From the 1982–2004 National Long-Term Care Survey. *The Journals of Gerontology: Series B* 63(5):S269-S281.

Oeppen, J. and J. W. Vaupel. (2002): Broken limits to life expectancy. *Science* 296:1029- 31.

Oshlansky, S.J. and B. Carnes (2001): "The Quest for Immortality: Science at the Frontiers of Aging", New York, W.W. Norton.

Preson, S.H. and J. Ho (2009): "Low Life Expectancy in the United States: Is the Health Care System at Fault?", NBER Working Paper 15213.

Rogers, R. G. and E. Powell-Griner (1991). "Life Expectancies of Cigarette Smokers and Nonsmokers in the United States." *Social Science and Medicine* 32(10): 1151-9.

Ruhm, C. (2007) "Current and Future Prevalence of Obesity and Severe Obesity in the United States," *Forum for Health Economics & Policy*: Vol. 10: Iss. 2 (Obesity), Article 6.

Smith, J. (2007a): " "The Impact of Socioeconomic Status on Health over the Life-Course," *Journal of Human Resources*, 42(4):739-764.

Smith, J. (2007b), "Nature and Causes of Trends in Male Diabetes Prevalence, Undiagnosed Diabetes, and the Socioeconomic Status Health Gradient", *Proceedings of the National Academy of Sciences*, 104(33) 13225-1323, 2007

Thorpe, K.E., D.H. Howard and K. Galactionova (2007): "Differences in Disease Prevalence as a Source of the U.S.-European Health Care Spending Gap", Health Affairs 26:6, pp. 678-686.

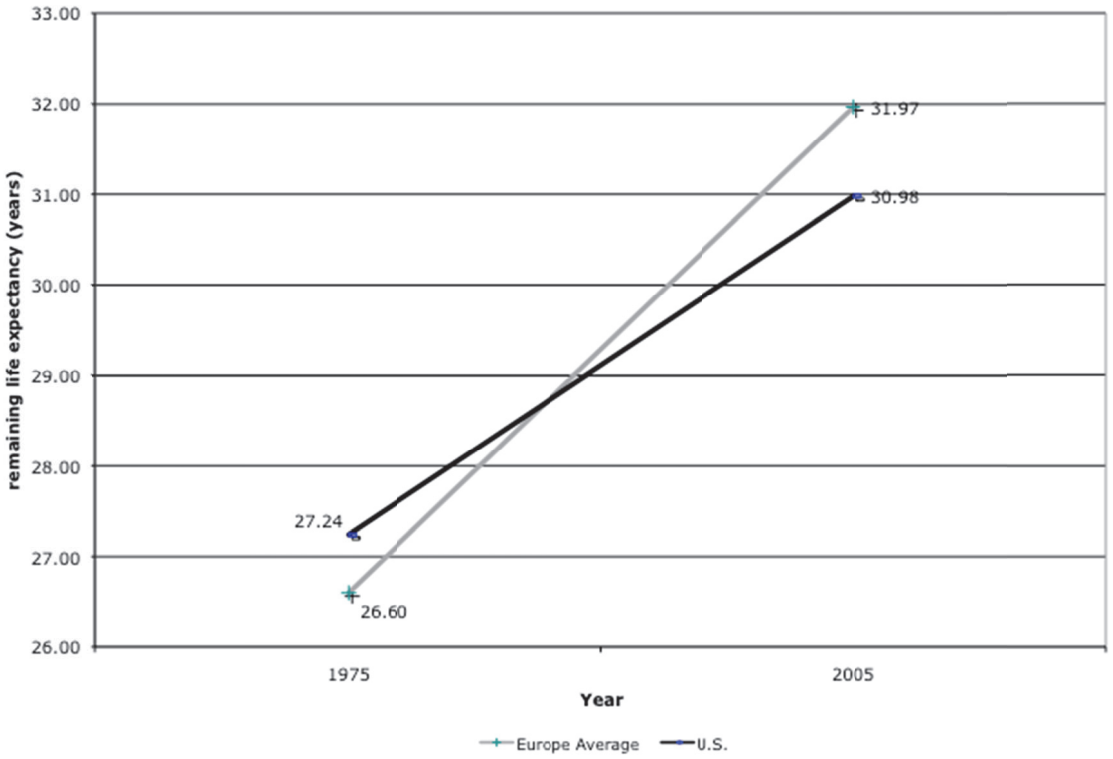
Tuljapurkar, S., N. Li and C. Boe (2000): "A Universal Pattern of Mortality Decline in the G7 Countries", *Nature* 405, pp. 789-792.

Viscusi, K. and J.E. Aldy (2003): "The Value of a Statistical Life: A Critical Review of Market Estimates throughout the World," Journal of Risk and Uncertainty, Vol. 27, No. 1 pp. 5-76.

White, K. (2002): "Longevity Advances in High-Income Countries, 1955-1996", Population and Development Review, 28:1, pp. 59-76.

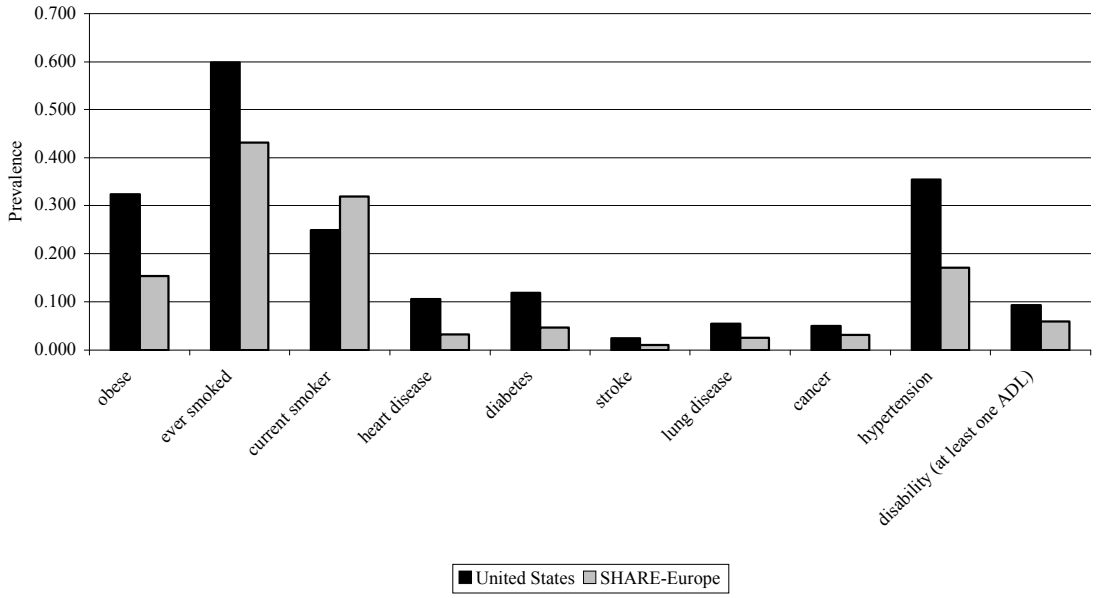
Willett, W. (1995). 'Weight, Weight Change and Coronary Hearth Disease in Women', *Jama*, 273,6, 461-5

Figure 1 Remaining Life Expectancy at Age 50: U.S. – Europe (EU-15) Differences from 1975 to 2006



Source: Human Mortality Database period life tables for 1975 and 2005. EU 15 countries are: Austria, Belgium, Denmark, Finland, France, Ireland, Italy, Luxembourg, The Netherlands, Norway, Portugal, Spain, Sweden, U.K. and West Germany. Weighted average using population size age 50.

Figure 2 Health Differences between U.S. and SHARE-Europe Population Aged 50-53



Source: Health and Retirement Study 2004 and Survey of Health Ageing and Retirement in Europe (SHARE) 2004 (Denmark, France, Germany, Greece, Italy, the Netherlands, Spain, Sweden). Data from Austria not included because of lack of appropriate population weights and Switzerland because of low response rate and small sample. Sample weights used. Question text and definition are available in Technical Appendix. Obese status defined as body-mass index greater than 30. ADL are limitations in activities of daily living.

Figure 3

Overview of the Future Elderly Model

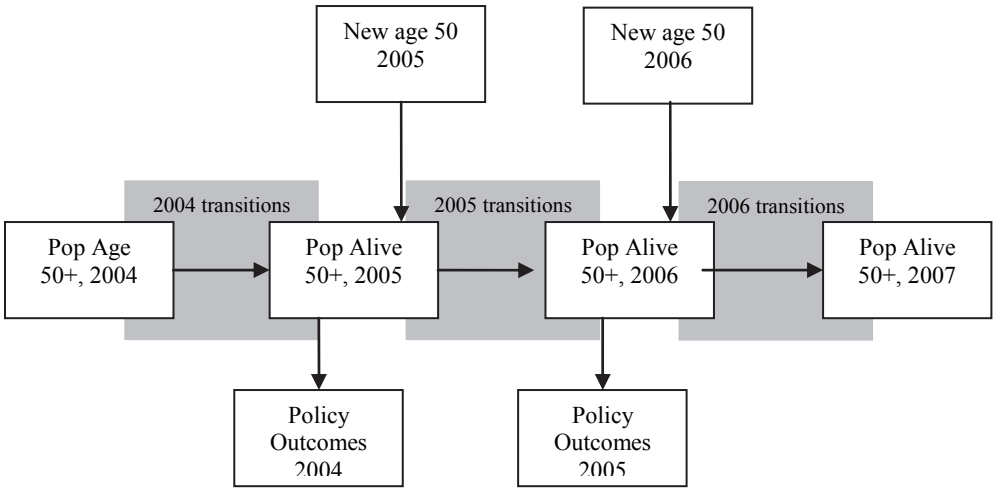
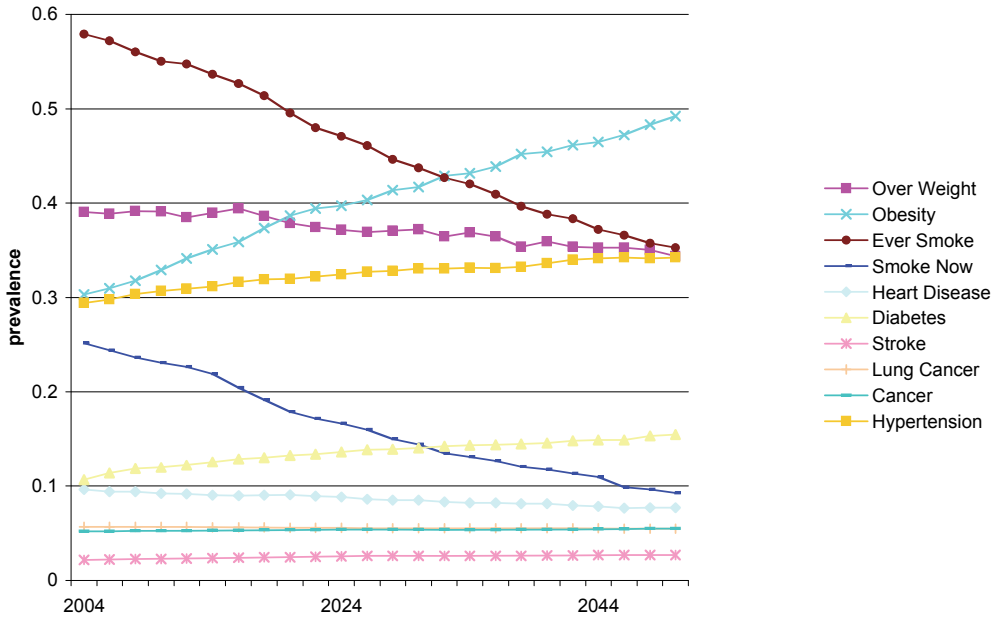


