# Power of Personalized Smoking Cessation:

# A Quantitative Lifecycle Framework for Policy Evaluation

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**Abstract**

Increasing medical evidence suggests that smokers' responsiveness to medication treatment depends critically on genotypes, but it remains unexplored whether personalized treatment in smoking cessation is cost-effective. We address this knowledge gap by developing a lifecycle model with various dimensions of heterogeneities in genotypes/demographics and behavioral considerations, where health capital evolution and life expectancy are endogenously determined, depending on individuals' smoking, health investment and consumption-savings decisions. We calibrate the model and evaluate three smoking cessation policies. We find personalized treatment the most cost-effective: every dollar of program cost generates $4.59 value in effectiveness, 21-44% higher than those under standard treatments.

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# 1 Introduction

Cigarette smoking is a leading preventable threat for global public health. Economic and medical evidence have shown large healthcare costs, morbidity (productivity) costs, and mortality losses associated with smoking, where the estimated total cost of smoking and value of life lost per death are sizable.[1] Thus, smoking cessation can play an important role in reducing the increased health risk and economic costs mentioned above. Recent medical research shows that use of genetic markers in smoking cessation treatments has great promise for predicting increased risk for developing nicotine dependence, delayed smoking cessation, and the likelihood to respond to medication treatment when receiving treatments. While pharmacogenetic testing may allow physicians to personalize treatments based on an individual's genetic factors to improve success rates in smoking cessation (rather than on a trial and error basis), whether or not to implement this method in the clinic requires a thorough cost-effectiveness analysis, which remains unexplored in both economics and medicine literature. Our paper is devoted to addressing this important issue concerning cost-effectiveness of personalized medicine treatment in smoking cessation that may yield large impact on mitigating the threat of smoking for global public health.

Concerning the usefulness of genetic markers in medical treatments, it has been documented in medical studies that different pharmacokinetic and pharmacodynamic markers have been implicated in personalized therapeutic approaches including nicotinic acetylcholine receptor genes ($nAChRs$).[2] To better understand smokers' quitting behavior and medical treatment responses, we focus on three common haplotypes spanning the *CHRNA5* gene region: H1, H2, H3. Chen et al. (2012) identified the usefulness of *CHRNA5* genetic markers in predicting both cessation difficulty and treatment response.[3] While the high-risk haplotype (H3) is unequivocally associated with heavy

[1] As reported in Max et al. (2004), in California in 1999, such cost was $3,331 per smoker, the value of life lost averaging $132,000 or 12.4 years. In particular, healthcare costs for smokers at a given age are as much as 40 percent higher than those for nonsmokers in the short term (Barendregt et al., 1997). Furthermore, it is evident that the costs of smoking are higher when including the negative external effects associated with second-hand smoking (Pickett et al., 2006). The reader is referred to Economics of Tobacco Toolkit (WHO, 2011) for a comprehensive review of methods of estimating the economic costs of smoking.

[2] That personalized medication is promising has been well-documented by Kortmann et al. (2010), Sturgess et al. (2011), Chen and Bierut (2013), and Bough et al. (2014), to name but a few. In particular, variants in the genes encoding the $\alpha 5 - \alpha 3 - \beta 4$ nicotinic receptor subunits are key drivers (Chen et al., 2015a).

[3] It is based on data from smoking cessation trials conducted at the University of Wisconsin Transdisciplinary Tobacco Use Research Center. The results have been replicated by Bergen et al. (2013) and Chen et al. (2015b).

smoking and most difficulty in smoking cessation, smokers of this type respond well to medication. In contrast, smokers with the low-risk haplotype (H1), though smoke less on average and are more likely to quit with counseling, do not benefit from medication. Based on this evidence, personalized medicine in smoking cessation means identifying a smoker's genotype with a genetic test first and then providing medication only to those who will most likely be responsive (i.e., the H2 and H3 types) based on the test result.

