The Costs and Benefits of Caring: Aggregate Burdens of an Aging Population

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Abstract

As the population of the United States ages, the number of elderly people who require living assistance is increasing. To understand how this impacts aggregate output, we calibrate an OLG model where growth endogenously depends on the care young agents choose to provide for their parents. Relative to an economy with a constant population distribution, we project that population aging will reduce GDP 15% by 2057 and 35% by 2097. Exogenous reductions in the incidence of diseases such as Alzheimer's and dementia can lead to 1.3% higher output relative to the baseline. In an economy where hypothetical drugs to treat these diseases are supplied by a competitive market as opposed to a patent-holding monopolist, lifetime welfare is between 2% and 4% higher.

Keywords: aging, growth, labor supply, altruism, idiosyncratic risk
JEL Classification: D15, J14, J22, O40

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1 Introduction

For the United States and other developed countries, population aging will increase the absolute number of individuals requiring some form of elder care.[1] Microeconomic evidence suggests that caring for infirm older adults requires substantial resources, both in terms of market-traded services and the off-market time of family members. As an example, the Alzheimer's Association estimates that caring for individuals diagnosed with Alzheimer's and dementia is almost triple the cost of caring for non-diagnosed individuals. While approximately 70% of these costs of care are covered by state and federal social insurance programs, Hurd et al. (2013) estimates that the time-value of informal care provided by family members in 2011 amounted to between $50 billion and $106 billion. Further, no known cures of treatments exist for diseases like Alzheimer's and dementia despite private and public investment in research and development for treatments. This paper features two main findings. First, we show that the ballooning number of elderly people requiring living assistance will have a modest impact on aggregate economic growth independent of the substantial impacts imposed by aging itself. Second, we find that an exogenous reduction in incidence of high cost-of-care old-age diseases can improve welfare for both diseased and healthy agents in a general equilibrium environment. When endogenously accounting for the resources required to make such a reduction, expected lifetime welfare is reduced by 2.1% due to patent laws that give drug-developing firms monopolistic pricing power for period of time.

Recent empirical evidence suggests that many working-age adults spend substantial shares of their available time providing informal care for sick and diseased elders with the average working-age household which engages in care for an infirm adult spending 5.22 hours per week doing so. Such households supply marginally less labor (approximately 11.28 minutes less per week), but enjoy 3.76 hours less leisure time.[2] The National Institutes of Health (NIH), the World Health Organization (WHO), and others have warned that the costs of providing assisted-living care for older adults could balloon as the population ages, suggesting that aggregate economic outcomes will be adversely affected by this phenomenon.[3]

[1]We use the terms "elder care," "informal care," and "assisted-living care" synonymously to refer to any kind of assistance received by diseased elderly individuals to perform day-to-day life functions.

[2]All time estimates are population weighted averages over the period 2003-2017 taken from the American Time Use Survey, from here on ATUS (Bureau of Labor Statistics 2017).

[3]See U.S. studies on the implications of aging from the National Institute on Aging (2011), National Research Council (2001), and Knickman and Snell (2002). Also, for costs associated with caring for elderly individuals with dementia and Alzheimer's, see Alzheimer's Association (2011), Hurd et al. (2013), and Lepore, Ferrell, and Wiener (2017).

The Alzheimer's Association rather starkly estimates the cumulative nominal cost of caring for patients with Alzheimer's and other dementias of $20.2 trillion from 2018 to 2050, two-thirds of which will be borne by Medicare and Medicaid. NIH invested $414 million in Alzheimer's research in 2018, though for every $9,700 Medicare and Medicaid spend caring for patients, the NIH only spends $100 on cure-related research (Alzheimer's Association 2019). Private firms invest substantially in research and development (R & D) to achieve a cure, anticipating huge profits from future patent rights if R & D proves successful. Yet, finding successful treatments that reduce the risk of contracting a welfare-debilitating disease like Alzheimer's or dementia has proven illusive, with biotech startups spending billions on failed endeavors (LaVito and Lovelace 2019). In the spirit of these recent events, one version of our model features an endogenous market for disease treatment which affects consumers' ex-ante risk of contracting a welfare-debilitating disease.

Our main findings confirm previous studies that showed population aging in general has a large, negative impact on aggregate output growth rates. In our baseline calculation, holding constant the risk rate of acquiring a debilitating, welfare-reducing disease (thus, assuming no treatment is found), we project average annual U.S. GDP growth to be 2.21% over the period 2017-2057 and 2.02% over the period 2017-2097. Eliminating the risk of needing long-term assisted-living care marginally increases projected future average annual growth rates for the United States economy over the period 2017-2057 by 5 basis points relative to the baseline. As in Hubbard, Skinner, and Zeldes (1995) and Prescott (2004) social insurance programs in a pay-as-you-go (PAYGO) structure crowd out investment, reducing long-run growth rates relative to a tax-free environment. In the presence of intergenerational transfers of off-market time from young to old, lifetime welfare increases when social insurance tax rates fall as savings and investment increase. Young agents expect to enjoy being cared for by their offspring when old and plan for this spillover effect when choosing savings. This is because endogenous time transfers from young to old of informal care can help offset the adverse welfare implications of incomplete markets for insurance against old-age welfare shocks. Yet, while reducing social insurance taxes may increase expected lifetime utility, a reduction is not necessarily Pareto improving if the working-age share of adults is low. This is because old agents afflicted with a welfare-reducing disease are made worse off as taxes fall and the number of workers is small enough. Reimbursing young agents at the market rate for the time they supply caring for diseased-elders provides a marginal increase in growth rates of 2 basis points by 2097, and leaves welfare relatively unaffected, suggesting such policies, already being implemented by Medicaid in several states (see Mommaerts (2016, 2017)) may be growth and welfare neutral. In various counterfactual simulations we explore the implications of all of these trends and various policies under different population growth rates and different adverse shock probabilities.

The paper proceeds as follows. In Section 2 we discuss the population trends and cost estimates associated with the prevalence of high cost-of-care old-age diseases, while also summarizing available data on the allocation of time to care for infirm elders. In Section 3 we outline an OLG model that captures the features discussed in Section 2. In Section 4, we calibrate this model to match observed data points. In Section 5 we simulate counterfactuals to understand how population changes affect long-run economic trends. In Section 6 we conclude.

2 Background & Discussion

The primary motivation for our undertaking is to understand how population aging affects aggregate economic outcomes when members face ex-post idiosyncratic risk to old-age welfare. While the effects of population aging have been discussed in many contexts, few studies have analyzed general equilibrium outcomes when young people save to insure against idiosyncratic risk that directly impacts old-age consumption utility.[4] The closest study that comes to mind is that of Hall and Jones (2007) who model health risk as endogenously affecting survival rates, along with a health status component in utility.[5] To the best of our knowledge nobody has attempted to place idiosyncratic endogenous health risk into a model where young agents provide informal hospice care to ailing loved ones. Our undertaking thus contextualizes diseases like dementia, including Alzheimer's, and other idiosyncratic old-age welfare shocks within an economic framework that features long-term informal, assisted-living care.

There have been no studies, to our knowledge, that estimate the impacts of providing informal care off-market on general equilibrium economic outcomes. This is important because an aging population will likely lead to higher levels of informal care being provided by young people to old people.[6] Several studies have examined how provisions of informal care impact individual labor force participation and earnings (Muurinen 1986; Carmichael, Charles, and Hulme; Leigh 2010; Van Houtven, Coe, and Skira 2013). In-

[4] We are aware of French and Jones (2011), DeNardi, French, and Jones (2010), Edwards (2008), and Palumbo (1999) who look at financial planning decisions within retirement in a partial equilibrium context.

[5] An unpublished study by Azomahou, Diene, and Soete (2009) models health risk as a shock to a health capital stock, as opposed to a direct change in the utility function, which is our approach.

[6] For our purposes, "informal care" encompasses all aspects of care which take place off market. "Formal care" will be used to refer to care paid for on the marketplace. These definitions are consistent with those in Hurd et al. (2013) and Lepore, Ferrell, and Wiener (2017).

formal caregivers who also participate in the formal labor force work on average 3 to 10 hours less per week than their non-caregiving peers (Van Houtven, Coe, and Skira 2013). Providing informal care can thus lead to considerable earnings losses (Muurinen 1986; Van Houtven, Coe, and Skira 2013). Recent work suggests that substitution rates between formal nursing home care and informal in-home care in the United States depend on individual states' complex Medicaid reimbursement structures (Mommaerts 2016, 2017). Indeed, paid long-term care and unpaid in-home care are imperfect substitutes (Mommaerts 2017). We conjecture that this imperfection is due to trade-offs faced by younger family members who willingly provide informal care to elders. Since providing off-market care requires a time investment, younger family members must weigh the altruistic benefits they receive from caring for older loved ones against the loss in lifetime permanent income due to working less.

Until recently there have been few aggregate data available on the rate at which informal elder care is supplied. From 2003-2017 ATUS asked respondents how much time they spent caring for or helping adults, not just the elderly, who require assistance. Weighted averages of time use for adults age 25-65, where our primary target variable is "adult care", are presented in Table 1.[7][8] At first glance, the time-use data suggest that the impact of increasing disease prevalence on the intensive margin of labor supply is significant in magnitude as the population ages. Consider now the effects of such a change: working less results in a reduction in permanent income, resulting in a reduction in investment, resulting in a reduction in aggregate output and social insurance tax receipts. However, our results in Section 5 show that young individuals adjust their time use in response to market conditions, including the population distribution, mitigating the aggregate impacts of this disease risk. In fact, changes in the population distribution alone appear to affect the labor supply greatest along the extensive margin. In steady state simulations, we show that young workers increase work time as the relative population of working-age adults to retirees falls, but this increase on the intensive margin does not offset the negative impacts on total labor supply due to a falling extensive margin.

[7] We take the denominator in our weekly time-share calculations to be 224 hours for two-person households and 112 hours for single-person households. This allows individuals 8 hours of non-allocatable personal time per day.

[8] We choose to use the total "adult care" data point rather than the "elder care" data point available in the ATUS only from 2011-2017 due to the small sample size of the latter. Empirical tests show that the differences in weighted averages of both data points are not significantly different from zero. More details are available upon request.

Table 1: Time Allocation of Adults $25-65$, (ATUS: 2003-2017)

Total Households, $N = 86337$			
Avg. Total Time	188.938		
Avg. Adults in Household	1.687		
	Leisure	Labor	Adult Care
Avg. Hrs. per Week	125.317	63.004	0.618
Share of Avg. Total Time*	0.663	0.333	0.003
Provide Positive Off-Market Adult Care, $N = 10330$			
Avg. Total Time	190.064		
Avg. Adults in Household	1.697		
	Leisure	Labor	Adult Care
Avg. Hrs. per Week	122.005	62.838	5.222
Share of Avg. Total Time*	0.642	0.331	0.027
Provide No Off-Market Adult Care, $N = 76007$			
Avg. Total Time	188.787		
Avg. Adults in Household	1.686		
	Leisure	Labor	Adult Care
Avg. Hrs. per Week	125.761	63.026	0
Share of Avg. Total Time*	0.666	0.334	0

* Weighted average shares of time per household.
2-person households face total time per week of $7 * 2 * (24 - 8) = 224$ hours.
Single person households face total time per week of $7 * (24 - 8) = 112$ hours.

To understand more broadly how long-run declines in aggregate output are related to population aging, in Figure 1 we plot the working-age population ratio ($wapr$), i.e. the ratio of adults age 25-65 to adults age 65 and over, along with the HP-filtered trend of year-on-year aggregate and per-worker GDP growth (g_Y and g_{Y/N_y}, respectively) for the United States economy since 1950.[9] When business cycles are removed, the long-run decline in annual GDP growth appears remarkably correlated with the decline in the working-age population ratio. A regression of $\ln g_{Y,t}$ on $\ln wapr_t$ reveals that the elasticity of the filtered trend in output growth with respect to the working-age population ratio is 1.372, so that a 1% relative increase in workers leads to an approximate 14 basis point increase in the growth rate. Falling $wapr$ accounts for almost 50% of the decline in g_Y since the 1950s.[10] The magnitude of this correlation affirms some of the alarm bells sounded recently in Cooley and Henriksen (2018).

As a motivating example, Alzheimer's disease and dementia impose substantial formal costs on the United States' social insurance system and informal costs on family members tasked with caring for diseased individuals. Total cost estimates for caring for demented elderly individuals range from \$157 to \$215 billion (2010 dollars) depending on the method used to impute time value of informal care (Hurd et al. 2013). Within this range, roughly \$11 billion is covered by Medicare, Medicaid, and Social Security, while the remainder includes both out-of-pocket costs paid by afflicted individuals and their families as well as the time value of unpaid, informal care provided by loved-ones (Hurd et al. 2013). Estimates of total time devoted to informal care for demented persons are not small in magnitude. The Alzheimer's Association estimates that in 2010, 17 billion hours of unpaid care were provided by loved ones to diseased elders, with over 80% of this time burden born by family members. Further, over 90% of those afflicted with Alzheimer's or dementia receive some form of informal care on top of care provided by professional hospice services. The spillover effects on working-age adults of shouldering this burden represent an additional societal cost, the impacts of which have not been directly quantified in past studies. As the population ages and Alzheimer's and dementia prevalences increase, it is reasonable to expect that the quantity of informal care provided by working-age adults to elderly adults will increase.