Figure 4 Population Scenarios

Status Quo Scenario (Prevalence in Entering Cohort)



European Scenario (Prevalence in Entering Cohort)

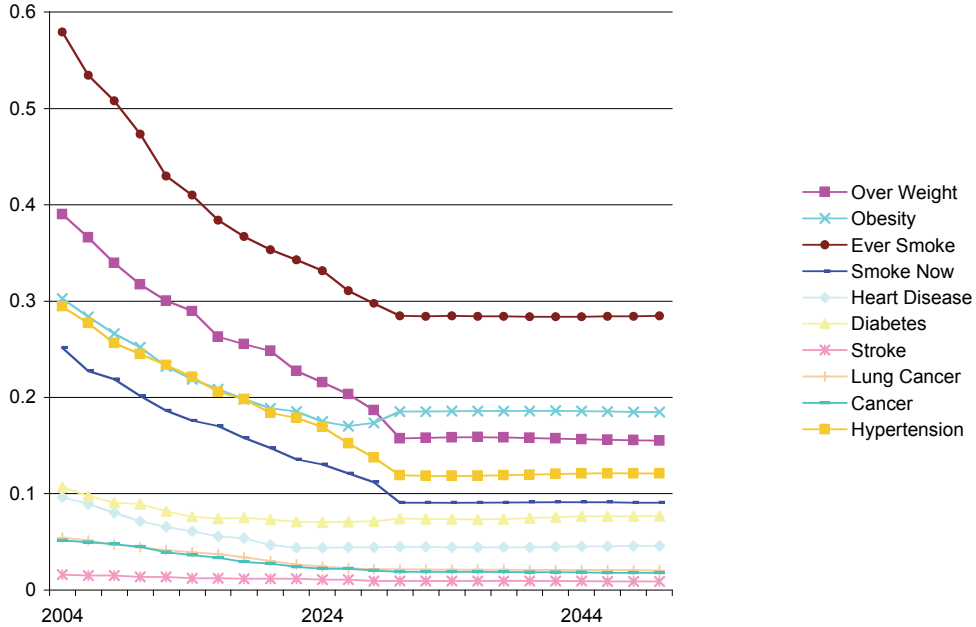
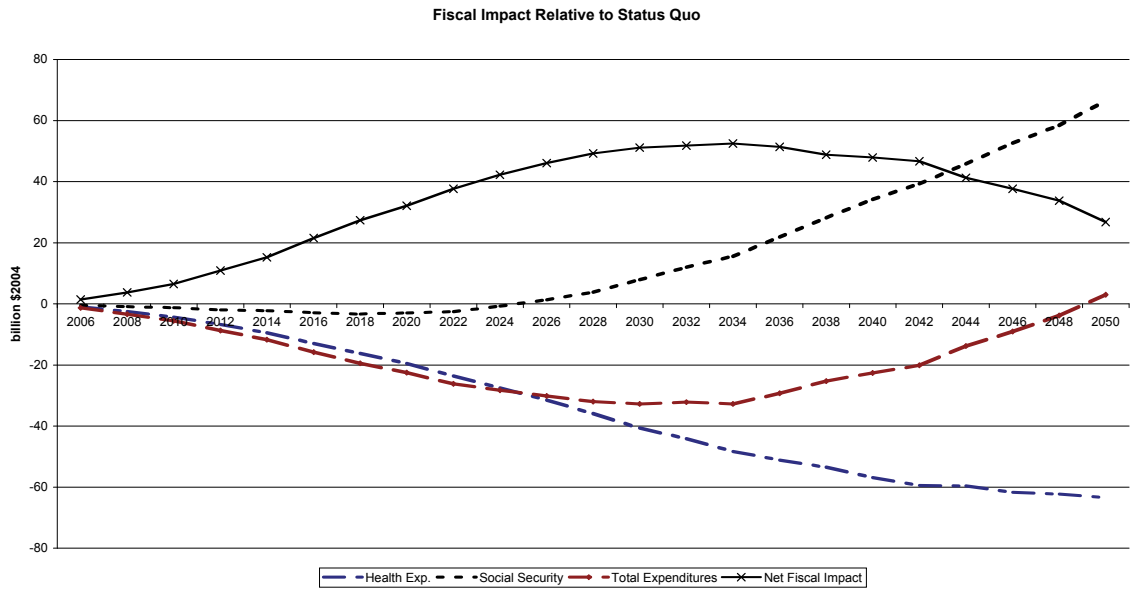
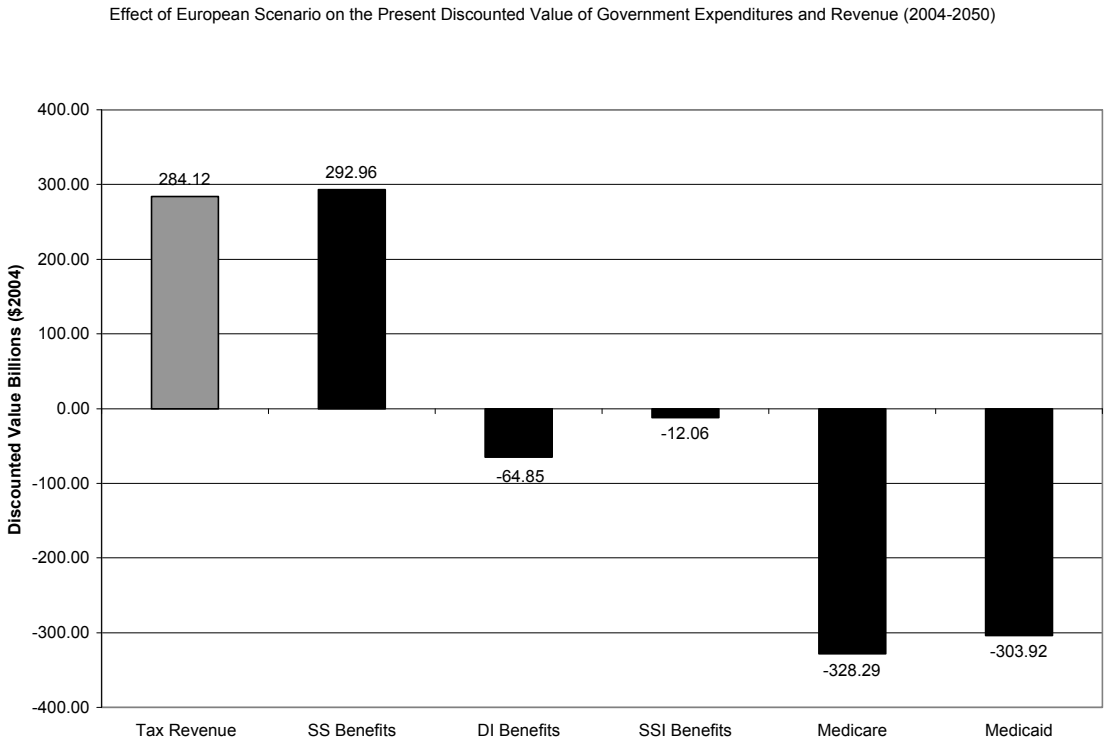


Figure 5



Source: Authors' own calculations using the microsimulation model. Health expenditures include Medicare and Medicaid. Social Security includes SSI, DI and OASI expenditures. Net Fiscal Impact is the revenue change minus the total expenditure change. All amounts in billions $2004 and refer to the difference between the European scenario and the status quo defined in Figure 4.

Figure 6



Source: Authors' calculations using the microsimulation model. Amounts in billion $2004 and represent the difference between the European scenario and the status quo scenario as defined in Figure 4. Present discounted value calculated using a 3% real discount rate from 2004 to 2050. Tax revenue includes Federal, State and Social Security and Medicare Taxes. SS stands for Social Security Benefit Payments, DI for disability insurance payments, SSI for Supplemental Security Income payments.

Table 1

Outcomes in the Future Elderly Model

Initial Conditions Outcomes	
Economic Outcomes	**Health Outcomes**
Employment	Hypertension
Earnings	Heart Disease
Wealth	Self Reported Health
Defined Contribution Pension Wealth	BMI Status
Pension Plan Type	Smoking Status
AIME (average indexed monthly earnings)	Functional Status
Social Security Quarters of Coverage	
Health Insurance	

Transition Outcomes	
Economic Outcomes	**Health Outcomes**
Employment	Death
Earnings	Heart
Wealth	Stroke
Demographics	Cancer
Health Insurance	Hyper-tension
Disability Insurance Claim	Diabetes
Defined Benefit Claim	Lung Disease
SSI Claiming	Nursing Home
Social Security Claiming	BMI Status
	Smoking Status
	Functional Status

Notes: More detail on each outcome in online technical appendix.

Table 2

Per Capita Lifetime Fiscal Effects of European Health Scenario for Cohort in 2004

Expenditure Category	Status Quo	European Scenario	Difference
Government Revenues			
Federal Tax	46,289	47,637	1,348
State Tax	16,035	16,535	500
Social security payroll taxes	16,566	17,031	465
Medicare payroll taxes	4,020	4,132	112
Total	82,910	85,335	2,425
Government Expenditures			
Old Age and Survivors Insurance benefits (OASI)	138,123	144,716	6,593
Supplementary Security Income (SSI)	3,454	3,471	17
Disability Insurance benefits (DI)	6,356	5,673	-683
Medicare costs	73,391	68,674	-4,717
Medicaid costs	21,745	18,058	-3,687
Total	243,069	240,592	-2,477
Net Fiscal Effect			4,902
Total Health Care Expenditures	210,993	193,202	-17,791

Source: authors' calculations using the microsimulation model. Amounts reported in $2004 USD. Present discounted values computed using a real discount rate of 3%. Total Health Care Expenditures include private health insurance and other expenditures not covered by Medicare and Medicaid.