To evaluate the cost-effectiveness of personalized medicine treatment in smoking cessation, we investigate, under the same cost, what outcome differences are between personalized treatment and non-personalized treatments. To undertake this task, we develop a lifecycle model in which key decisions on smoking, health and savings as well as life expectancy are all endogenously determined. We take into account the three main types of costs of smoking as in the literature. On the one hand, smoking accelerates health depreciation, thereby increasing demand for medical care (healthcare cost) and reducing labor income (morbidity cost). On the other hand, worse health results in lower life expectancy (mortality cost). We build upon the health capital framework (e.g., the pivotal work by Grossman 1972a,b) and the health and life expectancy framework (pioneered by Rosen 1988 and Ehrlich and Chuma 1990 and further advanced by Jones 2016).

To justify why policies matter, we highlight an important behavioral aspect of the addict, namely, peer, home and workplace influences, including most critically adolescent smoking when the young need not behave fully rationally.[4] For example, adolescent smoking may be due to teenage stress from family conflict and/or school work or any other forms of anger, anxiety and depression, teenage curiosity and rebelliousness, as well as the belief that smoking reflects a cool image or a mature appearance.[5] Due to such influences, one may become a smoker; after being addicted to smoking, he/she may not be able to quit despite lacking full enjoyment. Thus, incentivizing a grown-up addict to quit smoking is not only social but also individual welfare improving. Moreover, even in adulthood, incomplete health knowledge and bounded rationality in decision making may also lead to excessive smoking that may be corrected by publicly provided cessation programs. Furthermore, the effectiveness of such policies are even greater should one takes into account second-hand smoking that harm everyone including nonsmokers.

Our model is richer than previous settings not only because we endogenize consumption-saving,

[4] See, for example, Kelder et al. (1994) for medical evidence on such behavior concerning physical activities, food preferences and most importantly smoking and Cutler and Glaeser (2010) for economic study on smoking.

[5] See, for instance, critical reviews by Tyas and Pederson (1998) and Kobus (2003).

health investment, smoking and life expectancy and incorporate various important behavioral considerations of the addict, but also because we permit multi-dimensional heterogeneities. In addition to the key heterogeneity in genotypes stressed by the medical literature, we also allow for three crucial dimensions of heterogeneities in demographics: gender, race and education, which can influence smoking behavior as identified by many health economists (see a critical review by Chaloupka and Warner, 2000). The main advantage of our approach for policy analysis is that we take into account individuals' endogenous responses over their dynamic lifecycles, depending on their demographics as well as genotypes. This enables us to evaluate various smoking cessation policies by aggregating individual-specific effects in a systematic manner. Moreover, for policy relevance, we also take into account the negative externality associated with second-hand smoking, incomplete knowledge in the detrimental health effect of smoking, and bounded rationality in decision making.

We quantify our model by calibrating it to fit key observations from the U.S. data. The set of targets includes economics data such as income, various expenditure ratios, and life expectancy. It also consists of some medical experiment data such as gene-dependent smoking by Chen et al. (2012) and differential gains in life years from smoking cessation by Jha, et al. (2013). Our calibrated model predicts well that life expectancy of smokers is 12 years shorter than nonsmokers and that the high-risk H3-type smokers live slightly shorter than other types, consistent with medical evidence. Our computed potential gain in life years for smokers quitting successfully at age 35 varies from 9.18 to 10.03 years for the three genotypes.