Our work fits into a broader economic conversation about the role of precautionary

[9] In the HP filter we set the smoothing parameter to $\lambda = 400$ as recommended by Cooley and Ohanian (1991) and Correia, Neves, and Rebelo (1992). Though Ravn and Uhlig recommend a value of $\lambda = 6.25$, the purpose of smoothing here is solely to illustrate pictorially that growth has declined in conjunction with $wapr$, thus we choose the largest possible smoothing value recommended by the literature. For more information on the HP filter see Hodrick and Prescott (1997).

[10] Details of the regression from which these elasticity estimates are derived can be found in Appendix A.1.

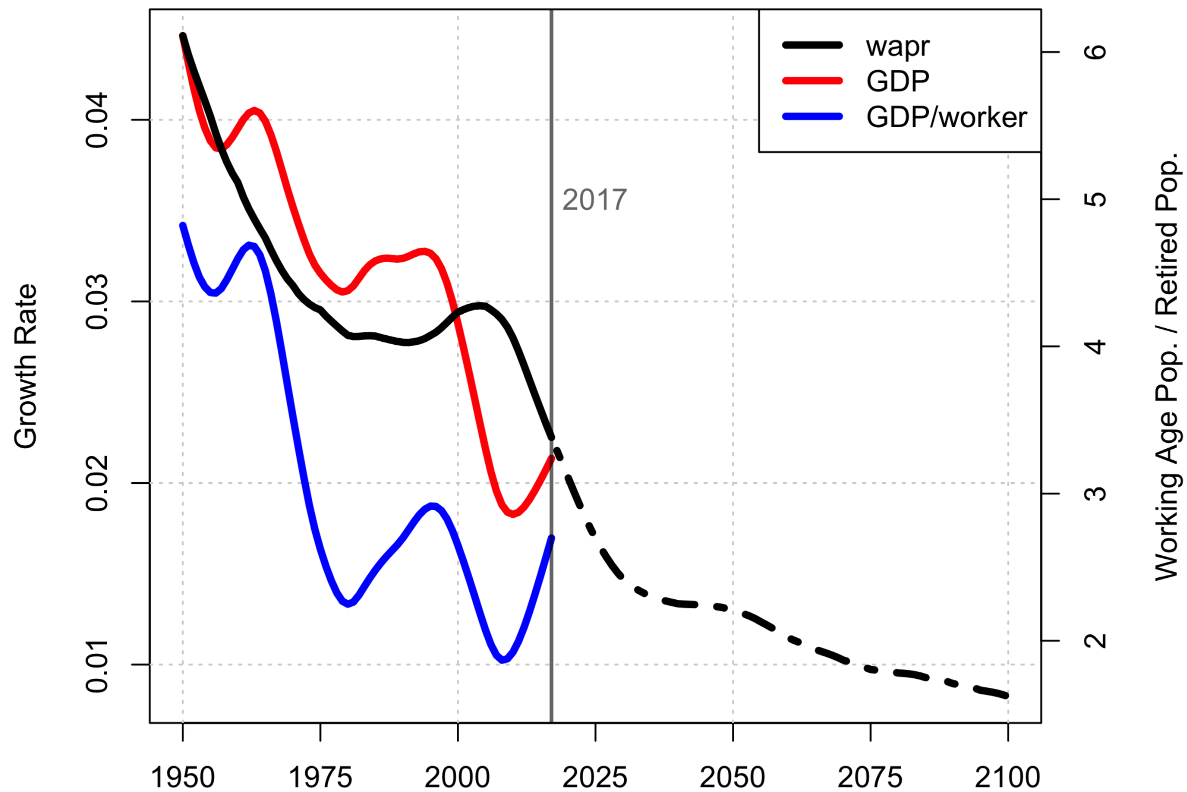

Figure 1: We plot HP-filtered year-on-year net growth: $Y_t/Y_{t-1} - 1$ for the aggregate case or $(Y_t/N_{yt})/(Y_{t-1}/N_{y,t-1}) - 1$ for the per-worker case. $wapr_t$ is unfiltered and downloaded from the United Nations: Department of Economic and Social Affairs (2017). The elasticities of filtered aggregate and per-worker growth with respect to $wapr_t$ are 1.372 and 2.269 respectively (see Appendix . If trends continue, we should expect growth rates to decline throughout the 21st century.

savings motives. Particularly, we add to the literature that argues individuals save to insure against illness in old age (see French and Jones (2011), DeNardi, French, and Jones (2010), and Palumbo (1999)) by endogenizing the decision of young individuals actively to care for their elders, incorporating new dynamics into consumption smoothing. Young households are subsidizing older households both directly thru labor taxes and indirectly by expending time to care for them. Thus, more broadly, modeling intergenerational transfers from young to old in this manner, coupled with the perceived guarantee provided by social insurance, may help account for the puzzle of why so many individuals save so little for retirement.[11] Further, there exists a vein of literature examining European demographic trends that shows that the structure of public pension systems can have

[11]See, for example, Benartzi and Thaler (2013) and Hubbard, Skinner, and Zeldes (1995, 1994).

an impact on the rates of intergenerational transfers from young to old and vice-versa (Deindl and Brandt 2011; Bonsang 2007; Attanasio and Burgiavini 2003). Designing social insurance systems to better acommodate both future population aging and increases in old-age disease prevalence, especially Alzheimer's and dementia, could help offset potential welfare loss to future generations due to this phenomenon.

3 Model

3.1 Households

Agents live a maximum of two periods, though they may die accidentally after their first period of life.[12] Each period, there exists a population of $N_{y,t}$ young households and $N_{o,t}$ old households.[13] There is only one type of young household and two types of old households. Old households can be either diseased, $d_t = 1$, or non-diseased, $d_t = 0$. For now, assume the population of young households grows at constant rate g_N so that $N_{y,t}/N_{y,t-1} = (1 + g_N)$, and the probability that a young household in period t lives to be an old household in period $t + 1$ is $s_{o,t+1}$.[14] [15]

We model the old agents' consumption processes in terms of home production, taking cues from Becker (1965). Young agents can subsidize the home production of diseased old agents' final consumption by supplying them care time $h_{y,t}$ outside of formal markets. Diseased individuals thus receive flow utility from final consumption $c_{o,t}(d_t = 1)$, which is produced in the home by using inputs of this off-market care time they receive from their children $h_{o,t}$ and market resources purchased $x_{o,t}(d_t = 1)$.[16] Meanwhile, their healthy peers only use market resources $x_{o,t}(d_t = 0)$ for production of final consumption because they do not require additional off-market care time from their children. The home production functions we employ for both diseased and non-diseased old are

$$c_{o,t}\big(x_{o,t}(d_t = 1), h_{o,t}, d_t = 1\big) = x_{o,t}(1)^{1-\sigma} h_{o,t}^{\sigma} \quad \sigma \in (0,1) \tag{1}$$

$$c_{o,t}\big(x_{o,t}(d_t = 0), d_t = 0\big) = x_{o,t}(0) \tag{2}$$

In Equation (1), σ is the elasticity of final consumption with respect to informal care time

[12]Young households cannot choose to die, nor can individuals, rather the household is thought to "disappear," in that all of its members have perished before becoming old.

[13]Since agents live only two periods, we use y and o subscripts to denote their ages.

[14]We will relax the constant growth, g_N, assumption in some of our simulations.

[15]The "survival" probability is the probability a young household that enters the economy survives to be an old household next period.

[16]We also refer to h_{ot} as "hospice" care.

received. Note that both diseased and non-diseased individuals may purchase hospice care or other health services on the formal market. Such a purchase would fall under market consumption, $x_{o,t}(d_t)$. Any additional services received by diseased old that are not accounted for on the formal market would fall under informal care-time received, $h_{o,t}$.

Old households have preferences over consumption that depend on health status d_t. The form of an old individuals' utility function is chosen to satisfy several conditions. First, we assume that individuals infected with a disease require more resources, both market and off-market, to care for than those who are not. It would be unreasonable to assume that these individuals, by consuming more, are necessarily better off than their non-diseased peers (after all, they are sick). Let $u_{o,t}\big(c_{o,t}(d_t), d_t\big)$ denote the flow utility from final consumption for an old individual with disease status d_t. This brings us to an assumption about an old individual's utility function.

Assumption 1. Suppose $c_{o,t}(1) = c_{o,t}(0) = c$, where c is any feasible level of final consumption. Then $u_{o,t}(c, 1) < u_{o,t}(c, 0)$. In words, for each level of final consumption, the non-diseased agent always receives higher consumption utility than the diseased agent.

Assumption 1 ensures that it is always better to be non-diseased than diseased. We choose a Stone-Geary flow utility function for diseased old which satisfies this assumption under certain parameter restrictions:

$$u_{o,t}\big(c_{o,t}(1), d_t = 1\big) = \ln\big(c_{o,t}(1) - \nu\big) \tag{3}$$

$$u_{o,t}\big(c_{o,t}(0), d_t = 0\big) = \ln c_{o,t}(0) \tag{4}$$

The flow utility parameterizations in (3) and (4) lead to two basic lemmas.

Lemma 1. For all $\nu > 0$, Assumption 1 holds.

Proof. See Appendix B □

Lemma 2. If $0 < \nu < c_{o,t}(1) - c_{o,t}(0)$ then the ratio of the marginal utility of non-diseased consumption to diseased consumption is such that $MU_{o,t}(0)/MU_{o,t}(1) > 1$.

Proof. See Appendix B □

Lemma 1 is trivial. Lemma 2 says that for certain combinations of the subsistence parameter ν and consumption policies, non-diseased agents benefit more from additional final consumption than diseased agents. In our calibration we find that the premise of Lemma 2 holds, which is one indicator that in this economy resources are inefficiently allocated in a steady state competitive equilibrium.

Young households use market resources $x_{y,t}$ and leisure time $l_{y,t}$ to produce a final consumption good c_{yt} according to the home production function:

$$c_{y,t} = x_{y,t}^{\gamma} \cdot l_{y,t}^{\mu} \tag{5}$$

Young households additionally supply off-market time $h_{y,t}$ to care for their elders, but since this does not affect the final production of their home-produced consumption good, $h_{y,t}$ does not enter into Equation (5). Rather, young households exhibit imperfect altruism toward their sick elders, discounting the diseased old household's utility at rate η. The flow utility of young households is

$$u_{y,t}(c_{y,t}, h_{y,t}) = \ln c_{y,t} + \eta \cdot \ln \left(c_{o,t}(h_{y,t}, d_t = 1) - \nu \right) \tag{6}$$

Additionally, young agents may purchase treatments or drugs $dr_{y,t}$ that help offset the risk they will be afflicted with a degenerative disease when old. Let $\psi_t(dr_{y,t-1})$ denote the share of old population which is afflicted with a welfare-reducing disease in period t. This fraction is a function of the level of treatments purchased by the current old generation in the previous period when they were young. We choose an inverse logit specification for the probability, conditional upon surviving, that a young agent in period t becomes a diseased old agent in period $t+1$:

$$\psi_{t+1}(dr_{y,t}) = \frac{1}{1 + \overline{\psi} \epsilon^{-dr_{y,t}}} \tag{7}$$

Young agents derive no direct, period t flow utility from consuming potential treatments. Rather, such treatments affect their expected future utility, which we will illustrate in full detail below. Note that $dr_{y,t}$ can be zero, in which case $\psi_{t+1} = \frac{1}{1+\overline{\psi}}$. This happens when no such cures have yet been produced, which is the case at the time this paper was written, In any given period, the total population of diseased old receiving care time from their children is then $N_{o,t} \cdot \psi_t(dr_{y,t-1})$. The supply of hospice care by young equals the total amount of hospice care received by diseased old

$$h_{y,t} = \frac{N_{o,t} \cdot \psi_t(dr_{y,t-1}) \cdot h_{o,t}}{N_{y,t}} \tag{8}$$

In addition to consuming treatments $dr_{y,t}$, other market goods $x_{y,t}$, and spending time caring for their parents, young agents supply both labor $1 - l_{y,t} - h_{y,t}$ and invest $i_{y,t}$ in

[17] Total available time is normalized to 1.

the market.[17] Young agents earn a before-tax wage rate w_t and pay social insurance taxes on their labor income at rate τ_t.

Old agents do not work but earn a gross return on their assets $a_{y,t}$ at rate R_t and also receive Social Security and Medicare transfer benefits from the government $T_t(d_t)$ which depend on disease status d_t. Normalize the price of non-treatment market purchases to 1 each period. For old agents, net outlay must satisfy the budget constraint:

$$x_{o,t}(d_t) \leq R_t \cdot a_{y,t} + T_t(d_t) \tag{9}$$

Old agents die with certainty at the end of their life and will choose to consume the entirety of their available cash-on-hand. At the end of each period, young agents who accidentally and unexpectedly die leave behind total net assets (capital) equivalent to $a_{y,t+1} \cdot (1 - s_{o,t+1}) \cdot N_{y,t}$. These assets are then distributed evenly and unexpectedly (i.e., "accidentally") as bequests $b_{y,t+1}$ to young agents entering the economy next period according to

$$b_{y,t+1} = a_{y,t+1} \cdot (1 - s_{o,t+1}) \cdot \frac{N_{y,t}}{N_{y,t+1}} \tag{10}$$

Since returns on investment are compounded at the beginning of the period, young agents earn gross return on these assets $R_t \cdot b_{y,t}$. Having fully-described the right-hand side of a young agent's budget constraint, their choices of market purchases $x_{y,t}$, drugs $dr_{y,t}$, asset-holdings $a_{y,t+1}$, and labor-supply must satisfy

$$x_{y,t} + p_t \cdot dr_{y,t} + a_{y,t+1} \leq R_t \cdot b_{y,t} + w_t \cdot (1 - \tau_t) \cdot (1 - l_{y,t} - h_{y,t}) \tag{11}$$

p_t is the period t price of drugs expressed in units of period t non-drug market purchases $x_{y,t}$. If $dr_{y,t} = 0$ due to no existing supply, then $p_t = \infty$, but we assume $p_t \cdot dr_{y,t} = 0$ to ensure that all markets clear. Naturally, the lack of supply for a cure means that households will not spend any resources purchasing a cure.