Table 3

Population Level Outcomes Under Status-Quo Scenario (2004-2050)

	Status Quo Estimates		
	Year		
	2004	2030	2050
Population size (Million)	80.71	122.13	145.05
Population 65+ (Million)	36.25	66.87	81.37
Prevalence of selected conditions			
Obesity (BMI >=30) (%)	28.1%	41.1%	45.8%
Overweight (25<=BMI<30) (%)	38.1%	37.8%	36.3%
Ever-smoked	58.6%	48.0%	38.6%
Smoking now	16.9%	9.6%	6.2%
Diabetes	17.0%	24.8%	27.8%
Heart disease	23.0%	28.1%	29.9%
Hypertension	50.9%	58.9%	62.2%
Government revenues from aged 51+ (Billion $2004)			
Federal personal income taxes	216.44	228.62	249.33
Social security payroll taxes	73.82	86.79	96.63
Medicare payroll taxes	18.67	20.98	23.33
Government expenditures from aged 51+ (Billion $2004)			
Old Age and Survivors Insurance benefits (OASI)	417.15	992.47	1,272.07
Disability Insurance benefits (DI)	36.99	36.02	40.77
Supplementary Security Income (SSI)	17.06	26.44	37.94
Medicare costs	290.24	549.44	735.69
Medicaid costs	118.72	152.66	228.44
Total medical costs for aged 51+ (Billion $2004)	851.05	1,412.58	1,826.03

Source: authors' calculation using the microsimulation model under the status quo scenario described in the text. All dollars are in 2004 values. Output reported for the years 2004, 2030 and 2050.

Table 4

Population Level Outcomes under European Scenario (2004-2050)

	European Scenario		Absolute Change	
	2030	2050	2030	2050
Population size (Million)	123.74	150.81	1.605	5.752
Population 65+ (Million)	67.95	86.52	1.072	5.147
Prevalence of selected conditions				
obesity (BMI >=30) (%)	27.5%	21.6%	-0.136	-0.242
over weight (25<=BMI<30) (%)	36.4%	35.6%	-0.013	-0.007
Ever-smoked	38.2%	28.0%	-0.098	-0.107
Smoking now	7.9%	5.3%	-0.016	-0.010
Diabetes	19.1%	17.0%	-0.057	-0.108
Heart disease	25.1%	25.7%	-0.030	-0.042
Hypertension	50.3%	48.3%	-0.086	-0.139
Government revenues from aged 51+ (Billion $2004)				
Federal personal income taxes	244.59	275.97	15.970	26.644
Social security payroll taxes	88.75	99.11	1.957	2.481
Medicare payroll taxes	21.46	23.95	0.480	0.620
Government expenditures from aged 51+ (Billion $2004)				
Old Age and Survivors Insurance benefits (OASI)	1 004.97	1 342.42	12.499	70.358
Disability Insurance benefits (DI)	32.29	35.94	-3.732	-4.837
Supplementary Security Income (SSI)	25.55	38.83	-0.890	0.890
Medicare costs	527.96	699.21	-21.481	-36.488
Medicaid costs	133.50	201.49	-19.158	-26.951
Net Fiscal Effect			51.169	26.773
Total medical costs for aged 51+ (Billion $2004)	1 327.72	1 701.61	-84.854	-124.422

Source: authors' calculations using the microsimulation model under the European scenario. All dollars in 2004 values

Paper Three: Patents, Innovation, and the Welfare Effects of Medicare Part D[25]

As previously seen in: Gailey, Adam H., Darius Lakdawalla, and Neeraj Sood. 2010. Patents, Innovation, and the Welfare Effects of Medicare Part D. In *Pharmaceutical Markets and Insurance Worldwide*, edited by A. Dor: Emerald Group Publishing Limited.

Abstract

Purpose: To evaluate the efficiency consequences of the Medicare Part D program.

Methods: We develop and empirically calibrate a simple theoretical model to examine the static and dynamic welfare effects of Medicare Part D.

Findings: We show that Medicare Part D can simultaneously reduce static deadweight loss from monopoly pricing of drugs and improve incentives for innovation. We estimate that even after excluding the insurance value of the program, the welfare gain of Medicare Part D roughly equals its social costs. The program generates $5.11 billion of annual static deadweight loss reduction, and at least $3.0 billion of annual value from extra innovation.

Implications: Medicare Part D and other public prescription drug programs can be welfare-improving, *even for risk-neutral and purely self-interested consumers*. Furthermore, negotiation for lower branded drug prices may further increase the social return to the program.

Originality: This study demonstrates that pure efficiency motives, which do not even surface in the policy debate over Medicare Part D, can nearly justify the program on their own merits.

[25] We are grateful to the National Institute on Aging for funding (1R01AG021940). For many helpful comments and suggestions, we thank seminar participants at the 2007 NBER Summer Institute Health Care session.

I. Introduction

Patents encourage innovation by awarding inefficient monopoly power to inventors. This leads to the familiar trade-off between inducing innovation, and ensuring the efficient utilization of invented goods. Public prescription drug insurance provides a way out of this dilemma, because it helps decouple the price consumers pay from the price innovators receive. By subsidizing co-insurance for drugs, public insurance encourages utilization, but without necessarily compromising innovators' prices, profits, and incentives for research. As such, public insurance can simultaneously promote static and dynamic efficiency, which are often at odds (Lakdawalla and Sood, 2009).

The rationale for public drug insurance is typically thought to rely on the social value of insuring against financial risk, and on the value of care provided to the less affluent. However, its static and dynamic efficiency effects imply that public drug insurance is valuable to *risk-neutral, self-interested consumers*. We demonstrate that this point has a considerable impact on the welfare analysis of Medicare Part D. Even though Part D is not a particularly generous insurance plan, we find that it generates an annual value of $5.1 billion in static deadweight loss reduction, and $3.0 billion in additional innovation. The total value of $8.1 billion is approximately 95% of Part D's social cost. Part D nearly breaks even, even ignoring the insurance and altruism aspects of the program typically thought to generate its entire social value.

We begin with some relevant background material, and then present our analysis in three parts. Section III presents a simple theoretical model that demonstrates the welfare effects of Medicare Part D, as well as the approach to calculating them. Section IV uses the theoretical model to quantify the welfare effects of the program.

II. The Medicare Part D Program

Medicare Part D subsidizes the costs of prescription drugs for Medicare beneficiaries and was introduced by the passage of the Medicare Prescription Drug, Improvement, and Modernization Act of 2003 (MMA). Beneficiaries can obtain the Medicare Drug benefit through two types of private plans: beneficiaries can join a Prescription Drug Plan (PDP) for drug coverage only, or they can join a Medicare Advantage plan (MA) that covers prescription drugs (MA-PD). Beneficiaries are required to make premium payments to obtain Part D coverage. However, premiums are highly subsidized. Medicare Part D covers roughly 75% of the costs (Kaiser Family Foundation, 2007, Kaiser Family Foundation, 2008a).

Medicare Part D establishes a standard drug benefit that Part D plans may offer. The standard benefit is defined in terms of the benefit structure and not in terms of the drugs that must be covered. In 2007, this standard benefit requires payment of a $265 deductible. The beneficiary then pays 25% of the cost of a covered Part D prescription drug up to an initial coverage limit of $2,400. Once the initial coverage limit is reached, the beneficiary is subject to another deductible, commonly known as the "Donut Hole," in which they must pay the full cost of drugs. When total out-of-pocket expenses on formulary drugs for the year, including the deductible and initial coinsurance, reach $3,850, the beneficiary then reaches catastrophic coverage, in which he or she pays a 5% coinsurance. In practice, Part D plans might deviate from this standard benefit but they must offer coverage that is equivalent to or better than the standard benefit in actuarial terms. The law also stipulates that employers sponsoring prescription drug coverage for retirees can receive a federal subsidy if the coverage is at least actuarially equivalent to the standard Medicare drug benefit. Employers would receive a 28% subsidy to their portion of the individual retiree's drug costs between $250 and $5,000. Finally, Medicare Part D also provides more

generous insurance and additional subsidies to low-income beneficiaries. Currently, dual-eligible (eligible for both Medicare and Medicaid) beneficiaries constitute the majority of the beneficiaries receiving low income subsidies as they are automatically enrolled in Part D plans (Kaiser Family Foundation, 2008a, Kaiser Family Foundation, 2008b).

One of the most controversial features of the MMA was it did not allow Medicare to negotiate prices directly with pharmaceutical companies. Many critics regard this as poor stewardship of tax dollars, while those in favor argue that price-negotiation could dampen innovation incentives by lowering pharmaceutical profits. While this study does not resolve this controversy, the absence of price-negotiation is relevant insofar as it exerts upward pressure on manufacturer prices, which in turn affects dynamic incentives to innovate.

III. The Welfare Effects of Stand-Alone Public Drug Insurance

In earlier theoretical work, we analyzed more generally the welfare effects of public prescription drug insurance (Lakdawalla and Sood, 2009). In this section, we provide a simpler and more concrete analysis applying these results to the case of Part D, which has static and dynamic effects on welfare. Public prescription drug insurance has the potential to lower deadweight loss and raise monopoly profits simultaneously. It can achieve this outcome by partially decoupling the consumer's price from the revenue earned by the monopolist.

Static Implications

The provision of drug insurance can reduce deadweight loss, because copayments below the monopoly price increase utilization by consumers. Define $D(p)$ as the demand function, $P(Q)$ as inverse demand, MC as the constant marginal cost of production, and p_m as the equilibrium monopoly price. The social surplus generated by competitive provision of the good is given by:

$$SS_c \equiv \left(\int_0^{D(MC)} P(Q)dQ - MC * D(MC) \right) \quad (1)$$

In the absence of insurance, deadweight loss in the branded pharmaceutical market is social surplus under competition, minus social surplus under monopoly:

$$DWL \equiv SS_c - \left(\int_0^{D(p_m)} P(Q)dQ - MC * D(p_m) \right) \quad (2)$$

Now suppose that the government offers prescription drug insurance. Specifically, suppose the government covers the share $(1-\sigma)$ of the market price, and leaves the consumer with the co-insurance rate σ. If the government continues to pay the monopoly price for pharmaceuticals, the actuarial cost of the insurance is $p_m(1-\sigma)D(\sigma p_m)$. From a purely static point of view, this cost is simply a transfer from the government to the pharmaceutical industry; later, we incorporate the deadweight cost of public funds, which is so far absent. The welfare effects emerge from the change in quantity induced by this policy.