We further propose four separate measures of effectiveness for three smoking cessation policies with the same program costs, two with standard and one with personalized treatments. The first two measures are the policy coverage rates given the same budget and the resulting quit rates. The third is Consumption Equivalent of a covered smoker, which is the percentage increase in consumption (at each point in time) that is needed, while fixing everything else unchanged, to reach the level of lifetime utility when being covered by a cessation program. The last is Income Equivalent of a covered smoker, defined in the same way but in terms of the percentage increase in labor income. These equivalent measures have taken into account all private benefits and costs associated with participation in a program. We further construct three unified measures, including an expected Consumption Equivalent measure of a smoker, an expected Income Equivalent measure and a cost/effectiveness ratio based on expected Income Equivalent of a smoker. We find that, under the same program costs, personalized treatment provides coverages for more and maintains high effectiveness. The expected value of statistical life (VSL) gain under personalized treatment is

$16,894 per smoker or a 0.93% increase from its benchmark value, much higher than the comparable figures under standard treatments ($13,922 and $11,745 or 0.77% and 0.65%, respectively). For every dollar of the program cost, it generates 4.59 dollars Income Equivalent in effectiveness, $21-44$ percent higher than the comparable figures under standard treatments. That is, personalized treatment is the most cost-effective in smoking cessation.

Furthermore, our counterfactual analyses show critical roles played by behavioral bias and negative externality associated with second-hand smoking in policy effectiveness. While incomplete health knowledge about smoking and bounded rationality in decision making elevate policy effectiveness by 50 and 15 percent, respectively, the exposure to second-hand smoke turns out to mitigate individual smokers' benefit from policy intervention substantively. The latter is because an individual would still suffer detrimental second-hand smoke even if she herself quits, dampening policy effectiveness. Thus, our results suggest the importance of expanding policy coverage to bring down society-wide level of smoking, enhancing policy effectiveness on individual smokers as well as nonsmokers.

By extending our benchmark model to various settings concerning no relapses, quitting at age 50, no quitting delays, and net deadweight cost of medical treatment, we verify that our main finding remains robust. For example, when policies treat smokers at age 50, every dollar of the program cost generates 2.70 dollars Income Equivalent in effectiveness under the personalized treatment policy, which is about 40% lower than the benchmark value where smokers are treated at 35. In all cases of our extensions, personalized treatment is $21-46$ percent more effective than standard treatments.

To this end, it is useful to stress that our effectiveness measures are more general than quality adjusted life years (QALYs) commonly used in the medical and public health literature. Specifically, our lifetime utility based effectiveness measures account for time discounting, diminishing marginal valuation and differential utility weights. More importantly, our measures incorporate heterogeneous individuals' dynamic responses that enable us to capture different responses across different individuals at different ages over their life course. While the purpose of the present paper is to evaluate smoking cessation policies, our theoretical framework and quantitative method may be readily applied to studying other types of addictive behavior, such as alcohol and illicit drugs, as well as to evaluating other types of precision medicine in treating various diseases that depend on genotypes.

## 2 Background and Evidence

Before setting up the model, we briefly describe the economics literature relating various dimensions of heterogeneities to smoking outcomes. We then summarize some of the key facts and medical evidence regarding the smoking population and cessation probability of smokers with different genotypes and demographics, using the medical experiment data collected by one of the coauthors of the present paper (Chen) to facilitate the study of Chen et al. (2012), that will be adopted in our quantitative analysis. We next briefly introduce the methods of smoking cessation treatment.

### 2.1 On Heterogeneous Genotypes and Demographics and Smoking

With regard to heterogeneities in demographics, it is found that male smokers typically smoke more cigarettes per day than women, and male demand for cigarettes tends to be more price elastic than is female demand (Giovino et al., 1994; Chaloupka, 1990; Chaloupka and Grossman, 1996). Moreover, blacks are less likely to smoke and smoke less than whites (Chaloupka and Grossman, 1996). Research has also shown a substantially positive effect of education on health (Kenkel, 1991; Grossman and Kaestner, 1997; Lleras-Muney, 2005) and that higher educated smokers are more likely to quit smoking (Chaloupkia, 1991). Furthermore, age is positively related to cigarettes smoked per day, or, CPD in short (see, e.g., Giovino et al., 1994). Finally, Adda and Cornaglia (2006 and 2010) use data on the concentration of cotinine, a biomarker affecting the enzyme *CYP2A6* activity that predicts exposure to tobacco smoking. With such a better measure of smoking intensity, the former paper (2006) shows addicts may raise smoking intensity in response to higher cigarette taxes in contrast to the conventional predictions, the latter (2010) finds adverse effect of smoking bans on passive (second-hand) smokers.