We assume young agents know the survival rate and how it evolves. However, young agents do not know whether they will survive to become old and if they do whether they will face a disease that adversely impacts their welfare. Thus, they make their investment choice both with the aim of smoothing consumption and imperfectly insuring themselves against the adverse welfare effects of contracting some kind of disease such as Alzheimer's or dementia, for example. In this model, given young agents in period t know the distribution of diseased agents in $t + 1$, expectations are perfectly rational. Let β be the time discount factor. In competitive equilibrium, utility maximizing young agents seek to smooth expected market consumption over the lifecycle according to the expected intertemporal Euler equation:

$$\begin{aligned}\underbrace{\frac{\gamma}{x_{y,t}}}_{MU_y(x)} &= \beta \cdot s_{o,t+1} \cdot R_{t+1} \Bigg[\psi_{t+1}(dr_{y,t}) \underbrace{\frac{1-\sigma}{x_{o,t+1}(1)^{1-\sigma} h_{o,t+1}^{\sigma} - \nu} \left(\frac{h_{o,t+1}}{x_{o,t+1}(1)} \right)^{\sigma}}_{MU_o(x(1))} \\ &\qquad + (1-\psi_{t+1}(dr_{y,t})) \underbrace{\frac{1}{x_{o,t+1}(0)}}_{MU_o(x(0))} \Bigg] \end{aligned} \tag{12}$$

Since the model contains only idiosyncratic uncertainty, R_{t+1} is pre-determined by the population distribution which is assumed known. Thus young agents choose investment by equating the marginal utility of present market purchases with the discounted expected marginal utility of future consumption given by weighting diseased and non-diseased marginal utilities by the endogenous probability mass $\psi_{t+1}(dr_{y,t})$. The period t choice of labor supply by young depends on the marginal rate of substitution between consumption and leisure and the marginal rate of substitution between leisure and off-market care time:

$$\frac{\mu}{l_{y,t}} = \frac{\gamma}{x_{y,t}} w_t(1-\tau_t) \tag{13}$$

$$\frac{\mu}{l_{y,t}} = \eta \cdot \frac{\sigma}{x_{o,t}(1)^{1-\sigma} h_{o,t}^{\sigma} - \nu} \left(\frac{h_{o,t}}{x_{o,t}(1)} \right)^{\sigma-1} \frac{N_{y,t}}{N_{o,t}\psi_t(dr_{y,t-1})} \tag{14}$$

Finally, if a market for effective treatments exists, then young agents choose $dr_{y,t}$ so as to equate the marginal utility of market consumption with the present, perceived marginal benefit of the treatment. Notice though that $dr_{y,t}$ enters into next period's probability of disease contraction. Thus, young agents receive no tangible, present benefits to consuming $dr_{y,t}$. Instead, the presence of a market for $dr_{y,t}$ provides young agents with additional insurance against the adverse impacts of a welfare debilitating disease, on top of the insurance they will receive via their children's altruistic time provisions. The problem however, is that for some diseases, particularly Alzheimer's and dementia, no such treatments (at the time this paper was written) presently exist. Given this, the choice of

$dr_{y,t}$ must satisfy:

$$\frac{\gamma}{x_{y,t}} = \beta \cdot s_{o,t+1} \cdot R_{t+1} \frac{\partial \psi_{t+1}(dr_{y,t})}{\partial dr_{y,t}} \left[\ln \left(x_{o,t+1}(1)^{1-\sigma} h_{o,t+1}^{\sigma} - \nu \right) - \ln x_{o,t}(0) \right] \quad \text{if} \quad p_t < \infty \tag{15}$$

$$dr_{y,t} = 0 \quad \text{if} \quad p_t = \infty \tag{16}$$

where

$$\frac{\partial \psi_{t+1}(dr_{y,t})}{\partial dr_{y,t}} = \frac{\ln(\epsilon) \overline{\psi} \epsilon^{-dr_{y,t}}}{(1 + \overline{\psi} \epsilon^{-dr_{y,t}})^2} \tag{17}$$

(17) requires implicit differentiation, which is straightforward. The details are in Appendix B.

3.2 Firms

The economy consists of two possible types of firms: 1) either a monopolistic or representative firm in a competitive environment trying to produce or indeed producing a cure DR_t for the welfare-debilitating disease, 2) a representative firm in a competitive environment producing all other market goods X_t and an investment good I_t.[18] We refer to the latter type of firm often as a "consumption producing" firm. Drug-producing firms only use capital inputs, while consumption producing firms use both capital and labor. Each type of firm faces the same pre-subsidy capital rental rate of r_t. We incorporate a mechanism into the model allowing the government to subsidize investment by drug-producing firms, so the post-subsidy capital rental rate faced by them is $r_t(1 - sub_t)$. In our simulations, we will explore how hypothetical economies function under various subsidization rates.

Aggregate capital K_t evolves according to

$$K_{t+1} \leq (1 - \delta) K_t + I_t \tag{18}$$

where δ is net depreciation. We denote capital used in the production of drugs as $K(DR)_t$ and capital used in the production of all other market goods as $K(X)_t$.[19] Aggregate capital

[18] Firm variables and aggregates are denoted with capital letters, sticking with established conventions.

[19] We acknowledge that $K(X)_t$ is used to produce both consumption goods X_t and investment goods I_t, but express its dependency on these arguments by simply using X_t for simplicity.

must add up:

$$K_t = K(X)_t + K(DR)_t \tag{19}$$

Further, household asset choices ensure $a_{y,t} \cdot N_{y,t-1} = K_t$. Note that households do not care for which production process their capital is used, since they receive the same gross rate of return R_t either way. This forces marginal products of capital between firms to equate.

The static, single period, profit maximization problem facing the representative firm producing $X_t + I_t$ is fairly standard. We assume period t production is Cobb-Douglas with total factor productivity z_t. Each period the consumption firm solves

$$\max_{K(X)_t, L_t} z_t K(X)_t^{\alpha} L_t^{1-\alpha} - r_t K(X)_t - w_t L_t \tag{20}$$

where $\alpha \in (0, 1)$ governs the elasticity of output with respect to capital. First order necessary conditions for this firm are then

$$r_t = z_t \alpha \left(\frac{L_t}{K_t} \right)^{1-\alpha} \tag{21}$$

$$w_t = z_t (1 - \alpha) \left(\frac{K_t}{L_t} \right)^{\alpha} \tag{22}$$

Meanwhile, the problem facing the drug-producing firm depends on whether a cure has been successfully found or developed yet. If there exists no cure for the welfare debilitating disease, then the drug-producing firm utilizes capital for research and development (R & D) in order to push out the technological frontier to the point where such a cure exists and the firm can then engage in positive production. Of course, there exists a degree of randomness associated with whether or not R & D toward a cure actually leads to a state of the world where such cures are successful and can be positively produced. By investing in R & D, the probability a cure is found increases. But a potential drug making firm that invests in R & D must have some expectation of a future payoff in order for such an investment to be viable. We assume that the firm investing in R & D prior to the existence of a cure knows that if its investment is successful, it will be awarded an exclusive patent for production of the drug. The term of this patent will be one model period, so immediately following the discovery of a cure, the drug making firm acts as a price-setting monopolist reaping positive economic profits. Once the patent expires, two periods after the discovery of a cure, the market for DR_t is assumed to be perfectly competitive thereafter. In that situation, a representative firm produces DR_t while earning zero economic profits.

Let $cure_t$ be a binary variable that equals zero if no cure exists in period t, or 1 if a cure exists. Suppose $cure_t = 1$ and $cure_{t-1} = 0$, then DR_t is produced in positive quantities by a monopolist. If $cure_t = 1$ and $cure_{t-1} = 1$, then DR_t is produced by a representative firm in a competitive environment. The most interesting production problem occurs for $cure_t = 0$. Let $\xi_{t+1}\big(K(DR)_t \mid cure_t = 0\big)$ be the probability a cure is not found in period $t+1$ given $cure_t = 0$. Investments in R & D affect this probability according to the same logistic structure as in (7):

$$\xi_{t+1}\big(K(DR)_t \mid cure_t = 0\big) = \frac{1}{1 + \bar{\xi}\varphi^{-K(DR)_t}} \tag{23}$$

Thus, ξ_{t+1} is decreasing in capital investment sending the probability of *not* finding a cure down, which means that $1 - \xi_{t+1}$ is increasing, sending the probability of finding a cure up. In period t with no existent cure, the representative firm expects that if it achieves a cure it will reap the profits of a monopolist in $t+1$, $\mathbb{E}_t\Pi_{t+1}\big(K(DR)_{t+1} \mid cure_{t+1} = 1, cure_t = 0\big)$. Thus a firm chooses $K(DR)_t$ so as to solve:

$$\begin{aligned}\max_{K(DR)_t} \Big\{ \Big(1 - \xi_{t+1}\big(K(DR)_t \mid cure_t = 0\big)\Big)\mathbb{E}_t\Pi_{t+1}\big(K(DR)_{t+1} \mid cure_{t+1} = 1, cure_t = 0\big) \\ - r_t(1 - sub_t)K(DR)_t \Big\}\end{aligned} \tag{24}$$

We assume that if no cure is achieved, so that $cure_{t+1} = 0$ and $cure_t = 0$, all firms competing for a patent go out of business at the end of period t and exit the market. In $t+1$, the competitive environment persists with new firms entering the market, solving the same problem as above. Thus, current R & D investors solve a static two period problem. This process repeats until a cure is found.

Let $p_t(DR_t)$ be the aggregate inverse demand function for DR_t. In the first period after a cure is found, the monopolist chooses DR_t so as to solve

$$\max_{DR_t} \Big\{ p_t(DR_t)DR_t - \mathcal{C}_t(r_t, sub_t, DR_t) \Big\} \quad \text{if} \quad cure_t = 1 \quad \& \quad cure_{t-1} = 0 \tag{25}$$

where $\mathcal{C}_t(r_t, sub_t, DR_t)$ is the cost of producing DR_t. DR_t is produced according to the following Cobb-Douglas specification with total factor productivity ζ_t:

$$DR_t = \zeta_t K(DR)_t^{\kappa} \tag{26}$$

with $\kappa \in (0, 1)$. Under Cobb-Douglas production, costs can be written:

$$\mathcal{C}_t(r_t, sub_t, DR_t) = r_t(1 - sub_t)\left(\frac{DR_t}{\zeta_t}\right)^{\frac{1}{\kappa}} \tag{27}$$

Beginning the period immediately after the monopolist is reaping profits by setting prices, perfect competition takes over and continues forever. The representative drug-producing firm takes prices as given and solves

$$\max_{K(DR)_t} \left\{p_t\zeta_t K(DR)_t^{\kappa} - r_t(1 - sub_t)K(DR)_t\right\} \quad \text{if} \quad cure_t = 1 \quad \& \quad cure_{t-1} = 1 \tag{28}$$

The drug-producing firm's first order conditions depend on the state of the market. For $cure_t = 0$ the firm chooses $K(DR)_t$ to solve the expected Euler equation:

$$-\frac{\partial \xi_{t+1}\big(K(DR)_t\big)}{\partial K(DR)_t}\mathbb{E}_t\Pi_{t+1}\big(DR, K(DR)_{t+1} \,|\, cure_{t+1} = 1, cure_t = 0\big) = r_t(1 - sub_t) \tag{29}$$

As with $\frac{\partial \psi_{t+1}(dr_{y,t})}{\partial dr_{y,t}}$, the same implicit differentiation routine applies, so we have

$$\frac{\partial \xi_{t+1}\big(K(DR)_t\big)}{\partial K(DR)_t} = \frac{\ln(\varphi)\overline{\xi}\varphi^{-K(DR)_t}}{(1 + \overline{\xi}\varphi^{-K(DR)_t})^2} \tag{30}$$

Note that $\frac{\partial \xi_{t+1}\left(K(DR)_t\right)}{\partial K(DR)_t} < 0$ for all positive $K(DR)_t$. Meanwhile, when $cure_t = 1$ and $cure_{t-1} = 0$, the monopolist solves the ordinary differential equation

$$\frac{\partial p_t(DR_t)}{\partial DR_t}DR_t + p_t = \frac{r_t(1 - sub_t)}{\kappa\zeta_t}\left(\frac{DR_t}{\zeta_t}\right)^{\frac{1-\kappa}{\kappa}} \tag{31}$$

And finally, the representative firm in competitive equilibrium, when $cure_t = cure_{t-1} = 1$ solves

$$r_t(1 - sub_t) = \zeta_t\kappa K(DR)_t^{\kappa-1} \tag{32}$$

Proposition 1. When both the market for $X_t + I_t$ and the market for DR_t are perfectly competitive and $\alpha = \kappa$

$$r_t = K_t^{\alpha-1}\left[\left(\frac{1 - sub_t}{\zeta_t p_t \alpha}\right)^{\frac{1}{\alpha-1}} + \left(\frac{1}{z_t\alpha}\right)^{\frac{1}{\alpha-1}} L_t\right]^{1-\alpha} \tag{33}$$

Proof. See Appendix B □

In Section 4, we will generally ignore the market for a cure, calibrating to an economy where no cure exists, and firms are not engaged in R & D toward developing a cure. This is mostly for identification purposes: it is not possible to identify or even exogenously fix parameter $\overline{\xi}$ and φ without more information about the ex-ante likelihood that investment in disease-elimination research will payoff. Given no such cure has yet occurred, we thus ex-ante possess no information about this probability. It could indeed be very small and relatively unaffected by increasing $K(DR)_t$. It could, on the other hand, be substantially impacted by changing $K(DR)_t$, though researchers have thus far just been unlucky. Both situations are observationally equivalent. Lacking any other information to go on, we choose to take a conservative approach to calibration. Instead, we consider some thought experiments in Section 5 examining both the steady state of an economy featuring a cure market and the dynamic evolution of that economy given the market structure we have imposed.