Of course, Part D could impact the monopoly price charged for pharmaceuticals. The monopolists may exploit the subsidy to consumers and choose to raise prices. In earlier theoretical work, we characterize the class of benefit designs that ensure efficiency gains from public prescription drug insurance even if the monopolist raises prices in response to drug coverage (Lakdawalla and Sood, 2009). Another possibility is that the government or private plans participating in the benefit may use their newfound monopsony power to negotiate prices downward (Duggan and Scott Morton, 2008). For now, we analyze the welfare effects of the program, given some arbitrary post-Part D monopoly price, $p_m^{'}$. In the empirical section we calibrate the welfare effects of Part D under alternate assumptions about the price charged by manufacturers.

Deadweight loss is a function of what the public program pays monopolists and offers consumers in terms of co-insurance. This relationship can be expressed as:

$$DWL(p_m^{'}, \sigma) \equiv \left(\int_0^{D(MC)} P(Q)dQ - MC * D(MC) \right) - \left(\int_0^{D(\sigma p_m^{'})} P(Q)dQ - MC * D(\sigma p_m^{'}) \right) \quad (3)$$

Ultimately, what matters for deadweight loss is simply $\sigma p_m^{'}$, the price faced by consumers. The welfare effect of lowering the consumer price — either by lowering the co-insurance or by lowering the price paid to monopolists — is given by:

$$-\frac{dDWL}{d\sigma p_m^{'}} |_{p_m^{'}, \sigma} = D'(\sigma p_m^{'})\left(\sigma p_m^{'} - MC\right) \quad (4)$$

From a static point of view, lowering the price paid by consumers always lowers deadweight loss, as long as $\sigma p_m^{'} > MC$, or that consumers continue to face a price that is at least as large as marginal cost. Empirically, this assumption seems to hold for Medicare Part D. Marginal cost is typically estimated to be 20% of the branded drug price (Caves et al., 1991, Grabowski and Vernon, 1992, Berndt et al., 1996). In contrast, the average coinsurance rate under Medicare Part D is currently about 62%, well above marginal cost.[27]

Dynamic Implications

The original intent of Medicare Part D was to provide drug insurance without affecting prices paid to innovators. Earlier, we showed that drug insurance improves static welfare by lowering deadweight loss. We now show that this original aspect of Part D induces more innovation, and increases dynamic social surplus.

[27] This number was calculated by the authors using the Medical Expenditure Panel Survey (MEPS) data described later. Appendix A presents a formal proof that the anticipated percentage change in co-insurance rate (along with the elasticity of demand) is a sufficient predictor of the change in utilization, even with a nonlinear benefit design.

Let I denote industry investment in research, and let $g(I)$ denote the probability of discovery with $g'(I) > 0$ and $g''(I) < 0$. In other words, R&D investment raises the probability of new drug discovery, but in a concave fashion. Suppose the innovator enjoys a patent monopoly for T periods after the discovery and will make zero profits thereafter. If the firm discounts the future at the rate r, it invests in research in order to maximize the present value of expected profits:

$$\Pi(I) = g(I)\left[\int_0^T e^{-rt}\pi(\sigma, p_m)dt\right] - I \tag{5}$$

By the envelope theorem, stand-alone drug insurance raises the expected profits of innovators, because $\frac{d\pi}{d\sigma} < 0$, and will also induce more innovation. The privately optimal level of innovation is given by:

$$g'(I) = \frac{1}{\left[\int_0^T e^{-rt}\pi(\sigma, p_m)dt\right]} \equiv \frac{1}{\Pi^m(\sigma, p_m, T)} \tag{6}$$

The marginal product of research is the reciprocal of monopoly profits, Π^m and, by extension, of patent length (Nordhaus, 1969). Therefore, since reductions in co-insurance raise profits, they must also stimulate innovation.

Define $I_{pat}(\Pi^m)$ as the level of investment induced by monopoly profits Π^m. Expected social surplus can be written as:

$$S(T, p_m, \sigma) \equiv g(I_{pat}(\Pi^m))\left[\int_0^\infty e^{-rt}SS_c dt - \int_0^T e^{-rt}DWL(\sigma p_m)dt\right] - I_{pat}(\Pi^m) \tag{7}$$

The marginal value of introducing stand-alone drug insurance is given by:

$$S_{\sigma}\,|_{\sigma=1} = I_{\pi}\Pi_{\sigma}^{m}\left\{g'(I)\left[\int_0^{\infty} e^{-rt}SS_c\,dt - \int_0^{T} e^{-rt}DWL(p_m)dt\right] - 1\right\} + g\left[-\int_0^{T} e^{-rt}DWL'(p_m)\,p_m dt\right] < 0 \tag{8}$$

The term in curly braces is strictly greater than unity, because total social surplus from the innovation must be strictly larger than the innovator's profits.[28] Therefore, the first term is negative. This measures the value of drug insurance as a stimulant to innovation. The second term is negative, because $p_m > MC$, implying that deadweight loss will rise with a higher price. This is the value of insurance in mitigating deadweight loss.

Notice the important presumption that the profits of innovators do not exceed social surplus. Clearly, this condition always holds in a completely private market, even one afflicted by moral hazard in insurance provision (Lakdawalla and Sood, 2006). Intuitively, consumers would never voluntarily pay more than their consumer surplus for a drug in a spot market, and they would never pay more for an insurance policy than the expected value of its covered treatments. Public subsidies for employer-provided health insurance make it theoretically possible that profits could exceed social surplus. However, given the estimated rate of surplus-appropriation by innovators (see the discussion beginning on page 83), this would require extremely large transfers. It is even less likely among the elderly population, where prescription drug insurance was relatively uncommon.[29]

Another more controversial question concerns whether there is currently too much or too little innovation, or equivalently, how much innovators ought to be able to appropriate. In the standard model, innovators ought to appropriate the full value of social surplus, which is

[28] In theory, distortions like subsidies for health insurance could result in profits being higher than social surplus. However, we later document empirical evidence confirming social surplus is larger than profits.

[29] As of 2003, 60% of the aged (65+) population had no drug insurance, or insurance that was less generous on average than the standard Part D benefit (based on authors' calculations from the Medical Expenditure Panel Survey).

impossible in the absence of price-discrimination. Some economists have pointed out that patent races, public subsidies, and other imperfections can alter this result, sometimes substantially. Others have emphasized the extremely low rates of social-surplus appropriation by innovators.[30]

Resolving this controversial question lies beyond the scope of this paper, but we can interpret our analytical results, regardless of whether innovation is too high or too low. If, as in the standard case, innovation is too low, Part D has a direct welfare benefit, without any auxiliary provisions. This is the analysis presented above. If in fact there is *too much* innovation, the direct effects of Part D on innovation reduce social welfare. However, this adds additional value to price-negotiation or similar measures to limit, or even reduce, the profits of innovators. In this case, a Part D program coupled with price-reductions that hold innovator profits constant would be strictly welfare-improving. The rest of the analysis is presented from the point of view of the standard model, that there is too little innovation; the possibility of "over-innovation" is discussed further in the sensitivity analyses of Section IV.

Public Financing and Deadweight Cost

In the analysis above, we abstracted from the costs of public-financing. When prescription drug insurance is wholly or partly financed by the public-sector, the last component of the welfare calculation must be the deadweight cost from public, rather than private, financing.

IV. Calibrating the Welfare Effects

In this section, we calculate—in a "back-of-the-envelope" fashion – the welfare impacts of Medicare Part D. We consider both the static and dynamic benefits of increased drug consumption and the associated increase in pharmaceutical innovation induced by Medicare Part

[30] For contrasting views in the context of pharmaceuticals, see Garber et al., (2006), compared with Philipson and Jena (2006). In a broader context, see Shapiro (2007), compared with Nordhaus (2004).

D. We also estimate the social costs of financing this benefit due to deadweight loss from increased taxation. We exclude dual-eligibles from the analysis, because they already receive generous public prescription drug insurance from Medicaid. The introduction of Medicare Part D does not substantially change the generosity of insurance for dual-eligibles; it merely transfers insurance from Medicaid to Medicare Part D.

The net benefits of Part D can be calculated according to the following equation:

$$NB = Enrollees * (PC\ Static\ Benefit) + (Dynamic\ Benefit) - (Deadweight\ Cost) \quad (9)$$

In words, the net benefit of the program is equal to: the number of enrollees multiplied by the per capita static benefits to those enrollees, plus the dynamic benefit of the program, minus the deadweight costs of financing. The first term embodies the utilization effects on enrollees, the second the innovation effects, and the third the social cost of funding premiums and employer subsidies. A more detailed exposition of this calibration is provided below.

Part D Enrollees and Non-Enrollees

We need to estimate both the number of enrollees, and the number of non-enrollees eligible for the employer subsidy payment. The latter group affects the financial cost of the program, if not the static welfare benefit. We used data from the 2005 Medical Expenditure Panel Survey (MEPS) and the formularies from the top 5 Medicare Part D insurance plans to estimate these quantities.

Methods

The Medicare Modernization Act classifies an individual's private prescription drug coverage as "creditable" or "non-creditable." Broadly speaking, "creditable coverage" is private insurance that is actuarially at least as generous as Medicare Part D. Individuals without creditable coverage are required to enroll in Part D within 63 days of being eligible, or face a late

enrollment penalty if and when they enroll. Those with creditable coverage are not subject to this penalty provision. Moreover, if a firm provides a Part D-eligible employee with creditable coverage, that firm is entitled to a 28% subsidy on their portion of the individual retiree's drug costs between $250 and $5,000.

In our calculations, we assume that there are no costs of switching to Part D. Therefore, everyone without creditable coverage (i.e., less generous than Part D) switches to the program. We also assume that people with creditable coverage refuse to switch.[31] Evidently, therefore, we need to identify individuals with and without "creditable coverage." To do so, we use actual prescription drug expenditures observed in the MEPS,[32] and calculate the average co-insurance rate generated by the individual's current plan (or lack thereof), and the lowest cost option among the top 5 Medicare Part D plans. Those whose average co-insurance rate is lower under Part D are classified as having no creditable coverage, and vice-versa. Appendix A presents a formal proof that the average co-insurance rate is a sufficient predictor of value for an insurance plan, even one with a nonlinear benefit design.