We then explore the basic facts about smoking behavior based on cross-sectional data from the Panel Study of Income Dynamics (PSID; 1999-2011) that will be used for our calibration. Specifically, we look at the share of smokers, CPD, and smoking expenditure-income ratio by gender, race, education and age. The data show that the share of current smokers among the US population (whites and blacks at age above 18) is 24.6%, the average CPD among smokers is 13.75, and the average expenditure on tobacco is 6.2% of labor income among smokers and 1.5% among the whole population. While the share of smokers is similar across genders and races, it does vary substantially across education groups. About 30% of the population without a college degree are currently smoking, while this figure is only about 10% for those with a college degree.

From Table 1 (column 4) which shows the share of smokers by eight demographic groups based on gender ($M = 1$ for male and $= 0$ for female), race ($W = 1$ for whites and $= 0$ for blacks) and education ($C = 1$ for college educated and $= 0$ for non-college educated), the group that has the largest share of smokers are non-college educated female whites (about 40%), followed by two non-college male groups (black and white, for which the share of smokers is about 30%). Regarding the level of smoking measured by CPD, it is noted that males smoke more than females on average (14.8 versus 11.1), whites smoke more than blacks (15.9 versus 9.8), and the non-college educated smoke more than college educated (14.1 versus 10.8). Moreover, the average CPD is the highest for the whites, non-college group (17.5 for males and 13.5 for females) (column 5), and the smoking expenditure to labor income ratio is highest for the non-college groups (column 6).[6]

Table 1. Smoking behavior by demographics

| M | W | C | share of smokers (%) | CPD of smokers | smoking-income ratio of smokers (%) |
|---|---|---|---|---|---|
| 0 | 0 | 0 | 23.4 | 8.76 | 8.5 |
| 0 | 1 | 0 | 39.8 | 13.53 | 7.5 |
| 0 | 0 | 1 | 11.6 | 13.24 | 5.2 |
| 0 | 1 | 1 | 12.2 | 8.57 | 3.0 |
| 1 | 0 | 0 | 31.2 | 10.37 | 6.4 |
| 1 | 1 | 0 | 29.3 | 17.52 | 6.0 |
| 1 | 0 | 1 | 15.8 | 9.42 | 1.7 |
| 1 | 1 | 1 | 8.0 | 11.47 | 2.1 |
| all | | | 24.6 | 13.75 | 6.2 |

To look into the age effect, we plot the lifecycle smoking behavior in Figure 1 using the same PSID data. While the share of smokers in the population declines with age, CPD conditional on smoking tend to increase with age. To incorporate quitting behavior in the measure of smoking intensity over the life course, we adjust CPD of smokers with the share of smokers at each age; that is, we multiply CPD of smokers at each age by the share of smokers at that age divided by the share of smokers in the whole population. We do the same adjustment for smoking expenditure

[6] In addition, we look at persistence of smoking activity using the duration of regular smoking for those who ever smoked (but are not smoking currently) from PSID, and find that on average males smoke longer than females (14.9 versus 13.4 years), whites smoke longer than blacks (14.9 versus 13.4 years), and the non-college educated smoke longer than college educated (15.0 versus 13.3 years). All the differences are statistically significant.

to labor income ratio for smokers. Both adjusted measures turn out to decline with age (see the bottom panels of Figure 1). Overall, the facts regarding smoking behavior related to gender, race, education and age from the PSID data are broadly consistent with what are documented in the literature.