3.3 Government

The government has several responsibilities. First, it must provide Social Security and Medicare/Medicaid benefits to elderly members of society. Second, it can decide how much to subsidize investment in R & D toward a cure, if such investment is taking place. Let $\rho_t = T_t(1)/T_t(0)$ be the ratio of diseased to non-diseased benefits received. The government balances Social Security and Medicare/Medicaid transfers and tax receipts, along with subsidization toward drug-producing firms according to:

$$N_{ot} \cdot \left(\psi_t \cdot \rho_t \cdot T_t(0) + (1-\psi_t)T_t(0)\right) \leq N_{yt} \cdot w_t \cdot \tau_t \cdot (1 - l_{y,t} - h_{y,t}) + r_t \cdot sub_t \cdot K(DR)_t \tag{34}$$

3.4 Recursive Equilibrium

Encode the current status of the drug market in mkt_t which captures both whether a cure was in place last period and/or this period. Given exogenously specified disease to non-disease benefit ratios, survival rates, population levels, productivities, and status of the drug marketplace $\{\rho_t, s_{o,t+1}, N_{y,t}, N_{o,t}, z_t, \zeta_t, mkt_t\}$, a recursive equilibrium consists of:

i. Policies for consumers: $\left\{x_{y,t}, l_{y,t}, h_{y,t}, dr_{y,t}, a_{y,t+1}, x_{o,t}(1), x_{o,t}(0)\right\}$.

ii. Policies for firms: $\left\{L_t, K(X)_t, K(DR)_t, \right\}$.

iii. Government policies $\{T_t(1), T_t(0), \tau_t, sub_t\}_{t\geq 0}$.

iv. Prices $\{p_t, R_t, w_t\}$.

such that

a. Young agents' choices satisfy (11) and (12) thru (14).

b. Old agent consumption policies satisfy (9).

c. Asset return rates ensure the marginal products of capital equate across industries for all market structures.

d. Wage rates satisfy (22).

e. Formal and informal markets clear.

f. The government's budget is balanced.

3.5 Growth

One purpose of this paper is to understand how time-use tradeoffs faced by working-age consumers with reverse altruism motives in a general equilibrium model with welfare risk impact aggregate economic growth above and beyond the effects of population aging already documented in Cooley and Henriksen (2018). Consider now the case where $\zeta_t = 0$ in all periods, so that curing the welfare-debilitating is impossible. Consumers thus know with certainty that they face a fixed risk of contracting a welfare-debilitating disease in old age of $\psi = \frac{1}{1+\overline{\psi}}$. In this situation, the insurance against such diseases provided by the presence of a market $DR_t > 0$ is eliminated. Yet, young agents make consumption and savings decisions today taking into consideration the fact that their offspring will help supplement their elder care if they get a disease. The reverse altruism thus takes the role of insurance. When it is known with certainty how the population distribution will evolve, only idiosyncratic risk is present. Further, in a representative agent environment with only one type of young agent, young agents today directly (in fact, deterministically) impact the amount of care time they will receive if such a disease is contracted by choosing $a_{y,t+1}$, the model's only endogenous state variable. Given the smooth evolution of long-run economic growth rates, it is within this model environment that we seek to consider how the exogenous evolution of the population distribution impacts aggregate output growth through this new insurance mechanism.

Herein lies he beauty of the two period assumption: only changes to the working age to retiree population ratio (not levels) $wapr_t$ affect equilibrium outcomes. This is illustrated in Proposition 2. As a corollary to Proposition 2, we also demonstrate that in this environment, aggregate output growth $g_{Y,t}$ depends only on $wapr_t$, not generational population levels.

Proposition 2. Assume z_t grows at constant rate g_z and $N_{y,t}$ grows at constant rate g_N. Suppose $\zeta_t = 0$, so that $K(DR)_t = 0$. Then a competitive equilibrium depends only on $wapr_t$, not the population levels.

Proof. See Appendix B □

Corollary 1. Assume z_t grows at constant rate g_z and $N_{y,t}$ grows at constant rate g_N. Suppose $\zeta_t = 0$, so that $K(DR)_t = 0$. Then aggregate growth $g_{Y,t}$ depends only on $wapr_t$, not population levels.

Proof. See Appendix B □

Continue operating under the assumption that $\zeta_t = 0$ for all t. To solve for a steady state, we assume a balanced growth path (BGP) and de-trend productivity growth as in Krueger and Ludwig (2007). For now, suppose $\tau_t = \tau$ is exogenously fixed. Along a BGP the population of young agents and consumption output productivity grow at constant exogenous rates, g_N and g_z. Proposition 2 demonstrates that if survival rates and disease risk are constant, then $wapr$ is constant across time.

Proposition 3. Assume z_t grows at constant rate g_z and $N_{y,t}$ grows at constant rate g_N. Suppose $\zeta_t = 0$, so that $K(DR)_t = 0$. Assume the survival rate $s_{o,t+1} = s_o$. Then along a BGP the working-age population ratio $wapr$ is constant and given by

$$wapr = \frac{1 + g_N}{s_o} \tag{35}$$

Proof. See Appendix B □

Clearly, the assumption that $wapr$ is constant is unrealistic in practice, as we see that $wapr$ has been falling over time and is projected to continue falling. This fact begs the question as to whether the U.S. economy in the 21st century is in fact on a balanced growth trajectory or rather is exhibiting structural change due to forces such as population aging and potentially associated idiosyncratic welfare risk affecting long-run growth rates. In Section 5 we simulate the future path of aggregate output growth to understand the extent to which falling $wapr_t$, coupled with idiosyncratic welfare risk and young agents' altruism, together impact aggregate growth.

4 Calibration

For our calibration we set the period length to 40 years and assume young agents enter the economy at age 25 and turn old at age 65. Our calibration assumes the economy is in steady state in 2017, thus taking the 2017 observed population distribution as the initial steady state distribution. Assume that $\zeta_t = 0$ for all periods, so that there is no market for investment towards a cure. We choose parameters to match a set of carefully selected data moments from around 2017. Specifically, we calibrate to leisure, labor, and hospice care average time shares from the 2003-2017 ATUS data, the personal savings rate from the BEA's personal income and outlay data series which measures personal savings as a percentage of disposable income, the ratio of diseased to non-diseased consumption computed from estimates made by Hurd et al. (2013), the 2017 consumption and investment shares of output, and the 2015 U.S. labor and capital income shares from Penn World Table 9.0. Table 2 presents the data moment targets and their simulated model counterparts, while Table 3 presents the calibrated parameter values and their sources.

Table 2: Calibration Targets

Moment	Model	Data	Source
l_y^*	0.647	0.663	ATUS, 2003-2017 Avg.
$1 - l_y^* - h_y^*$	0.347	0.333	ATUS, 2003-2017 Avg.
h_y^*	0.006	0.003	ATUS, 2003-2017 Avg.
Savings Rate	0.152	0.179	NIPA Table 2.6., January 2017
$x_o^*(1)/x_o^*(0)$	1.311	1.360	From Hurd et al. (2013)
X^*/Y^*	0.789	0.799	NIPA Table 1.5.6., 2017
I^*/Y^*	0.211	0.201	NIPA Table 1.5.6., 2017
Labor Income Share	0.651	0.686	NIPA Table 2.6., January 2017
Capital Income Share	0.349	0.314	NIPA Table 2.6., January 2017

The calibration requires a couple of assumptions for identification purposes. First, we exogenously set the benefits ratio ρ using estimates by Hurd et al. (2013) and Mommaerts (2016) to get $\rho = 1.923$.[20] Thus, diseased agents receive almost double the benefits from the government as non-diseased agents. To calibrate to a steady state assuming it has been de-trended from a BGP, we only have to pick two of g_N, s_o, and $wapr$, due to Equation (35). We set g_N to accommodate growth in the young population since 1976 and $wapr$ to equal the observed population ratio for workers to retirees in 2017. We exogenously fix the parameters μ, γ, and η to reflect the observed ATUS time-use averages from 2003-

[20]The procedure used to set this parameter is described in detail in Appendix C.

2017. The output elasticity α is chosen to match the average U.S. capital share since World War II. The baseline risk parameter $\overline{\psi}$ is fixed to match risk rate estimates from Hurd et al. (2013). We calibrate the subsistence parameter ν and intensity of hospice care parameter σ to match aggregate data moments, including the ratio of diseased to non-diseased consumption $x_o(1)/x_o(0)$ taken from estimates in Hurd et al. (2013).

Table 3: Calibrated Parameter Values

	Value	Source
g_N	0.643	Growth in Age 25-65 Pop. (1977-2017)
δ	0.9517	40 years of 6% annual depreciation
α	0.35	Post-war avg. capital share
β	0.4457	Annual discounting of 0.98 over 40 years
μ	0.663	ATUS (2003-2017) avg. leisure
γ	0.333	ATUS (2003-2017) avg. work
η	0.003	ATUS (2003-2017) avg. adult care
$wapr$	3.382	U.S. Working-age pop. ratio 2017
τ	0.153	S.S. + Medicare tax rate 2017
ρ	1.923	Ratio of diseased/non-diseased benefits
$\overline{\psi}$	6.143	Risk of contracting dementia of 0.14 (Hurd et al. 2013)
ν	0.100	Subsistence of old (calibrated to match data)
σ	0.659	Intensity of h_{ot} in home production (calibrated to match data)

5 Findings

We engage in a series of quantitative exercises to understand how the type of welfare risk we are modeling affects agents' choices and, via their decisions, long-run economic growth. For all of our counterfactuals except those in Section 5.3, we assume that $\zeta_t = 0$ for all periods, so that there is no market for investment towards a cure, as in the calibration. In these initial exercises, we find that risk reduction affects growth marginally, though population aging is still the biggest driver of predicted long-run growth declines, consistent with Cooley and Henriksen (2018). We show that policymakers may be able to both offset growth declines and improve welfare by implementing a reimbursement scheme where young agents who provide time off market caring for their elders receive market-rate compensation.

In Section 5.3 we simulate the transition path of an economy with a monopolistic drug provider with $DR_t > 0$ transitioning into one where the supply of these treatments for the welfare-debilitating disease is still positive, but resulting instead from perfect competition on the producer side. We also examine how demand for $dr_{y,t}$ affects the equilibrium risk rate in a competitive steady state, varying $wapr_t$ and the productivity ratio ζ_t/z_t for different parameter values of ϵ. Our findings suggest that the equilibrium elasticity of the risk rate with respect to $wapr_t$ and the relative price of treatments is extremely low. These exercises open more questions as to whether subsidization of R & D toward a cure will lead to significant ex-ante purchases of treatments by consumers to reduce this risk.

5.0.1 Predicted U.S. Aggregate Output Growth

We want to understand how well the model predicts different aggregate growth rates when the population is evolving in ways inconsistent with balanced growth. We compute a transition path from starting steady states with $wapr$ equivalent to those observed in 1950, 1960, 1970, 1980, and 1990, simulating toward a terminal steady state with $wapr = 3.382$ as observed in 2017. We assume period 0 of the model is in one of the old $wapr$, and then the economy suddenly changes to $wapr = 3.382$, allowing 200 simulated periods to facilitate convergence to the new steady state.[21] For each simulation, we set the initial steady state's τ to the actual employee and employer combined Social Security and Medicare tax rate for the given year.[22] Productivity growth is set to accommodate the observed average annual private multi-factor (MFP) productivity growth rates for the periods 1950-2017, 1960-2017, 1970-2017, 1980-2017, and 1990-2017.[23] We compare both predicted productivity re-trended aggregate output growth and per-worker re-trended output growth from the first period after the sudden change in working age population ratio to that observed in the data. These values are presented in Table 4 under columns labeled "Model g_Y" and "Model g_{Y/N_y}," where the former describes aggregate growth and the latter growth per working-age adult. Model predictions slightly undershoot aggregate growth rates for all periods.