Estimates

Using the MEPS sampling weights we estimate that, excluding dual-eligibles, 36 million beneficiaries would be eligible for Medicare Part D. Next, for each MEPS respondent we then calculate the average coinsurance for prescription drugs under 2 scenarios: (1) their current coverage; or (2) enrollment in most generous among the top 5 Part D plans, described earlier.

[31] This assumption has less transparent foundations. Clearly, people with very generous coverage fail to switch, but the effects for people with marginally more generous coverage are theoretically unclear. On the one hand, the subsidy of the Part D premium suggests that some may switch to Part D. On the other hand, employers could pass along the subsidy they receive for privately insuring a worker who is still employed, which may fully counteract the value of the premium subsidy. We adopt the simple assumption of no-switching, because it generates results that reasonably match actual enrollment data.

[32] Notice that we calculate the effective co-insurance rate from current spending. This is equivalent to calculating the first-order welfare effect of switching to Part D. Individuals with a positive first-order welfare effect will benefit, and vice-versa.

Based on this analysis, we identify individuals with and without creditable coverage. We estimate that 61% of eligible respondents, or roughly 22 million beneficiaries, have no creditable coverage. We assume all these individuals enroll in Part D.

The remaining 14 million already have more generous insurance compared to the standard Part D benefit and are assumed not to enroll in Part D. Based on information about the source of coverage in MEPS we estimate that roughly 57% of those with creditable insurance have it through an employer or union, which would then receive the employer subsidy instituted by Medicare Part D. The remaining 43% of those with creditable insurance have it from other sources such as the Veterans Administration, Indian Health Service and state pharmaceutical assistance programs. The employer subsidy is not paid in these cases. Our estimates are presented in Table 1.

As a validity check, we compared our enrollment estimates to the actual enrollment rates reported by the Department of Health and Human Services (HHS), also shown in Table 1. The HHS estimates show that, as of January 2007, and excluding dual-eligibles, 36 million beneficiaries were eligible for Medicare Part D. Of these, 21 million were estimated to have no creditable coverage prior to Part D.[33] The remaining 15 million had creditable coverage from employer/union or from other sources such as Veterans Administration, Indian Health Service and state pharmaceutical assistance programs (Kaiser Family Foundation, 2007). These numbers are quite similar to the estimates we derived from analysis of the MEPS.

[33] Of the 21 million beneficiaries, 17 million enrolled in Part D and the remaining 4 million continued to have no creditable coverage. We assume that, over the long-run, these remaining 4 million respondents will switch into the more generous coverage afforded by the Part D program. If not, this incomplete take-up rate would lower the welfare benefits of the program.

Static Benefits

The next step is to compute the static benefit enjoyed by enrollees in Part D. For both static and dynamic benefits, prices and expenditures are for branded drugs only, due to the lack of monopoly power in generic drugs. Using a linear approximation to demand, the benefit associated with a particular change in price and quantity for branded drugs is simply the reduction in the size of deadweight loss "triangle," or

$$\frac{1}{2}(p_{nd} - mc)(q_{nd} - q_c) - abs(\frac{1}{2}(p_d - mc)(q_d - q_c)).$$

In this expression, p_{nd} is the price of drugs without Part D, mc is the marginal cost of producing drugs, p_d is the price of drugs under Part D, q_c is the quantity of drugs consumed under perfect competition, q_{nd} is the quantity of drugs without Part D, and q_d is the quantity of drugs consumed with Part D. Overall, this yields the new level of the deadweight loss triangle subtracted from the original level of dead weight loss. This benefit can be equivalently written using the anticipated percentage changes in price and quantity, along with the elasticity of demand. Appendix A derives the formal conditions under which the anticipated percentage change in co-insurance rate and elasticity of demand are sufficient predictors of the change in utilization, even with a nonlinear benefit design.

Assuming that the price paid to the manufacturer does not change, some simple algebra yields the equivalent formulation of the static benefit for an enrolling consumer[34]:

[34] The baseline simulation assumes that prices do not change. The policy simulations section presented later considers the welfare effects of changing manufacturer prices.

$$SB = \frac{1}{2}(\sigma_{ND} - MC)(Expenditures_c - Expenditures_{ND})$$
$$- abs(\frac{1}{2}(\sigma_D - MC) \qquad (10)$$
$$* ((\left[\frac{(\sigma_{ND} - \sigma_D)}{\sigma_{ND}} e\right] + 1)(Expenditures_{ND}) - Expendiutres_c))$$

The terms σ_D and σ_{ND} are the average share of price paid by the consumer with and without Part D, respectively. $Expenditures_{ND}$ measures expenditures under the status quo; $Expenditures_c$ measures them under perfect competition. The term e is the elasticity of demand. To calculate the static welfare impact of Medicare Part D, we need empirical estimates of: (1) The percentage change in branded drug price to the consumer induced by Medicare Part D, (2) the elasticity of demand for prescription drugs, and (3) the expenditures on branded prescription drugs, (4) the marginal cost of production, (5) expenditures under perfect competition.

Percentage Change in Price

We calculate the percentage price change that enrolling consumers would enjoy if they took up the program by computing — for each elderly consumer in MEPS — the difference in average coinsurance between: (1) status quo insurance, and (2) the most generous of the top 5 Part D insurance plans. We calculate this percentage change in price for each MEPS respondent estimated not to have creditable coverage. Respondents with creditable coverage are assumed not to enroll in Part D and thus experience no change in price. Based on these calculations the average percentage change in coinsurance for branded drugs for those without creditable coverage due to the standard Medicare Part D benefit was estimated to be 25%.

Price Elasticity of Demand

Long-run generic prices (assumed to be equal to marginal cost) are approximately 20% of the prices charged for the corresponding on-patent drug (Caves et al., 1991, Grabowski and Vernon, 1992, Berndt et al., 1996). Thus we assume that the monopoly mark-up over marginal cost is 400%. The standard theory of monopoly would then imply a price elasticity of uninsured demand around 1.25, or $\frac{1}{1-0.2}$. However, the above elasticity is not the relevant one for insured patients who face copayments, rather than manufacturer prices (Chandra et al., 2007). Thus, for the insured consumers we use elasticity estimates that rely on changes in patient cost sharing among the insured elderly population. For example, Chandra et al. (2007) estimate the price elasticity of prescription drugs among the elderly by studying a policy change that raised patient cost-sharing for retired public employees in California. Their estimates of price elasticity range from 0.5 to 1.5; these elasticities do not vary significantly by age, income, or health status. We take the midpoint of their range, and assume that the price elasticity is 1.0 among the insured elderly population. Based on these estimates of the elasticity of demand and percentage change in price, the average percentage change in number of prescriptions for those without creditable coverage was estimated to be 29%.

Out-of-Pocket Costs

The out-of-pocket costs of purchasing branded drugs are available directly from MEPS. For those without creditable coverage, expenditures are estimated to be $1,190.

Results

Based on these estimates, we estimate the aggregate static benefit of Medicare Part D to be $5.11 billion or $236 per eligible beneficiary. There is wide variation in the per capita benefit enjoyed by beneficiaries depending on insurance coverage, or lack thereof, prior to the

introduction of Medicare Part D. Since private insurance coverage is highly correlated with income, the poor enjoy greater benefits than the rich. For example, we estimate the per capita benefits to be $270 for those with incomes less than $15,000 per year, $207 for those with income between 15,000 and 50,000, and $222 for those with incomes greater than $50,000. Similarly, beneficiaries in poor self-reported health and those with higher prescription drug costs also enjoy greater benefits. Those reporting their general health to be "poor" enjoy per capita benefits of $394, those reporting "good" health enjoy per capita benefits of $247 and those reporting "excellent" health only receive $157 in per capita benefits.

Dynamic Benefits

Since Medicare Part D likely increases pharmaceutical company profits, it has the dynamic benefit of inducing additional innovation. We can estimate the value of this induced innovation just as we estimated the static value of the program.[35] First, we maintain the assumption (inherent in the original MMA legislation) that Part D continues to forbid price-negotiation, and that pharmaceutical firms will continue to receive the monopoly prices set before Medicare Part D (p_m). However, firms do experience an increase in demand for their products due to the reduction in price for consumers after the introduction of Medicare Part D.

Step 1: Change in Pharmaceutical Revenues

For a given consumer, the percentage change in total drug expenditures is equal to the percentage increase in the quantity of drugs consumed, $\frac{(\sigma_{ND} - \sigma_D)}{\sigma_{ND}} e$, which is calculated as above. Assuming manufacturer prices have so far been unaffected by Part D, the percentage change in quantity is equal to the percentage change in revenues for innovators. The average

[35] We are proceeding under the standard assumption that there is too little innovation, because innovators cannot capture full social surplus. In our sensitivity analysis, we discuss how to interpret our results if in fact there is over-innovation in the status quo.

percentage change in price for all eligible beneficiaries was estimated to be 25%. This estimate combined with the elasticity estimates, also discussed earlier, implies an increase in drug expenditures of $9 billion, or 6% of branded revenues.

Duggan and Scott-Morton (2008) find an increase of 33% for branded pharmaceutical revenues for drugs with 100% Medicare market share. Translated using MEPS estimates of the share of branded revenues from Medicare Part D recipients (19.7%), this would be a 6.5% increase in the revenue of branded drugs overall. Our estimate is thus quite close to, if slightly smaller than, the Duggan and Scott-Morton estimate. Lichtenberg and Sun (2007) find an increase in expenditures from Medicare part D of $9 billion (5.5% of branded drug revenues) using a differences in differences approach utilizing data from a retail pharmacy chain. Yin et al (2008) also find smaller effects on utilization than in our baseline, again using data from a pharmacy chain, they find a reduction in price of 17% (as compared with 25% in our estimation). Our estimation may be higher than these two estimates, because we assign enrollees to their best plan, thereby giving them the lowest possible price.

Step 2: Creation of New Chemical Entities

The increase in pharmaceutical revenue will induce more R&D and innovation. The number of new drug introductions induced by Part D will depend on the elasticity of new drug introductions with respect to pharmaceutical revenues. Acemoglu and Linn (2004) estimate that the elasticity of non-generic drug approvals with respect to revenues is roughly 3.5. We use this elasticity and the estimate of change in pharmaceutical revenues to calculate the percentage change in the number of new chemical entities (NCEs). This estimate is consistent with Blume-Kohout and Sood (2009) who estimate the effects of Medicare Part D on pharmaceutical R&D. The baseline rate of NCE introduction is assumed to be 32 NCEs per year. This is the average

number of NCEs introduced per year during the period 1995 to 2004 as reported in the FDA Orange Book. Applying the estimated percentage change to this baseline level yields the absolute number of new drugs projected from the passage of Part D.