We run two additional simulations adjusting g_z to match observed g_Y and g_{Y/N_y}. Implied productivity growth from these simulations is presented in columns labeled "Implied g_z" and "Implied g_{z/N_y}" in Table 4. In the data, g_{z/N_y} is negative since the 1960s when the denominator we use to compute output per-worker is the entire working-age adult population. To reconcile per-worker growth, our model requires growth in produc-

[21] For a thorough explanation of how to accomplish this simulation technique in an overlapping generations model see the endogenous grid point method of Carroll (2006) and Appendix B of Krueger and Ludwig (2007).

[22] The tax rates are as follows: 3% (1950), 6% (1960), 9.6% (1970), 12.26% (1980), 15.3% (1990 and thereafter).

[23] MFP is real value-added output divided by combined inputs — *U.S. Bureau of Labor Statistics, Private Business Sector: Multi-factor Productivity [MFPPBS], retrieved from FRED, Federal Reserve Bank of St. Louis; https://fred.stlouisfed.org/series/MFPPBS, Accessed: March 19, 2019.*

Table 4: Model Performance: Predicted Avg. Annual Growth to 2017

		Growth in Aggregate Output, (%)			
Data Period	Starting $wapr$	Model g_Y	Data g_Y	Implied g_z	Data g_z
1950-2017	6.111	3.076	3.083	1.186	1.162
1960-2017	5.114	2.897	2.995	1.174	1.107
1970-2017	4.414	2.474	2.732	1.092	0.869
1980-2017	4.075	2.179	2.619	1.260	0.844
1990-2017	4.028	1.926	2.371	1.232	0.862
		Growth in Output Per Working Age Adult, (%)			
Data Period	Starting $wapr$	Model g_{Y/N_y}	Data g_{Y/N_y}	Implied g_{z/N_y}	Data g_{z/N_y}
1950-2017	6.111	1.155	1.949	2.008	0.049
1960-2017	5.114	1.206	1.825	1.665	-0.076
1970-2017	4.414	0.960	1.465	1.409	-0.374
1980-2017	4.075	1.045	1.420	1.198	-0.333
1990-2017	4.028	1.227	1.368	1.035	-0.126

tivity per-worker to exceed growth in aggregate productivity as can be seen by comparing the "Implied" column of the bottom half of Table 4 to the top half. The model thus appears to do a decent job of matching aggregate output growth but not output per-worker. This is due to the fact that we assume a 40-year transition period regardless of the starting $wapr$ being associated with the year 1950 or 1990. Comparisons between aggregate numbers are not biased by this fact because the aggregate growth rate does not depend on a scaling with the growth rate of newborns entering the economy, g_N, which must be computed to accommodate the transition from the initial steady state $wapr$ to the terminal one. If we allow for the possibility that perhaps measurements of MFP in the NIPA tables themselves are biased, failing to account for endogeneity due to g_z's dependence on the population distribution, then U.S. productivity growth over the second half of the twentieth century has perhaps been overstated, or at the least misunderstood. At first consideration, it is hard to ignore the positive correlation between measured productivity per-worker and $wapr$. One can think of a number of possible ways in which $wapr$ may affect productivity: younger workers have more energy and work more in order to build up a nest egg from scratch, for example. In the context of our formulation, a relatively large population of retirees could negatively weigh on aggregate productivity by diverting working-age adults' attention from their jobs because they provide informal care. If this explanation were true, z_t would be an endogenous function of $wapr_t$, and $g_{z,t}$ would vary in time, falling along with $wapr_t$. We do not take a stance on the mechanism by which z_t may partially depend endogenously on $wapr_t$. Rather, the decomposition in Table 4 illustrates what the $wapr_t$-independent component of $g_{z,t}$ would need to be in order to match observed output growth under our parameterization. In general, the results of these simulations show that the given model can accurately predict aggregate growth, suggesting researchers should take our future growth estimates presented in Section 5.1 seriously, affirming the general spirit of the results in Cooley and Henriksen (2018).

5.1 Future Growth Under Different Counterfactual Regimes

One goal of this project is to understand how the welfare risk of contracting a debilitating old-age disease may affect future aggregate output growth while the population is aging. As a baseline, we follow techniques described in Krueger and Ludwig (2007) to simulate a transition path between the calibrated 2017 steady state and a far-off future steady state assuming the population converges after 200 periods to the United Nations predicted, 2097 median-variant population distribution.[24] We then examine projected growth rates and lifetime welfare under the following policy reforms. First, we consider how the economy evolves when the "dynamically ignorant" government suddenly sets $\tau = 0$ one period into the future and households are surprised by this change, failing to anticipate it.[25] Second, we consider a policy reform where the government decides to fully reimburse working-age adults for their off-market time at the before-tax market wage.[26] Finally, we simulate a dynamic transition path under unexpected, exogenous changes to the disease risk rate. We consider growth under an exogenous elimination of risk by 2057 and by 2097, as well as growth under 10%, 20%, 50%, and 100% increases in cross-sectional risk by 2097.[27] These changes are all based on the value of $\psi = 0.14$ used in calibration, taken from estimates of dementia risk for 70 year olds in Hurd et al. (2013).

[24] See United Nations: Department of Economic and Social Affairs 2017.

[25] Here, we consider the 2017 $wapr = 3.382$ as "present."

[26] Currently, some U.S. state Medicaid programs reimburse family members for care time they provide to Medicaid recipients, though the rates of reimbursement and restrictions vary substantially across states. Current data on state-level Medicaid policies does not appear to be readily available in a central source.

Under this reform, the young agent's budget constraint is:

$$x_{y,t} + a_{y,t+1} \leq R_t \cdot b_{y,t} + w_t(1-\tau_t)(1-l_{y,t}-h_{y,t}) + w_t \cdot h_{y,t} \tag{36}$$

while the government faces budget constraint:

$$N_{ot} \cdot \big(\psi_t \cdot \rho_t \cdot T_t(0) + (1-\psi_t)T_t(0)\big) \leq N_{yt} \cdot \big(w_t \cdot \tau_t \cdot (1-l_{y,t}-h_{y,t}) - w_t \cdot h_{y,t}\big) \tag{37}$$

[27] For a "cure" we consider a situation where ψ drops to 0.0001 to ensure Inada conditions hold.

Table 5: Growth Under Different Regimes, $dr_{y,t} = 0$, $g_z = 1.4\%$

Model	Pop. Transition?	Predicted Avg. Annual Growth g_Y, (%)		
		2017-2057	2017-2097	2017-2137
BGP ($\tau = 0.153$, $\psi = 0.14$)*	No	2.67	2.67	2.67
Baseline	Yes	2.208	2.020	1.862
$\tau = 0$	Yes	2.359	2.271	2.072
Reimbursement of $h_{y,t}$ at w_t	Yes	2.206	2.042	1.885
Exogenous Cures				
$\psi_{2057} = 0.0001$, $\psi_{2097} = 0.0001$	Yes	2.262	2.113	1.941
$\psi_{2057} = 0.07$, $\psi_{2097} = 0.0001$	Yes	2.232	2.086	1.934
$\psi_{2057} = 0.146$, $\psi_{2097} = 0.154$	Yes	2.210	2.037	1.877
$\psi_{2057} = 0.154$, $\psi_{2097} = 0.168$	Yes	2.207	2.032	1.871
$\psi_{2057} = 0.175$, $\psi_{2097} = 0.210$	Yes	2.200	2.016	1.851
$\psi_{2057} = 0.21$, $\psi_{2097} = 0.280$	Yes	2.188	1.987	1.815
End-of-period $wapr$ used in simulations:		2.089	1.651	1.550

* $\tau = 0.153$ and/or $\psi = 0.14$ unless otherwise noted.

For computational reasons, we assume changes are permanent after 2097 so the economy has some steady state outcome to which to converge.

Table 5 presents simulated average annual aggregate output growth rates (g_Y). For the baseline simulations holding τ and ρ at their observed and calibrated 2017 values, we compare the dynamic transition path of an economy aging according to U.N. projections. Our regime-change counterfactuals operate as follows. First, we suppose that the economy is in the initial 2017 steady state, then the regime change occurs suddenly. For all of these changes, in period $t = 2$ right after the 2017 steady state, the economy has changed unexpectedly, but agents have not updated their dynamic plans. We thus simulate the economy for 200 periods to allow for it to converge to the new steady state, which generally happens after only 7-10 model periods anyway. All of our counterfactual simulations occur off a BGP, where the population distribution is evolving exogenously according to U.N. estimates.

From these simulations it is apparent that an aging population substantially reduces growth relative to a BGP where $wapr$ remains constant. In fact this reduction is on the order of 65 basis points annually, leading to compounded aggregate output losses of 15% by 2057 and 35% by 2097 relative to an economy where $wapr$ held constant at the 2017 level. Though perhaps politically unrealistic, it is illustrative that in this economy setting $\tau = 0$

can lead to both Pareto improvements along the dynamic transition path and increases in compounded aggregate output relative to the baseline with population transition — 4% higher by 2057 and 12% higher by 2097. Figure 2 presents the fully compounded predicted population baseline growth relative to counterfactual growth projections, including the BGP. Implementing a before-tax reimbursement policy while holding $\tau = 0.153$ fixed yields Pareto improvements but adversely affects compounded growth relative to the population transition baseline in the first period — output is 0.2% lower by 2057 — though growth improves slightly by the end of the century — 0.2% higher by 2097. The predicted baseline falls the most relative to BGP, then the tax-free environment, then finally the full elimination of risk. Neither stabilizing the population distribution to achieve a BGP nor eliminating Social Security and Medicare are realistically feasible, yet scientists are working to find cures for dementia-like diseases. In Section

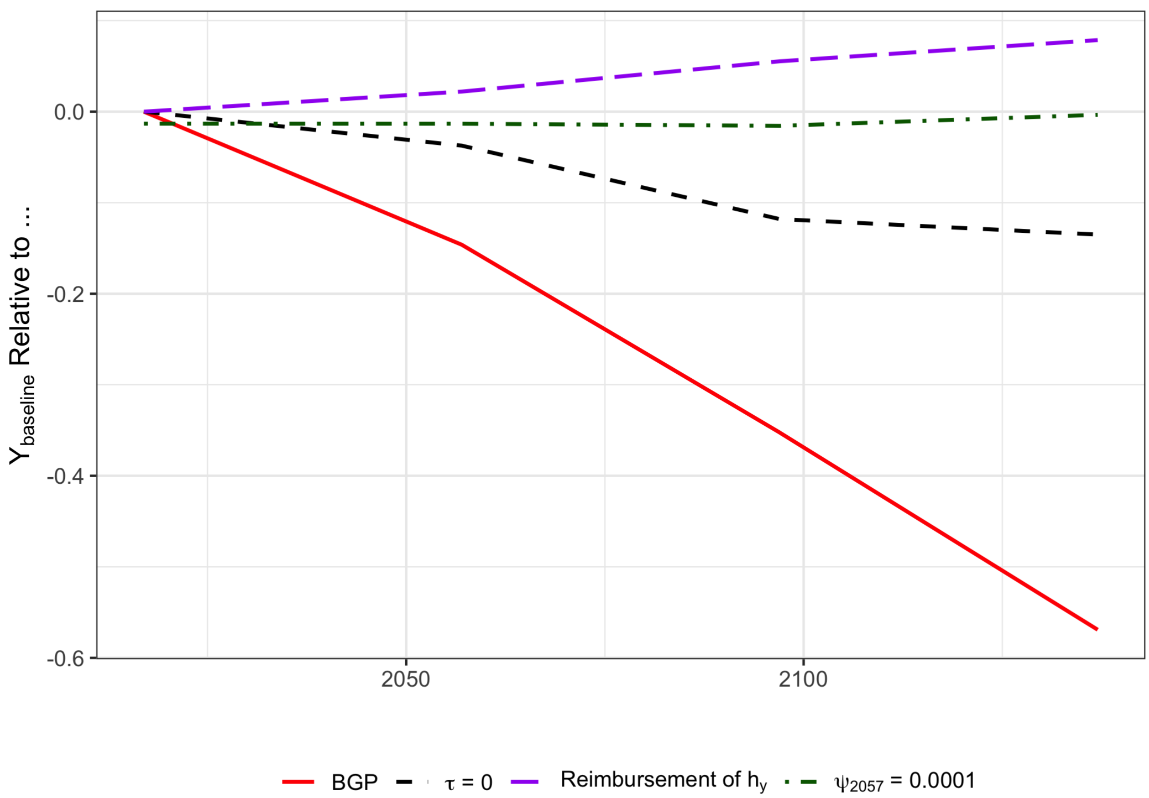

Figure 2: Here we present predicted baseline output relative to various counterfactuals, $(Y_{\text{baseline}} - Y_{\text{counter}})/Y_{\text{counter}}$. A cure for dementia by 2057 ($\psi = 0.0001$; green dashed line) can lead to modest improvements relative to the baseline.

One takeaway we wish to emphasize is that achieving a full cure — $\psi = 0.0001$ by 2057 — would have a small impact on growth, increasing g_Y by only about 10 basis points. Long-run growth rates and welfare are decreasing in ψ. The most striking thing about our simulations under different ψ is that changing the risk rate hardly matters for long-run growth prospects. Rather, the population distribution, regardless of the risk rate, has the largest effect on long-run growth, which can be seen by comparing any of the simulations that account for population transitions with the BGP. All counterfactuals result in anywhere from 60 to 90 basis point relative declines in the average annual growth rate by 2137, and 40 to 70 basis point relative declines by 2097. While this result should mitigate concerns that the burdens of old-age care alone will tamp down growth, we confirm recent findings in Cooley and Henriksen (2018) suggesting a long-run "demographic deficit" may be coming to the United States economy.

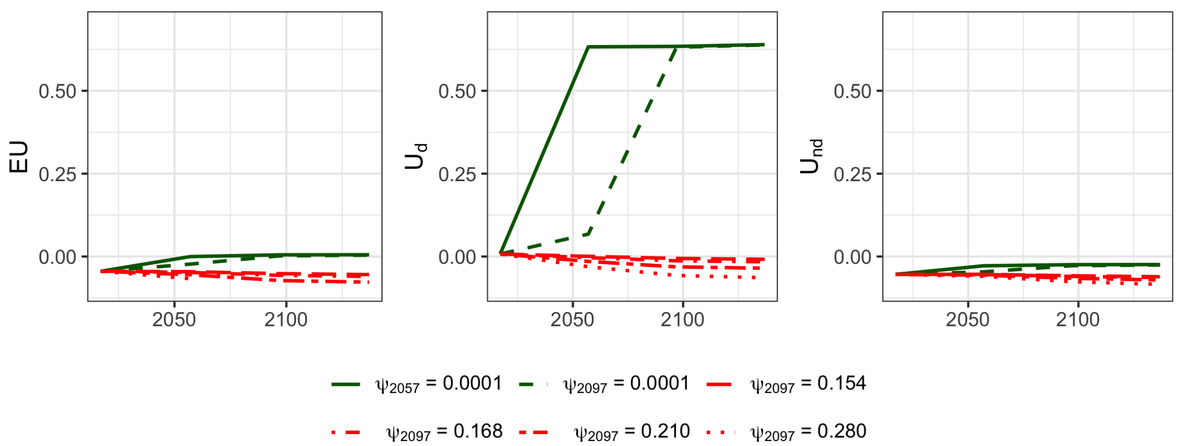

Figure 3: We present welfare as share of the predicted baseline with population transition. Full cures ($\psi \to 0$; green lines) generate Pareto improvements for all types of agents. As ψ increases (red lines), welfare falls relative to the predicted baseline with $\psi = 0.14$.

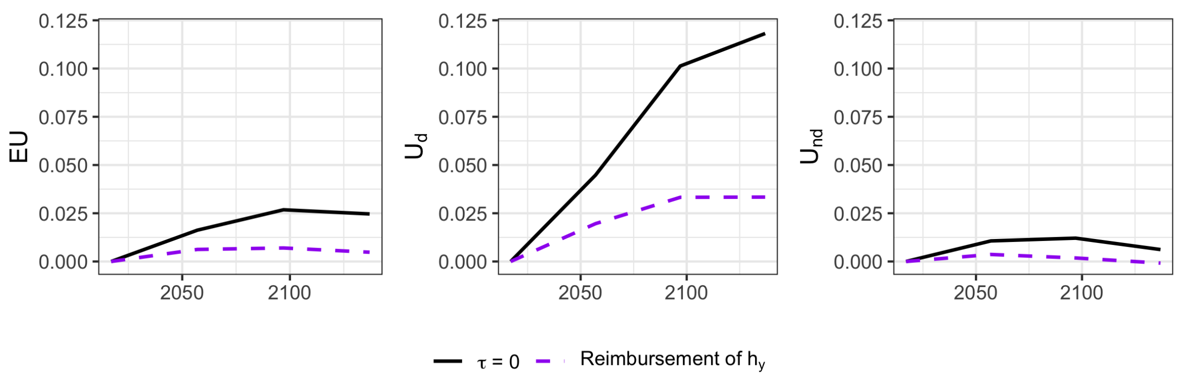

Figure 4: Again, welfare is presented as share of predicted baseline with population transition. Pareto improvements are generated when Social Security and Medicare taxes are unexpectedly eliminated. Reimbursing young agents' time supplying care on the informal market $h_{y,t}$ at the market rate w_t also yields Pareto improvements.

Yet an exogenous reduction in disease risk, despite having minimal impact on growth, is still welfare improving. Figure 3 compares welfare paths for different possible risk rates, relative to a baseline economy where $\psi = 0.14$, as estimated by Hurd et al. (2013). Notice that welfare improves for all agents as $\psi \to 0$, though risk reduction has the most pronounced effect on the welfare of those very few agents who remain diseased, leading to a greater than 60% lifetime gain relative to the baseline. Next, young agents enjoy higher expected lifetime utility, but are also hit hardest relative to the baseline when ψ increases. This is because children shoulder the burden of increased numbers of diseased old through the altruism mechanism. Meanwhile, Figure 4 compares welfare paths over the 21st century relative to the population transition baseline for the tax-free environment and an economy with a reimbursement scheme. Lifetime welfare of all agents over the 21st century is improved from baseline under the reimbursement scheme, though again the most notable improvement is for diseased agents. This finding is particularly promising since growth is relatively unaffected by such a scheme, yet all agents are better off. Further, reimbursement schemes are already implemented in certain states. Our results suggest that more adoption of these policies will lead to welfare improvements across the board.

5.2 Steady State Comparative Statics, $\zeta_t = 0$

Using the calibrated parameters from Table 3 we simulate steady state outcomes under different working-age population ratios and compare them to an economy without a social insurance system. We present selected policies and aggregate outcomes in Figure 5. The model predicts that both labor supply and total time spent providing informal care is higher when the social safety net is eliminated. Young people sacrifice leisure time for work because wages are higher and pick up the slack caring for their elders at all levels of $wapr$. All values are monotonic in $wapr$, though the signs of some of the relationships may be surprising. Not surprisingly, labor and output are increasing in $wapr$, but work hours are increasing because wages are decreasing: holding productivity fixed, wages are bid down as the number of workers increases. Steady state savings rates increase in $wapr$ as a response to higher interest rates, driven up by increases in the labor supply

[28] Let $\mathbb{E}u(d)$ be expected lifetime steady state utility, $u(d=1)$ be realized lifetime utility for a diseased agent, and $u(d=0)$ be realized lifetime utility for a non-diseased agent. These values are as follows:

$$\mathbb{E}u(d) = u_y + \beta \cdot \left[\psi \cdot u_o(1) + (1-\psi) \cdot u_o(0)\right] \tag{38}$$

$$u(d=1) = u_y + u_o(1) \tag{39}$$

$$u(d=0) = u_y + u_o(0) \tag{40}$$

forcing firms to acquire more capital to efficiently utilize the skills of increasing numbers of workers. U.S. personal savings rates have generally fallen since the 1950's, from 11.3% in January 1959 when *wapr* was at 5.176 to 6.3% in December 2016 with a *wapr* of 3.475, confirming the validity of the sign of the relationship we observe here.

For each of the $\tau = 0$ and $\tau = 0.153$ case, we simulate expected lifetime utility for a young agent who has not yet realized his old-age disease status as well as realized lifetime utility for both diseased $d = 1$ and non-diseased $d = 0$ old agents.[28] Figure 6 presents these welfare values as functions of *wapr*. This exercise demonstrates that for small enough *wapr*, higher social insurance taxes can lead to higher steady state lifetime welfare for diseased agents, though not non-diseased agents. In steady state, reducing taxes from the 2017 value of $\tau = 0.153$ is Pareto improving as long as $wapr > 2.986$. Why is this? Consider informal care time supplied by young $h_{y,t}$. Figure 5 shows that time supplied per-individual is decreasing in *wapr* though aggregate time supplied is increasing in *wapr*. At a certain threshold, the extensive margin — the total number of young people — dominates the intensive margin — the time supplied by each young person, leading to adverse welfare effects on diseased old.

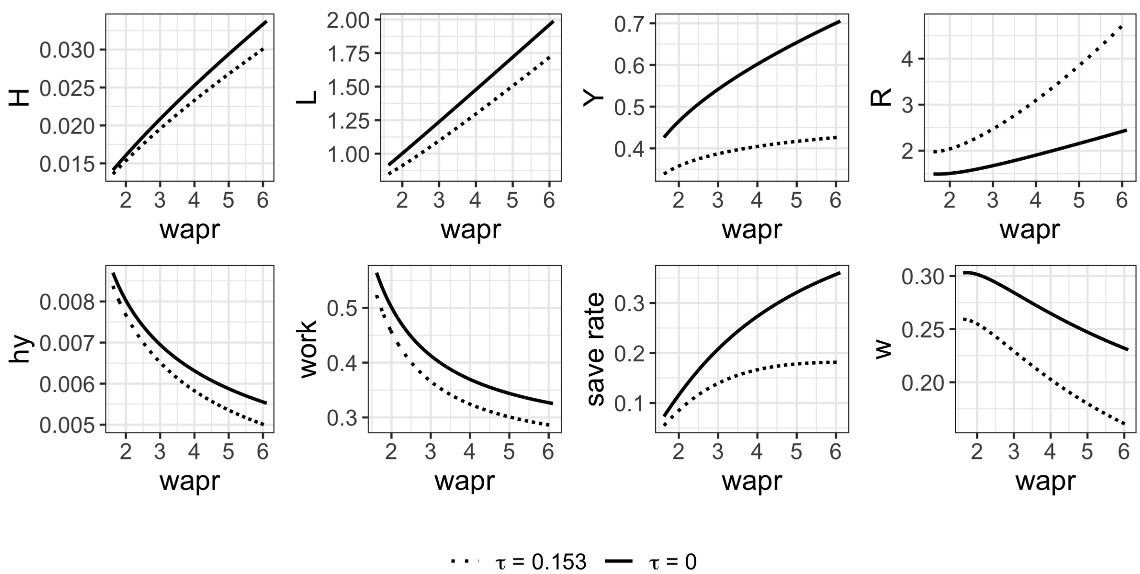

Figure 5: Here we plot steady state outcomes as a function of *wapr* and different tax rates. Solid lines represent economic variables when the government chooses $\tau = 0$, and dotted lines represent variables under $\tau = 0.153$, the 2017 Social Security and Medicare tax rate.

Notice also that diseased old utility falls faster than non-diseased utility as *wapr* decreases. This is because the decline in the extensive margin drives down total off-market time supplied by young agents as *wapr* falls, even though every individual young agent is supplying more informal care time on the intensive margin. Meanwhile, as *wapr* increases, diseased lifetime utility increases faster than non-diseased lifetime utility as the total supply of informal care time increases, allowing diseased agents to supplement their market consumption with increasing amounts of care from their children. Since these are steady state comparisons only, they should be interpreted with caution as such analyses fail to account for productivity gains. We present them to illustrate the co-dependence of lifetime welfare on both $wapr$ and τ.

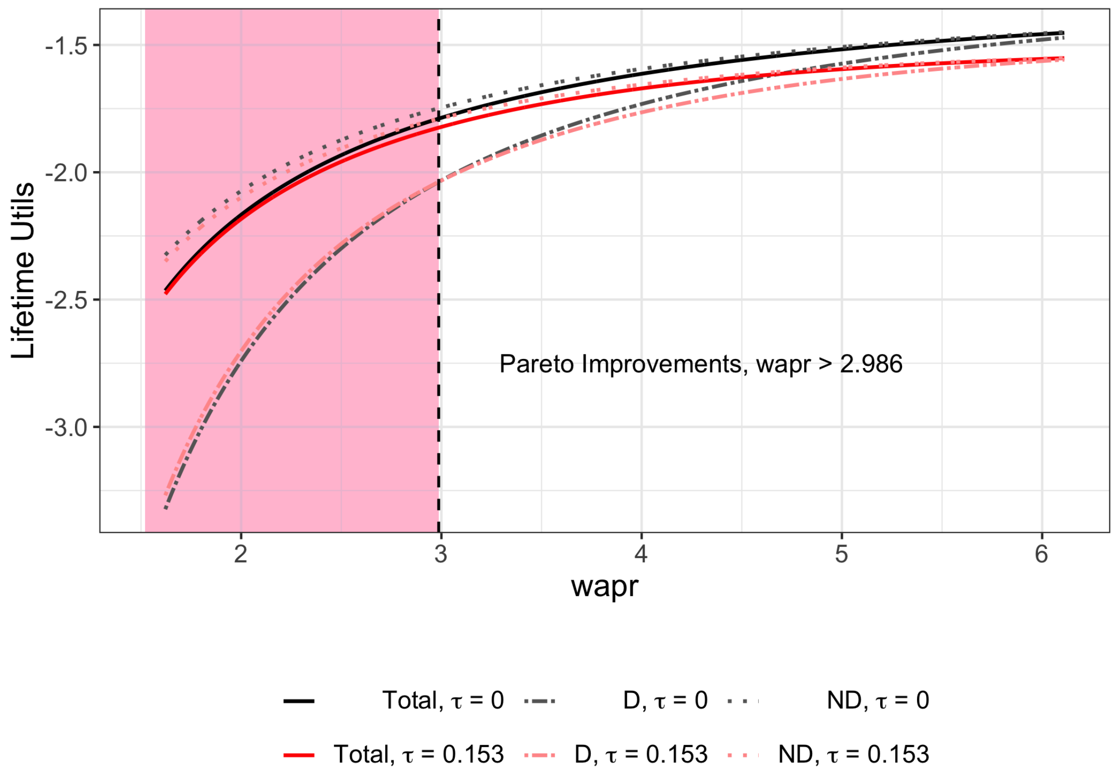

Figure 6: For all $wapr \leq 2.986$ — in the pink box to the left of the dashed vertical line — diseased agents are worse off with $\tau = 0$ than under the baseline 2017 tax rate.