Step 3: Innovator's Private Value of New Chemical Entities

The next step is to compute the annual private value of these additional drugs to their innovators. In general, it is quite difficult to compute the expected value of the *marginal* drug directly, because it is hard to identify the marginal drug and just as hard to identify *expected* value. However, it is easier to calculate the actual marginal cost of bringing an additional drug to market. Theory suggests that this marginal cost ought to be equal to the expected marginal private value of an additional drug; Grabowski et al (2002) provide empirical evidence that the theory is consistent with the data in the pharmaceutical industry. Di Masi et al. (2003) estimate that the marginal research and development cost of bringing an NCE to market is $939 million in 2005 dollars. To annualize this cost, we use a standard empirical estimate of the annual cost of capital in the pharmaceutical industry, of 12% per year, calculated from annual estimated costs of capital in the pharmaceutical industry (http://pages.stern.nyu.edu/~adamodar/). This estimated cost is similar to estimates of private rates of return on R&D investments in the pharmaceutical industry (Grabowski et al., 2002). Therefore, the annualized marginal cost of bringing an NCE to market is expected to be (0.12)*($939m) = $113 million. This then yields our estimate of the annualized marginal expected private benefit.

Step 4: Marginal Social Value of New Chemical Entities

The last step is to infer the marginal social value, which theory predicts will exceed the private value to the innovator (although see the caveat in footnote 35). To estimate the social return, we must estimate the fraction of social surplus captured by the innovator. Several

estimates are available from the literature. Based on data from 1948 to 2001, for example, Nordhaus (2004) estimates that innovators capture just 2.2% of the total present value of social returns to innovation. In a pharmaceutical context, Philipson and Jena (2006) use data from over 200 published studies of healthcare innovations to estimate the distribution of surplus-appropriation by innovators. They find that the median producer share of social surplus is 17%, the first quartile is roughly 10% and the third quartile is roughly 25%. To be conservative, we assume that innovators are able to capture as much as a quarter of the social surplus from pharmaceutical innovation. This parameter yields an estimated social rate of return on pharmaceutical R&D investments of 48% per year, four times the estimated private return. This suggests that the annual social value of the marginal drug is equal to ($113 million)/(0.25) = $452 million.

This approach presumes that innovators should capture the full social value of their inventions. As discussed in Section III, there exists a great deal of controversy about this issue. The impact of this assumption on our results is discussed in our sensitivity analysis, later in this section.

Gross Static and Dynamic Benefit of Part D

Using the methods described in Step 1 we estimate that Medicare Part D would increase pharmaceutical sales by $9 billion per year. Given baseline branded pharmaceutical sales of $162 billion in the U.S. in 2005 (as estimated in MEPS), this corresponds to a 6% increase. The 3.5 innovation elasticity from Acemoglu and Linn (2004) then implies that the number of new drugs per year would increase by roughly 21%, or 6.6 NCEs. Earlier, we calculated an annual social value of $452 million for the marginal additional drug, which yields a gross dynamic benefit of

$3.0 billion annually. Combining the dynamic with the static benefit yields a gross risk-neutral welfare benefit for Medicare Part D of $8.1 billion annually.

Deadweight Costs of Financing Medicare Part D

It remains to compare the aggregate benefits of Part D with its social cost. The program itself is just a costless transfer. However, since it is publicly financed, there are deadweight costs associated with its financing.

The actuarial cost of the Medicare Part D insurance for a beneficiary who enrolls in the standard Part D plan is simply:

$$s\left[Total\_Exp(1-\sigma_D)+(1-\sigma_D)Total\_Exp\frac{(\sigma_{ND}-\sigma_D)}{\sigma_{ND}}e\right] \qquad (11)$$

s is the subsidy provided by Medicare Part D and is estimated to be 75%. The first term in the square brackets is the actuarial cost of the benefit under the initial demand for pharmaceuticals and is simply the total cost of prescription drugs times the plan share of costs. The second term is the actuarial cost of the additional demand induced by Medicare Part D and is equal to the change in total drug costs times the plan share of costs. As discussed earlier, the percentage change in total drug costs, $\frac{(\sigma_{ND}-\sigma_D)}{\sigma_{ND}}e$, is simply the percentage change in price to consumers induced by Part D times the elasticity of demand. All the above quantities can be easily estimated from available data. Using data from the MEPS, we estimate average costs of covered drugs (both branded and generic) for MEPS respondents without creditable coverage who are likely to enroll in Medicare Part D to be $1,520. As discussed earlier, we can also calculate the price change consumers would enjoy if they took up the program; price-elasticities of demand for pharmaceuticals (taken from the literature) then imply the associated increase in

drug costs. Similarly, the costs of providing the employer subsidy (28% of costs between $250 and $5,000) can be estimated easily using data on prescription drug expenditures for those with creditable employer/union provided insurance. The results show that for those receiving the premium subsidy, Medicare costs are $1,109 per enrollee and for those receiving the employer subsidy, Medicare costs are $546 per enrollee.

The true social cost of the program is the deadweight cost associated with paying the actuarial cost out of public funds. While there is debate in the public finance literature on the magnitude of deadweight loss, we use a conventional estimate — that each additional dollar spent on Medicare Part D generates 30 cents of deadweight costs due to increased taxation (Jorgenson and Yun, 2001).

Based on these estimates, we estimate the deadweight costs of financing Medicare Part D to be $8.5 billion per year, 95% of which is covered by the risk-neutral benefits of the program. This analysis reveals that conventional estimates of demand, dynamic benefit, and deadweight loss yield the surprising result that Part D insurance is nearly "break-even" for a society of risk-neutral and self-interested consumers. The baseline estimates for static and dynamic benefits, as well as deadweight costs, are summarized in Table 2.

Sensitivity Analysis

We perform two sets of sensitivity analyses. Figure 1 shows the effect of changing the assumption on the elasticity of innovation. This assumption only has an effect on the dynamic benefit, not on static benefit or social cost. Increasing the elasticity of innovation proportionately increases the number of new drugs for a given change in revenues, and through this mechanism the dynamic benefit. The estimates show that even if we assume a very conservative elasticity of

innovation of 1, the benefits of Part D are sizeable and would cover 70% of the social costs of the program. However, note that if the optimal reward for innovation is lower than social surplus, increases in manufacturer revenues may even be welfare-reducing from a dynamic point of view. In this case, a program that holds manufacturer revenues fixed – for instance, through government price-negotiation -- while reducing consumer copays, would be optimal (Lakdawalla and Sood, 2009).

In Table 3 we perform a sensitivity analysis on the elasticity of demand for drugs for the insured. We estimate the social benefits and costs of the program using estimates of the demand elasticity for the insured from -0.2 to -1.5, a slightly wider range than estimated by range consistent with Goldman et al. (2007) and Chandra et al. (2007). Reducing the elasticity of demand reduces the effect of Medicare Part D's change on prices on the quantity of drugs consumed. For example, reducing the elasticity of demand from -1.25 to -0.2 reduces the percentage change in drugs from 31% to 21%. This change is smaller than one might expect because only the elasticity of insured users is altered, the elasticity for the uninsured remains at -1.25. This in turn reduces the static benefit, dynamic benefit (through lower revenues) and program costs. The overall benefit of the program is relatively stable under the alternative parameter estimates, with changes in program costs largely off-setting changes in static and dynamic benefits.

V. Policy Simulations

The discussion above takes a very conservative view of the welfare benefits associated with Part D. It is useful to consider some commonly proposed Part D reforms from this perspective, which emphasizes just static deadweight loss and dynamic incentives to innovate. We consider reductions in consumer cost-sharing requirements, as well as changes in the prices paid to

producers. The latter might occur if the government were to alter its stance on price-negotiation by the Part D program.

Changes in Consumer Prices

In Table 4 we consider a publicly financed reduction in copayment or coinsurance for Part D beneficiaries. We assume that the government mandates lower copayment or coinsurance rates, and finances 100% of the marginal cost associated with this reduction.

In this simulation, we reduce or increase the present formularies copays (or co-insurance depending on the formulary) by particular percentages. Thus, in the simulation where we change reduce copays by -50%, we would take a $5 copayment and make it $2.50. Similarly, if the co-insurance rate had previously been 25%, we make it 12.5% under this scenario. Then, using these adjusted formularies we assign to each person their optimal plan. Overall, reducing consumer prices yields higher static and dynamic benefits. Consider reducing copays by 50%., as compared with the baseline estimate, which in this case is a change of 0%. This reduction in consumer price entices an additional 3 million people (25 million versus 22 million) to enroll in Medicare Part D. Reducing copayment or coinsurance also results in a much larger average price change, of 40%, and a corresponding increase in the average change in quantity of 44%. Through increases in quantity and plan participation reducing the copayment by 50% increases the static benefit from $5.1 to 6.0 billion. The same mechanism also increases the change in pharmaceutical revenues (from $9.4 billion to $13.7 billion), resulting in a change in the dynamic benefit of $1.35 billion ($4.3 billion – $3.0 billion). However, this reduction in price is not free. The social cost of the program (from increased usage and lower prices) increases from a baseline of $8.5 to $11.2 billion under this scenario. The benefit cost ratio was 95% under the baseline scenario, and is only 93% when copays are reduced by 50%. Reductions in consumer

prices of this form generally decrease the net social return of the program as the increase in social costs of financing the program more than outweigh the increase in static and dynamic benefits. The reason is that reducing copays also increases the use of generics that do not add to the static or dynamic benefits of the program.

Changing Producer Prices

Finally, we simulate the effect of changing producer prices for branded drugs, and through this consumer prices (Table 5). As prices increase, Medicare Part-D becomes less attractive. A 25% increase in the price paid to manufacturers of drugs in Medicare Part D is associated with a reduction of 2 million enrollees (from 22 million to 20 million). Increasing prices paid to manufacturers also means that consumers pay higher prices when they are in the donut hole or when they face co-insurance. A 25% increase in price to the manufacturer implies that Medicare Part D only reduces prices for consumers by 18%, consequently increasing utilization by only 21%. This reduction in the quantity also changes the dynamic benefit. While there is a decrease in the quantity demanded, the price paid for these drugs increases, resulting in a small reduction in the dynamic benefit of $0.15 billion. Similarly, program costs remain largely unchanged (a change of $0.22 billion) as the increase in price is offset by reduced enrollment in the program. The net effect of increasing the producer price by 25% is a change in the benefit cost ratio from 95% to 85%. Similarly, were producer prices on brand name drugs to decrease by 25%, the benefit-cost ratio would increase from 95% to 111%. This suggests that some level of price negotiation on behalf of Part D may be welfare enhancing.

VI. Conclusion

The static benefits of Part D could be considerable. While controversy exists surrounding the relative efficiency of dynamic investments in pharmaceutical innovation; it remains possible that

the dynamic benefits are equally important. Regardless, our exercise demonstrates that the net social value of Part D extends well beyond the usual calculus of altruism and insurance that is typically associated with Medicare and other public health insurance schemes.

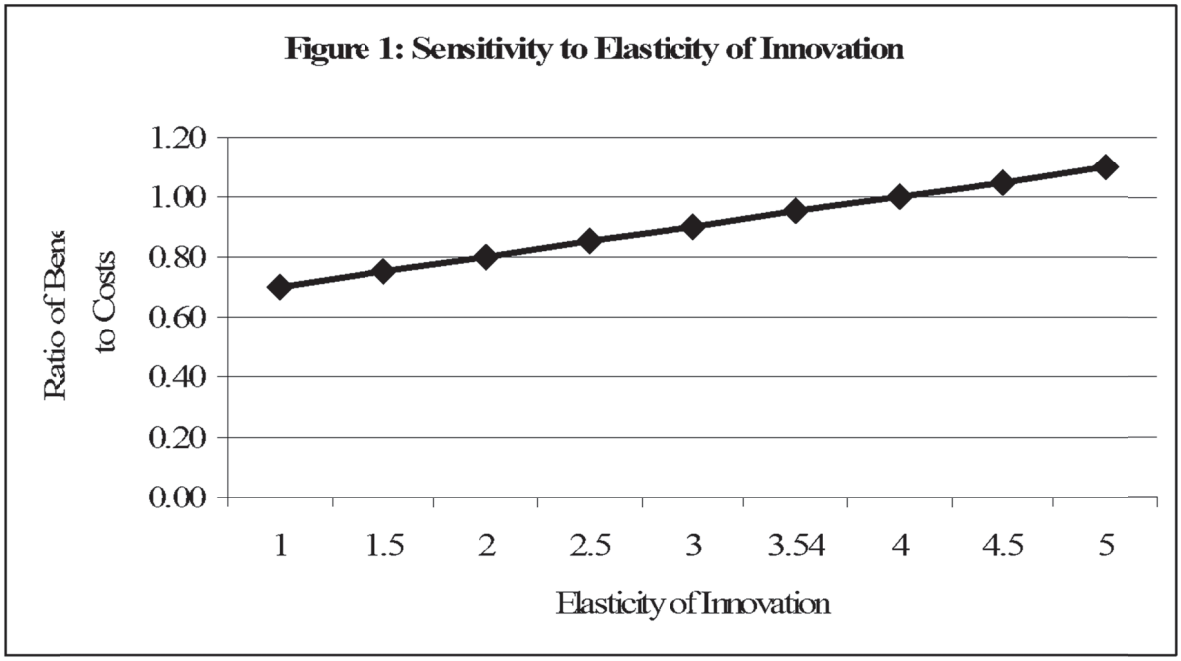
In the design of the benefit, a great deal of attention was paid to traditional "insurance" issues of adverse selection and moral hazard, but less effort was devoted to understanding the risk-neutral efficiency effects on utilization and innovation. Our analysis suggests that these might be quite important. In particular, any evaluation of alternate benefit design or price negotiations with manufacturers should take into account its effect on deadweight loss from monopoly and innovation. At a minimum, the economic case for Part D appears quite a bit broader and more complex than it may initially appear.

Table 1: Prescription Drug Coverage Sources Among Medicare Beneficiaries

	HHS Estimates		MEPS Estimates	
	Population (mil)	**Per cent**	**Population (mil)**	**Per cent**
Coverage Type				
Enrolled/No Creditable Coverage	21	59 %	22	61 %
Creditable Employer Coverage	10	28 %	8	21 %
Others with Creditable Coverage	5	13 %	6	18 %
Total	36	100 %	36	100 %

Table 2: Baseline Estimates of Benefits and Costs of Medicare Part D	
Description	**Value**
Plan Participation	
No Creditable Coverage (millions of people)	21.7
Creditable Employer Coverage (millions of people)	7.7
Other Creditable Coverage (millions of people)	6.4
Total (millions of people)	35.8
Static Benefit	
Change in Price for Enrollees (%)	25%
Change in Quantity for Enrollees (%)	29%
Branded OOP Expenditures – Enrollees Pre-Part D ($s)	958
Branded Drug Expenditures – Enrollees Pre-Part D ($s)	1190
Static Benefit Per Enrollee ($s)	236
Total Static benefit ($ billions)	5.11
Dynamic Benefit	
Change in Branded Drug Revenues ($ billions)	9.42
Change in Branded Drug Revenues (%)	5.81%
Change in Number of New Drugs (%)	21%
Number of New Drugs	6.58
Private Return per New Drug ($ millions)	113
Social Return per New Drug ($ millions)	451
Total Dynamic Benefit ($ billions)	2.97
Social Cost	
Average Covered Drug Expenditures for Enrollees Pre-Part D ($)	1520
Change in Quantity for Enrollees (%)	61%
Actuarial Cost of Part D Benefit ($ billions)	24.06
Costs of Employer Subsidy ($ billions)	4.19
Total Costs of Part D Insurance and Employer Subsidy ($ billions)	28.25
Dead Weight Costs of Part D Insurance and Employer Subsidy ($ billions)	8.48
Ratio of Social Benefit to Cost of Medicare Part D	0.95

Notes: Baseline estimate of the welfare benefits and costs of Medicare Part D using 2005 MEPS data.



Notes: Estimation of the sensitivity of baseline estimates of the welfare effects of Medicare Part D to changes in the elasticity of innovation using 2005 MEPS data. The elasticity of innovation has no effect on present consumption of drugs, and thus has no effect on the static benefit or social cost of the program. The elasticity of innovation changes the number of new drugs associated with a particular change in branded drug revenues and through this the dynamic benefit. 3.54 (from Acemoglu and Lin (2004)) represents our baseline scenario.

Table 3: Sensitivity to Demand Elasticity

Demand Elasticity	-0.20	-0.50	-0.75	-1.00	-1.25	-1.50
Plan Participation						
No Creditable Coverage (millions of people)	22	2	2	2	2	2
Static Benefit						
Change in Price for Enrollees (%)	25%	2 5%	2 5%	2 5%	2 5%	2 5%
Change in Quantity for Enrollees (%)	21%	2 4%	2 6%	2 9%	3 1%	3 3%
Branded OOP Expenditures – Enrollees Pre-Part D ($s)	95 8	9 58	9 58	9 58	9 58	9 58
Branded Drug Expenditures – Enrollees Pre-Part D ($s)	11 90	1 190	1 190	1 190	1 190	1 190
Static Benefit Per Enrollee ($s)	22 4	2 28	2 32	2 36	2 40	2 43
Total Static benefit ($ billions)	4.8 6	4. 95	5. 03	5. 11	5. 19	5. 27
Dynamic Benefit						
Change in Branded Drug Revenues ($ billions)	5.5 1	6. 98	8. 20	9. 42	1 0.64	1 1.85
Change in Branded Drug Revenues (%)	3.4 0%	4. 31%	5. 06%	5. 81%	6. 56%	7. 32%
Change in Number of New Drugs (%)	12 %	1 5%	1 8%	2 1%	2 3%	2 6%
Number of New Drugs	3.8 5	4. 88	5. 73	6. 58	7. 44	8. 29
Private Return per New Drug ($ millions)	11 3	1 13	1 13	1 13	1 13	1 13
Social Return per New Drug ($ millions)	45 1	4 51	4 51	4 51	4 51	4 51
Total Dynamic Benefit ($ billions)	1.7 4	2. 20	2. 58	2. 97	3. 35	3. 74
Social Cost						
Average Covered Drug Expenditures for Enrollees Pre-Part D ($)	15 20	1 520	1 520	1 520	1 520	1 520
Change in Quantity for Enrollees (%)	32%	4 3%	5 2%	6 1%	7 0%	7 9%
Actuarial Cost of Part D Benefit ($ billions)	19. 66	2 1.31	2 2.69	2 4.06	2 5.44	2 6.81
Costs of Employer Subsidy ($ billions)	4.1 9	4. 19	4. 19	4. 19	4. 19	4. 19
Total Costs of Part D Insurance and Employer Subsidy ($ billions)	23. 85	2 5.50	2 6.88	2 8.25	2 9.63	3 1.00
Dead Weight Costs of Part D Insurance and Employer Subsidy ($ billions)	7.1 6	7. 65	8. 06	8. 48	8. 89	9. 30
Ratio of Social Benefit to Cost of Medicare Part D	0.9 2	0. 93	0. 94	0. 95	0. 96	0. 97

Notes: Estimation of the sensitivity of baseline estimates of the welfare effects of Medicare Part D to changes in the elasticity of demand for the insured using 2005 MEPS data. The elasticity of demand effects the change in drugs consumed for the price change given in Medicare Part D. This change in consumption alters the static benefit, dynamic benefit, and social cost. -1.00 represents the baseline scenario.