5.3 Monopolistic Pricing Power

Now assume $\zeta_t > 0$, so that a market for DR_t exists. For our final quantitative exercises we do two things. First, we simulate a dynamic transition path from the steady state of an economy with a monopolistic producer of drugs to an economy where the exclusive patent rights have expired and a continuum of identical firms are engaging in perfect competition for customers. Second, we consider how demand for dr_y affects the equilibrium risk rate ψ in a competitive steady state as the population distribution and relative productivity of making DR versus X varies.

Given no risk-reducing treatments for diseases like Alzheimer's and dementia currently exist, it is impossible to know how effective such treatments will be if and when they are found. Thus, calibrating the parameters associated with the functions $\psi_{t+1}(dr_{y,t})$ and $\xi_{t+1}\big(K(DR)_t\big)$ requires some seemingly arbitrary choices. After the cure has been found, the values of $\overline{\xi}$ and φ which enter into $\xi_{t+1}\big(K(DR)_t\big)$ have no leverage over equilibrium outcomes. Yet the value of ϵ in $\psi_{t+1}(dr_{y,t})$ is still meaningful. In Figures 7 and 8 we simulate different competitive steady state values of $\psi(dr_y)$ under different values of ϵ over grids of $wapr$ and relative productivities, ζ/z. Under our current parameterization, ψ does not appear to change much as $wapr$ or ζ/z increases. Smaller ϵ ensure a faster decline of ψ as ζ/z increases and $wapr$ falls. It is interesting to note that risk falls as $wapr$ falls, as in Figure 7. This suggests that consumption smoothing motives outweigh welfare risk as $wapr$ increases. This is because higher $wapr$ means more young people to care for welfare-debilitated old, so consumers are willing to shoulder a higher disease risk knowing somebody will be there to spend time caring for them, illustrating how the two insurance mechanisms in the model interact with each other.

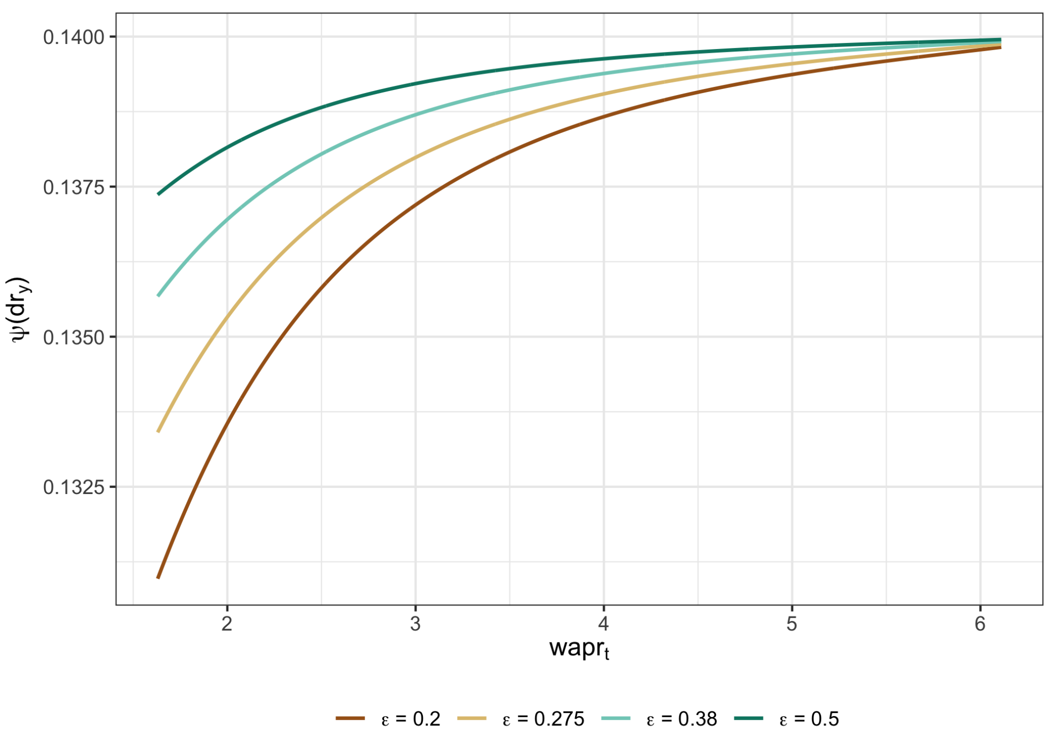

Figure 7: Competitive steady state values of ψ increase as $wapr$ increases, illustrating how the reverse altruism insurance mechanism helps offset disease risk when there are more young people in the economy.

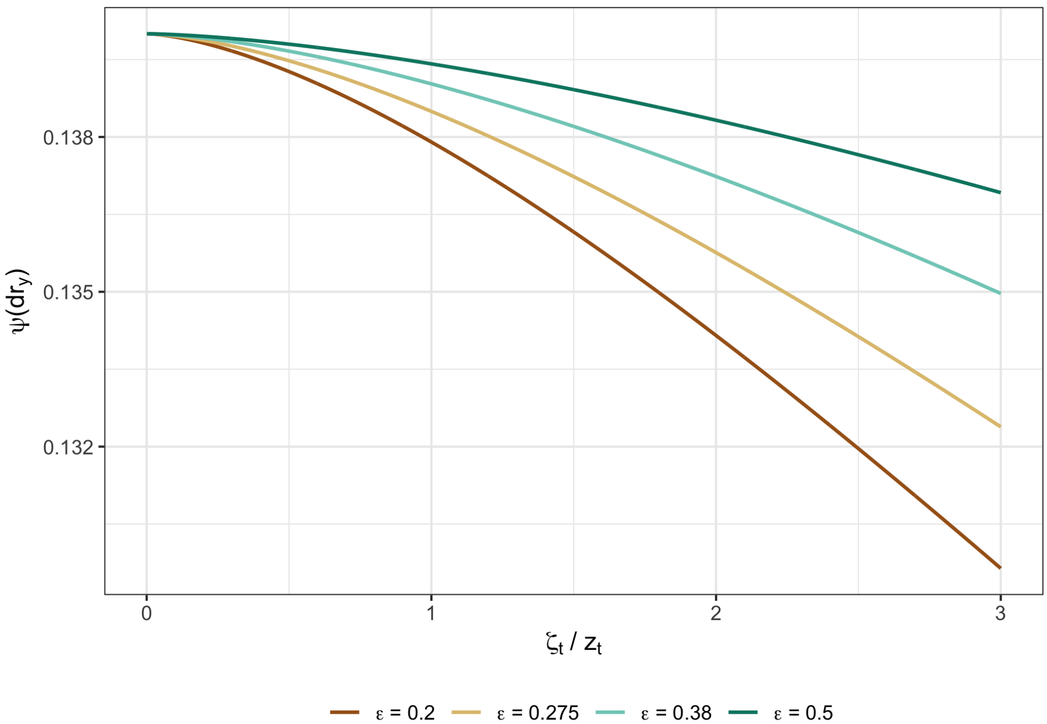

Figure 8: Naturally as relative productivity rises, more DR_t can be produced for every input of capital, sending ψ down.

To understand how the model's dynamics function, we simulate a transition path from a steady state with a monopolistic supplier to one with a competitive equilibrium. For this exercise, ϵ is fixed at $\epsilon = 0.2$. In Figure 9 we show that competition increases welfare for diseased agents by almost 4% in the period immediately following the monopolist's market domination (i.e., when the patent expires). Welfare increases for all agents in fact, even the non-diseased who still demand some quantity of $dr_{y,t}$ when young and can purchase it at a cheaper price due to market competition. The supply of DR_t increases by 163% in the initial period before settling in at 159% of the monopoly level after competition is well established. This results in a 35 basis point reduction in disease risk under the parameters we feed the model.

Future work should examine how competition in the $cure_t = 0$ state increases the probability a potential treatment is achieved. The difficulty in doing this lies in the fact that one cannot observe the probability a treatment is achieved when no such treatments have ever been achieved. There is thus no way to know how the probability changes as $K(DR)_t$ increases. Preliminary steady state analysis suggests that equilibrium outcomes are extra sensitive to the choices of $\overline{\xi}$ and φ. Future work will thus examine how these features of the model influence potential market outcomes, providing possible insights as to how much innovations that lead to treatments will truly reduce disease incidence.

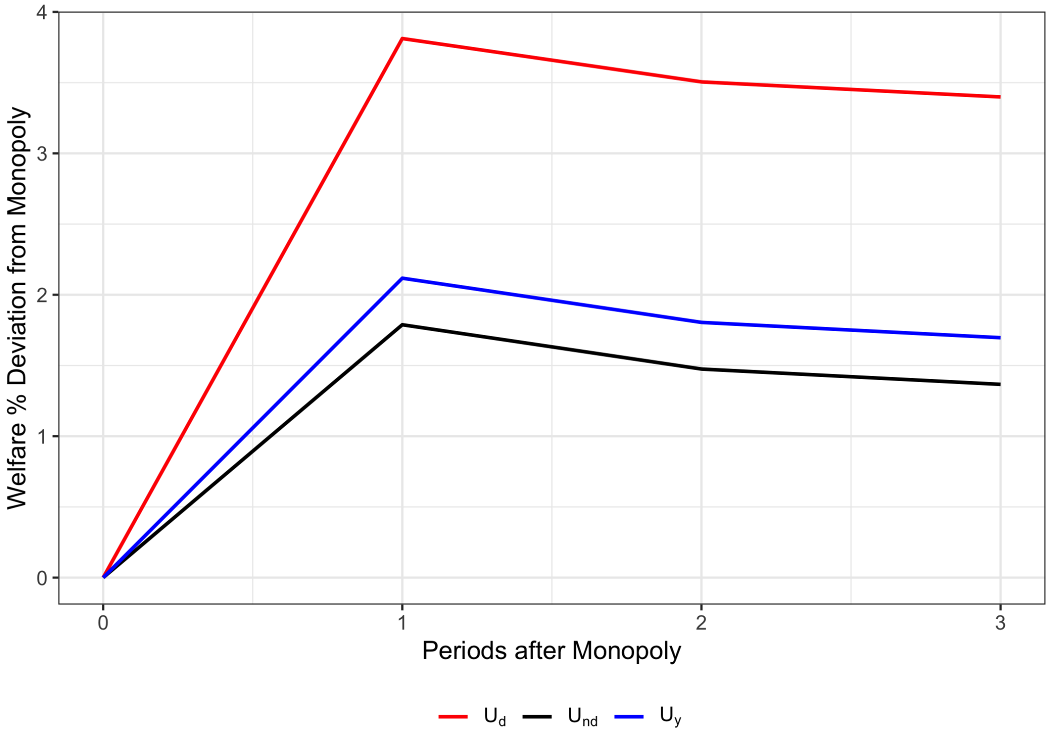

Figure 9: Competition increases welfare for all types of agents, including the non-diseased who still are able to buy more $dr_{y,t}$ when young and reduce their ex-ante risk rate.

6 Conclusion

Including both idiosyncratic health risk and a motive for young people to engage in informal care of their elders allows the standard, two-period overlapping generations model with production and social insurance taxes to broadly describe the observed decline in aggregate output growth since the 1950s. The model we present qualitatively describes and matches the observed tradeoffs from the ATUS data that agents face when making a decision to provide time on the informal market. These results are important because they should encourage researchers to take seriously the model's predictions about future economic outcomes in an environment with a rapidly aging population. Due to incomplete markets, the rate at which the population ages can adversely impact lifetime welfare of diseased agents when not enough young agents are alive to supply informal care. In counterfactual simulations, reimbursement of informal care time and reductions in the incidence of dementia-like diseases improve both growth and welfare over the

U.N.'s medium-variant projected population distribution throughout the 21^{st} century. These results should encourage policy makers to consider how the age-distribution and idiosyncratic risk affect economic aggregates when proposing reforms to address stagnating growth. Aging appears to have broad impacts on long-run GDP growth, regardless of old-age disease risk. Future work will seek to better understand the costs associated with developing treatments for these diseases, and whether the demand for such treatments will lead to significant growth and welfare improvements.

A Data

A.1 GDP Growth and $wapr$

To construct the gross domestic product (GDP) growth rate series and GDP per worker growth rate series, we use the annual-frequency, seasonally-adjusted real GDP time series in billions of chained 2012 dollars from the National Income and Product Accounts (**bea**). We then use the United Nations population data to define the normalized working-age population level $N_{y,t}$ in terms of working-age adults per 2017 old person:

$$N_{y,t} = \frac{\text{Observed Population of Adults Age } 25 - 65_t}{\text{Observed Population of Adults Age} > 65_{2017}} \tag{A.1}$$

This implies that $N_{y,2017} = wapr_{2017}$, facilitating ease of calibration. Denote GDP by Y_t. GDP per worker is then $Y_t/N_{y,t}$. We can then define annual growth rates as:

$$g_{Y_t} = \frac{Y_t}{Y_{t-1}} \tag{A.2}$$

$$g_{Y_t/N_{y,t}} = g_{Y_t}\frac{N_{y,t-1}}{N_{y,t}} \tag{A.3}$$

To get the time series in Figure 1, we apply the HP filter with smoothing set at $\lambda = 400$ to the sequences $\{g_{Y_t}\}_{t=1951}^{2017}$, $\{g_{Y_t/N_{y,t}}\}_{t=1951}^{2017}$ and remove the cyclical component following Hodrick and Prescott (1997).