Table 4: Simulation of Changes in Copayment or Coinsurance

Percentage Change in Copayment or Coinsurance	-75%	-0.50%	-0.25%	0%	25%	50%	75%
Plan Participation							
No Creditable Coverage (millions of people)	26	25	23	22	20	19	18
Creditable Employer Coverage (millions of people)	5	6	7	8	8	9	10
Other Creditable Coverage (millions of people)	5	6	6	6	7	7	8
Total (millions of people)	36	36	36	36	36	36	36
Static Benefit							
Change in Price for Enrollees (%)	51%	40%	32%	25%	19%	12%	6%
Change in Quantity for Enrollees (%)	55%	44%	36%	29%	23%	16%	9%
Branded OOP Expenditures – Enrollees Pre-Part D ($s)	878	90	927	95	97	99	10
Branded Drug Expenditures – Enrollees Pre-Part D ($s)	1219	12	1191	11	11	11	11
Static Benefit Per Enrollee ($s)	245	24	245	23	22	20	18
Total Static benefit ($ billions)	6.33	6.04	5.64	5.11	4.63	4.05	3.42
Dynamic Benefit							
Change in Branded Drug Revenues ($ billions)	16.73	13.71	11.34	9.42	8.09	6.69	5.38
Change in Branded Drug Revenues (%)	10.32%	8.46%	7.00%	5.81%	5.01%	4.13%	3.32%
Change in Number of New Drugs (%)	37%	30%	25%	21%	18%	15%	12%
Number of New Drugs	11.70	9.59	7.93	6.58	5.68	4.68	3.76
Private Return per New Drug ($ millions)	113	113	113	113	113	113	113
Social Return per New Drug ($ millions)	451	451	451	451	451	451	451
Total Dynamic Benefit ($ billions)	5.27	4.32	3.58	2.97	2.55	2.11	1.70
Social Cost							
Average Covered Drug Expenditures for Enrollees Pre-Part D ($)	1516	1517	1510	1520	1504	1507	1505
Change in Quantity for Enrollees (%)	74%	68%	64%	61%	58%	55%	53%
Actuarial Cost of Part D Benefit ($ billions)	39.56	33.68	28.26	24.06	20.33	17.34	14.80
Costs of Employer Subsidy ($ billions)	3.217	3.47	3.926	4.19	4.46	4.72	4.84
Total Costs of Part D Insurance and Employer Subsidy ($ billions)	42.77	37.15	32.18	28.25	24.79	22.06	19.63
Dead Weight Costs of Part D Insurance and Employer Subsidy ($ billions)	12.83	11.15	9.668	8.48	7.44	6.62	5.89
Ratio of Social Benefit to Cost of Medicare Part D	0.90	0.93	0.95	0.95	0.97	0.93	0.87

Notes: Estimation of the effect of changing co-copays on the welfare effects of Medicare Part D using 2005 MEPS data. Reducing the co-pays for prescription drugs makes Medicare Part D more attractive to more people, increasing enrollment. It also reduces the prices people pay, increasing the quantity of drugs demanded. These two effects lead to changes in the static benefit, dynamic benefit, and social cost of the program. 0% represents the baseline scenario.

Table 5: Changes to Producer Branded Drug Prices

Percent of Original Price	150%	125%	100%	75%	50%
Plan Participation					
No Creditable Coverage (millions of people)	18	20	22	24	26
Creditable Employer Coverage (millions of people)	10	9	8	6	5
Other Creditable Coverage (millions of people)	8	7	6	6	5
Total (millions of people)	36	36	36	36	36
Static Benefit					
Change in Price for Enrollees (%)	11%	18%	25%	34%	44%
Change in Quantity for Enrollees (%)	14%	21%	29%	38%	48%
Branded OOP Expenditures – Enrollees Pre-Part D ($s)	930	950	958	952	927
Branded Drug Expenditures – Enrollees Pre-Part D ($s)	1071	1134	1190	1269	1351
Static Benefit Per Enrollee ($s)	159	198	236	271	284
Total Static benefit ($ billions)	2.92	3.94	5.11	6.45	7.45
Dynamic Benefit					
Change in Branded Drug Revenues ($ billions)	8.58	8.95	9.42	11.13	14.64
Change in Branded Drug Revenues (%)	5.29%	5.52%	5.81%	6.87%	9.04%
Change in Number of New Drugs (%)	19%	20%	21%	24%	32%
Number of New Drugs	6.00	6.26	6.58	7.78	10.24
Private Return per New Drug ($ millions)	113	113	113	113	113
Social Return per New Drug ($ millions)	451	451	451	451	451
Total Dynamic Benefit ($ billions)	2.70	2.82	2.97	3.51	4.62
Social Cost					
Average Covered Drug Expenditures for Enrollees Pre-Part D ($)	1876	1713	1520	1309	1061
Change in Quantity for Enrollees (%)	55%	57%	61%	65%	70%
Actuarial Cost of Part D Benefit ($ billions)	19.58	21.83	24.06	26.41	27.45
Costs of Employer Subsidy ($ billions)	5.00	4.67	4.19	3.49	2.17
Total Costs of Part D Insurance and Employer Subsidy ($ billions)	24.57	26.50	28.25	29.91	29.62
Dead Weight Costs of Part D Insurance and Employer Subsidy ($ billions)	7.37	7.95	8.48	8.97	8.89
Net Social Benefit of Medicare Part D	0.76	0.85	0.95	1.11	1.36

Notes: Simulation of the effect of changing producer prices on the welfare effects of Medicare Part D using 2005 MEPS data. Under this scenario, there changing producer prices changes consumer prices, enrollment, and drug revenues and thus the static and dynamic benefits, but this also changes the social cost of the program. 100% represents the baseline scenario

Appendix A

Consider a health insurance plan that has a deductible of X dollars and offers a coinsurance of c for expenditures above the deductible. Consider an uninsured consumer with total expenditure E_1, where $E_1 > X$. Assume that the elasticity of demand is e.

The objective is to predict total expenditures, E_2, for this consumer when he or she enrolls in the health insurance plan with this non-linear benefit structure. First, note that the change in expenditures is the same as the change in quantity, because we assume prices paid to the manufacturer do not change. The consumer price does change, and it does so in a non-linear fashion, because it depends on the level of expenditures. Following the standard theory of demand, total expenditure when the consumer enrolls in this health insurance plan is given by:

$$E_2 = X + (E_1 - X) + (E_1 - X)(1 - c)e$$

The above equation shows that for the first X dollars in expenditures there is no change in demand as the consumer is below his or her deductible and therefore does not experience any change in price. For the next E_1-X dollars the consumer faces a percentage price reduction of 1-c. Therefore, the percentage change in demand or expenditures for this region is simply the percentage change in price times the elasticity of demand.

In the paper, we argue that the percentage change in coinsurance and elasticity of demand are sufficient predictors of the percentage change in utilization. In other words, we argue that the percentage change in total expenditures in this scenario can also be calculated by simply multiplying the percentage change in coinsurance times the elasticity of demand. Calculated this way, expenditures when the consumer enrolls in the health plan are given by:

$$E_2' = E_1 + E_1 e\left(\frac{1 - \bar{c}}{1}\right)$$

Where $\bar{c}$ is the average coinsurance when enrolled in the health plan?

$$\bar{c} = \frac{X + c(E_1 - X)}{E_1}$$

After substituting for $\bar{c}$ in the above equation, some simple algebra yields:

$$E_2' = X + (E_1 - X) + (E_1 - X)(1 - c)e$$

Hence, we get that $E_2' = E_2$.

A corollary of the above result is that the percentage change in coinsurance is a sufficient predictor of whether the consumer would benefit from insurance. A negative percentage change in average coinsurance or decrease in coinsurance implies an increase in use and thus an increase in static welfare.

References

Acemoglu, D. and Linn, J. (2004), "Market Size in Innovation: Theory and Evidence from the Pharmaceutical Industry," Quarterly Journal of Economics, Vol. 119 No. 3, pp. 1049-90.

Berndt, E. R., Cockburn, I. M. and Griliches, Z. (1996), "Pharmaceutical Innovations and Market Dynamics: Tracing Effects on Price Indexes for Antidepressant Drugs," Brookings Papers on Economic Activity, pp. 133-88.

Blume-Kohout, M. and Sood, N. (2009), "Medicare Part D and Pharmaceutical R&D," RAND Corporation Working Paper, Santa Monica, CA.

Caves, R. E., Whinston, M. D. and Hurwitz, M. A. (1991), "Patent Expiration, Entry, and Competition in the U S Pharmaceutical Industry," Brookings Papers on Economic Activity, pp. 1-48.

Chandra, A., Gruber, J. and McKnight, R. (2007), "Patient Cost-sharing, Hospitalization Offsets, and the Design of Optimal Health Insurance for the Elderly," National Bureau of Economic Research Working Paper 12972, Cambridge, MA.

DiMasi, J. A., Hansen, R. W. and Grabowski, H. G. (2003), "The Price of Innovation: New Estimates of Drug Development Costs," Journal of Health Economics, Vol. 22 No. 2, pp. 151-85.

Garber, A., Jones, C. and Romer, P. (2006), "Insurance and Incentives for Medical Innovation," Forum for Health Economics and Policy, Vol. 9 No. 2, pp. Article 4.

Grabowski, H., Vernon, J. and DiMasi, J. A. (2002), "Returns on Research and Development for 1990s New Drug Introductions," PharmacoEconomics, Vol. 20, pp. 11-29.

Grabowski, H. and Vernon, J. M. (1992), "Brand Loyalty, Entry and Price Competition in Pharmaceuticals After the 1984 Drug Act," Journal of Law and Economics, Vol. 35 No. 2, pp. 331-350.

Jorgenson, D. and Yun, K.-Y. (2001) Investment, Volume 3, Lifting the Burden: Tax Reform, the Cost of Capital, and U. S. Economic Growth, MIT Press, Cambridge, MA.

Kaiser Family Foundation (2007), "The Medicare Prescription Drug Benefit," Kaiser Family Foundation Report 7044-06, Menlo Park, CA.

Kaiser Family Foundation (2008a), "Fact Sheet: The Medicare Prescription Drug Benefit," Henry J. Kaiser Family Foundation Publication 7044-08, Menlo Park, CA.

Kaiser Family Foundation (2008b), "The Medicare Part D Coverage Gap: Costs and Consequences in 2007," Kaiser Family Foundation, Menlo Park, CA.

Lakdawalla, D. and Sood, N. (2006), "Health Insurance as a Two-Part Pricing Contract," National Bureau of Economic Research Working Paper 12681, Cambridge, MA.

Lakdawalla, D. N. and Sood, N. (2009), "Innovation and the Welfare Effects of Public Drug Insurance," Journal of Public Economics, Vol. 93, pp. 541-548.

Lichtenberg, F. R. and Sun, S. X. (2007), "The impact of Medicare Part D on prescription drug use by the elderly," Health Affairs, Vol. 26 No. 6, pp. 1735-1744.

Nordhaus, W. D. (2004), "Schumpeterian Profits in the American Economy: Theory and Measurement," National Bureau of Economic Research Working Paper 10433, Cambridge, MA.

Philipson, T. J. and Jena, A. B. (2006), "Surplus Appropriation from R&D and Health Care Technology Assessment Procedures," National Bureau of Economic Research Working Paper 12016, Cambridge, MA.

Shapiro, C. (2007) Patent Reform: Aligning Reward and Contribution. *National Bureau of Economic Research, Inc, NBER Working Papers.*

Yin, W., Basu, A., Zhang, J. X., Rabbani, A., Meltzer, D. O. and Alexander, G. C. (2008), "The Effect of the Medicare Part D Prescription Benefit on Drug Utilization and Expenditures," Ann Intern Med*,* Vol. 148 No. 3**,** pp. 169-177.