Constructing the working age population ratio $wapr_t$ is simple. We use the 2017 revision of the World Population Prospects from the United Nations: Department of Economic and Social Affairs, specifically the table denoted Age Composition: Population by Age Groups – Both Sexes (United Nations: Department of Economic and Social Affairs 2017). Population is reported for five-year age bins, starting at $0 - 4$ and extending to $95 - 99$ and $100+$ for each year from 1950 to 2015.[29] For years 2015 to 2017, we use the "medium variant" table which projects the population forward up to the year 2100 using advanced demographic analysis (see United Nations: Department of Economic and Social Affairs (2017)). This is the series we also use for the future population distribution in our calibrations. In each year, the working age population ratio is

$$wapr_t = \frac{\text{Sum of Age Bins } 25 - 29 \text{ thru } 60 - 64_t}{\text{Sum of Age Bins } 65 - 69 \text{ to End}_t} \tag{A.4}$$

[29]Prior to 1990, age bins are truncated at $75 - 79$ with everyone over the age of 80 collected into an 80+

We construct this series for all years $1950 - 2100$.

To estimate the elasticities of GDP and GDP per-worker growth with respect to $wapr$, we regress the natural logs of the HP filtered series with cycles removed for g_{Y_t} and $g_{Y_t/N_{y,t}}$ on the natural log of $wapr_t$. Table 6 presents the results of these regressions.

Table 6: Elasticity of Growth w.r.t. $wapr$

	Dependent variable:	
	$\ln(g_{Y_t})$	$\ln(g_{Y_t/N_{y,t}})$
	(1)	(2)
$\ln(wapr)$	1.372^{***}	2.269^{***}
	(0.171)	(0.218)
Constant	-5.511^{***}	-7.326^{***}
	(0.254)	(0.323)
Observations	68	68
R^2	0.492	0.622
Adjusted R^2	0.485	0.616
Residual Std. Error (df = 66)	0.181	0.230
F Statistic (df = 1; 66)	64.017^{***}	108.584^{***}
Note:	*p<0.1; **p<0.05; ***p<0.01	

B Proofs

Lemma 1. For all $\nu > 0$, Assumption 1 holds.

Proof. This proof is trivial, but requires the assumption that $c > \nu$ so that Inada conditions are satisfied and c is thus feasible. Under that assumption, clearly $\ln(c - \nu) < \ln c$. □

Lemma 2. If $0 < \nu < c_{ot}(1) - c_{ot}(0)$ then the ratio of the marginal utility of non-diseased consumption to diseased consumption is such that $MU_{ot}(0)/MU_{ot}(1) > 1$.

Proof. Assume Inada conditions are satisfied such that $c_{ot}(1) > \nu$ and $c_{ot}(0) > 0$. Given

age bin. This does not impact our analysis.

Lemma 1, $\nu > 0$. Note that:

$$MU_{ot}(1) = \frac{1}{c_{ot}(1) - \nu} \tag{B.1}$$

$$MU_{ot}(0) = \frac{1}{c_{ot}(0)} \tag{B.2}$$

Rearranging the inequality in the premise we get

$$c_{ot}(0) - \nu < c_{ot}(0) < c_{ot}(1) - \nu \tag{B.3}$$

$$\Rightarrow \quad \frac{1}{c_{ot}(0)} > \frac{1}{c_{ot}(1) - \nu} \tag{B.4}$$

□

Proposition 1. When both the market for $X_t + I_t$ and the market for DR_t are perfectly competitive and $\alpha = \kappa$

$$r_t = K_t^{\alpha-1}\left[\left(\frac{1 - sub_t}{\zeta_t p_t \alpha}\right)^{\frac{1}{\alpha-1}} + \left(\frac{1}{z_t \alpha}\right)^{\frac{1}{\alpha-1}} L_t\right]^{1-\alpha} \tag{B.5}$$

Proof. Invert the marginal products of capital to get:

$$K(DR)_t = \left(\frac{r_t(1 - sub_t)}{\zeta_t p_t \kappa}\right)^{\frac{1}{\kappa-1}} \tag{B.6}$$

$$K(X)_t = \left(\frac{r_t}{z_t \alpha}\right)^{\frac{1}{\alpha-1}} L_t \tag{B.7}$$

Then exploit capital market clearing:

$$K_t = \left(\frac{r_t(1 - sub_t)}{\zeta_t p_t \kappa}\right)^{\frac{1}{\kappa-1}} + \left(\frac{r_t}{z_t \alpha}\right)^{\frac{1}{\alpha-1}} L_t \tag{B.8}$$

Now, use the assumption that output elasticities are identical across industries (i.e., $\alpha = \kappa$) to write

$$K_t = \left(\frac{r_t(1 - sub_t)}{\zeta_t p_t \alpha}\right)^{\frac{1}{\alpha-1}} + \left(\frac{r_t}{z_t \alpha}\right)^{\frac{1}{\alpha-1}} L_t \tag{B.9}$$

$$\Leftrightarrow \quad r_t = K_t^{\alpha-1}\left[\left(\frac{1 - sub_t}{\zeta_t p_t \alpha}\right)^{\frac{1}{\alpha-1}} + \left(\frac{1}{z_t \alpha}\right)^{\frac{1}{\alpha-1}} L_t\right]^{1-\alpha} \tag{B.10}$$

□

Proposition 2. Assume z_t grows at constant rate g_z and $N_{y,t}$ grows at constant rate g_N. Suppose $\zeta_t = 0$, so that $K(DR)_t = 0$. Then a competitive equilibrium depends only on $wapr_t$, not the population levels.

Proof. First, note that $K(X)_t = K_t = N_{y,t-1} \cdot a_{y,t} = \frac{N_{y,t}}{1+g_N} \cdot a_{y,t}$. Prices w_t and R_t can then be written solely as a function of growth rates and individual choices $a_{y,t}$, $l_{y,t}$, and $h_{y,t}$. Now, using (18) and the normalization $z_0 = (1+g_N)^\alpha$, we can write the aggregate resource constraint

$$\begin{aligned} N_{y,t} \cdot x_{y,t} + N_{o,t}\big(\psi \cdot x_{o,t}(1) + (1-\psi) \cdot x_{o,t}(0)\big) + N_{y,t} \cdot a_{y,t+1} \\ \leq (1-\delta)\frac{N_{y,t}}{1+g_N} a_{y,t} + (1+g_z)^t \cdot N_{y,t} \cdot a_{y,t}^\alpha (1 - l_{y,t} + h_{y,t})^{1-\alpha} \end{aligned} \tag{B.11}$$

Dividing both sides by $N_{o,t}$, we can write

$$\begin{aligned} wapr_t \cdot x_{y,t} + \psi \cdot x_{o,t}(1) + (1-\psi) \cdot x_{o,t}(0) + wapr_t \cdot a_{y,t+1} \\ \leq (1-\delta)\frac{wapr_t}{1+g_N} a_{y,t} + (1+g_z)^t \cdot wapr_t \cdot a_{y,t}^\alpha \cdot (1 - l_{y,t} + h_{y,t})^{1-\alpha} \end{aligned} \tag{B.12}$$

In (10), under constant g_N, $b_{y,t+1}$ can be written

$$b_{y,t+1} = a_{y,t+1} \cdot (1 - s_o) \cdot \frac{1}{1+g_N} \tag{B.13}$$

In (8) $h_{y,t}$ can be written

$$h_{y,t} = \frac{\psi}{wapr_t} h_{o,t} \tag{B.14}$$

The government budget constraint in (B.15) can be written

$$\big(\psi \cdot \rho_t \cdot T_t(0) + (1-\psi) T_t(0)\big) \leq wapr_t \cdot w_t \cdot \tau_t \cdot (1 - l_{y,t} - h_{y,t}) \tag{B.15}$$

Finally, young household policies must satisfy (12) thru (14). Population levels only enter (14), which can be written

$$\frac{\mu}{l_{y,t}} = \eta \cdot \frac{\sigma}{x_{o,t}(1)^{1-\sigma} h_{o,t}^\sigma - \nu} \left(\frac{h_{o,t}}{x_{o,t}(1)}\right)^{\sigma-1} \frac{wapr_t}{\psi} \tag{B.16}$$

□

Corollary 1. Assume z_t grows at constant rate g_z and $N_{y,t}$ grows at constant rate g_N. Suppose $\zeta_t = 0$, so that $K(DR)_t = 0$. Then aggregate growth $g_{Y,t}$ depends only on $wapr_t$, not population levels.

Proof. Define the period t gross output growth rate as $(1 + g_{Y,t}) = \frac{Y_t}{Y_{t-1}}$. Note that

$$Y_t = N_{yt} \cdot (1 + g_z)^t \cdot a_{yt}^{\alpha}(1 - l_{y,t} - h_{y,t})^{1-\alpha} \tag{B.17}$$

so

$$(1 + g_{Y,t}) = \frac{Y_t}{Y_{t-1}} = (1 + g_N)(1 + g_z)\left(\frac{a_{yt}}{a_{y,t-1}}\right)^{\alpha}\left(\frac{1 - l_{y,t} - h_{y,t}}{1 - l_{y,t-1} - h_{y,t-1}}\right)^{1-\alpha} \tag{B.18}$$

In Proposition 1 we showed that household policies depend only on $wapr_t$. Thus $g_{Y,t}$ depends only on $wapr_t$. □

Proposition 3. Assume z_t grows at constant rate g_z and $N_{y,t}$ grows at constant rate g_N. Suppose $\zeta_t = 0$, so that $K(DR)_t = 0$. Assume the survival rate $s_{o,t+1} = s_o$. Then along a BGP the working-age population ratio $wapr$ is constant and given by

$$wapr = \frac{1 + g_N}{s_o} \tag{B.19}$$

Proof. The population of young agents entering the economy in period t is

$$N_{y,t} = (1 + g_N)N_{y,t-1} \tag{B.20}$$

The population of old agents evolves according to

$$N_{o,t} = s_o N_{y,t-1} \tag{B.21}$$

Substituting for $N_{y,t-1}$ we can write:

$$\frac{N_{y,t}}{N_{o,t}} = \frac{1 + g_N}{s_o} \tag{B.22}$$

Note that $\frac{N_{y,t}}{N_{o,t}}$ is the working-age population ratio $wapr$. The right-hand side of the above does not depend on t. Thus:

$$wapr = \frac{1 + g_N}{s_o} \tag{B.23}$$

$\square$

B.1 Solving for $\frac{\partial \psi_{t+1}(dr_{y,t})}{\partial dr_{y,t}}$

Here, we derive $\frac{\partial \psi_{t+1}(dr_{y,t})}{\partial dr_{y,t}}$. First, rewrite $\psi_{t+1}(dr_{y,t})$ and take logs as follows:

$$\psi_{t+1}(dr_{y,t}) = \frac{1}{1+\overline{\psi}\epsilon^{-dr_{y,t}}} \tag{B.24}$$

$$\Leftrightarrow \quad \psi_{t+1}(dr_{y,t})\left[1+\overline{\psi}\epsilon^{-dr_{y,t}}\right] = 1 \tag{B.25}$$

$$\Leftrightarrow \quad \ln\overline{\psi} - dr_{y,t}\ln\epsilon = \ln\left[1-\psi_{t+1}(dr_{y,t})\right] - \ln\psi_{t+1}(dr_{y,t}) \tag{B.26}$$

Implicitly differentiate (B.26) as follows, letting ψ'_{t+1} be the derivative of ψ_{t+1} with respect to $dr_{y,t}$:

$$-\ln\epsilon = \frac{-\psi'_{t+1}}{1-\psi_{t+1}} - \frac{\psi'_{t+1}}{\psi_{t+1}} \tag{B.27}$$

$$\Leftrightarrow \quad \frac{\partial\psi_{t+1}(dr_{y,t})}{\partial dr_{y,t}} = \ln\epsilon\left[\psi_{t+1}(1-\psi_{t+1})\right] \tag{B.28}$$

$$\Leftrightarrow \quad \frac{\partial\psi_{t+1}(dr_{y,t})}{\partial dr_{y,t}} = \frac{\ln(\epsilon)\overline{\psi}\epsilon^{-dr_{y,t}}}{(1+\overline{\psi}\epsilon^{-dr_{y,t}})^2} \tag{B.29}$$

C Calibration

C.1 Setting ρ — Ratio of Diseased to Non-Diseased Benefits

We calibrate ρ by using estimates from Hurd et al. (2013) and Mommaerts (2016). Mommaerts (2016) uses RAND Health and Retirement Study (HRS) data to estimate median permanent income (\$14,157 in 2010 dollars) of sample respondents over age 65 from 1998-2010. We then use the Social Security Administration's rule of thumb permanent income replacement rate (0.4) to compute the implied Social Security benefits for the median retiree:

$$14,157 \cdot 0.4 = 5,662.80 \tag{C.1}$$

Using HRS data, Hurd et al. (2013) estimates that average total annual Medicare spending for demented individuals is \$5,226. To compute a baseline for total benefits received by diseased agents we add \$5,226 to Equation (C.1) to get \$10,888.80. The steady state ratio of diseased to non-diseased benefits is:

$$\rho = \frac{10,888.80}{5,662.80} = 1.923 \tag{C.2}$$

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