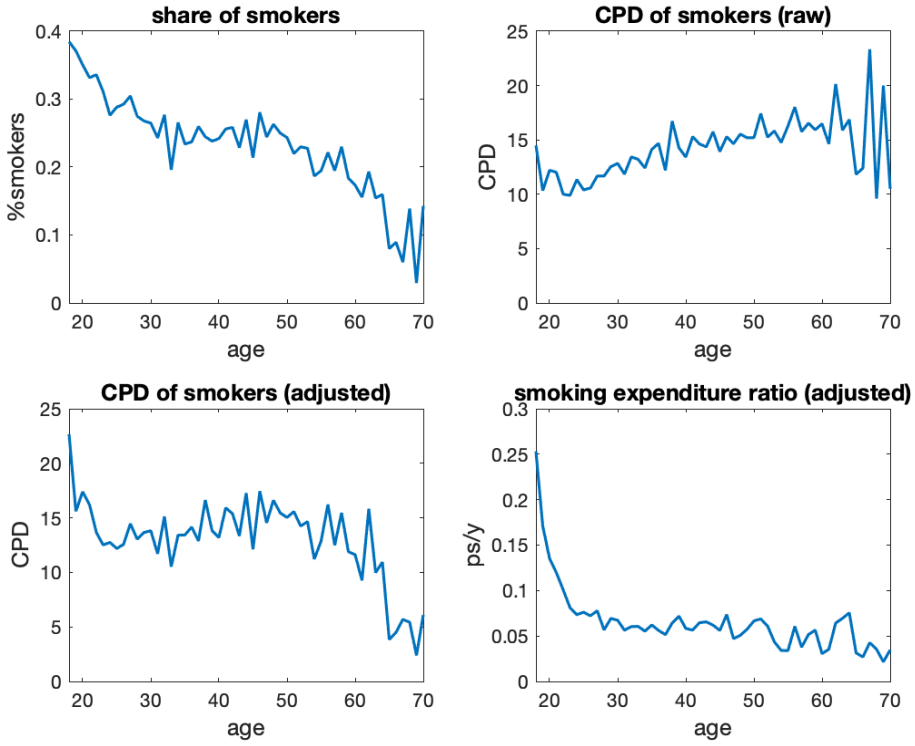


Figure 1: Lifecycle smoking behavior in PSID data

Turning now to examining the medical experiment data (Chen et al. 2012), we note that the share of H1, H2 and H3 genotypes among smokers is about 25, 44 and 31 percent, respectively. Interestingly, while the shares of three genotypes do not vary significantly across gender or education groups, they vary a great deal across racial groups. In particular, whites have a much larger share of the H3 type than blacks (35% versus 6%), and blacks have a much larger share of the H1 type than whites (51% versus 21%), while the share of the H2 type is similar for whites and blacks (about 44%) (see the top panel of Table 2).[7]

[7] The gene of our interest (*CHRNA5*) is most specific to smoking and is not studied extensively in other addiction;

Medical studies suggest that among the three genotypes, H3 smokers (the high-risk haplotype) are less able to quit, more likely to smoke longer or inhale more deeply, and more likely to have longer duration of smoking; thus, the risk in mortality is higher for H3 as a result of smoking. The bottom panel of Table 2 summarizes smoking and quitting behavior of the three genotypes in terms of CPD and quit rates in a three months' treatment of different types (without considering relapse), based on the medical experiment data mentioned above. The level of smoking measured by CPD before any treatment is about 20.7 for H1, 21.1 for H2 and 22.5 for H3. Despite belonging to the high risk group, H3 smokers are most responsive to medication treatment compared with other two genotypes. Without considering relapse, the quit rate in a three months' treatment is only about 21.4% for H3 smokers if only counseling is provided, while it increases to about 45.4% when medication is combined with counselling. This quit rate, however, is almost unchanged for H1 smokers (about 44.5% regardless of the provision of medication), and for H2 smokers, it is about 31.9% without medication and 45.4% with medication.

Moreover, different education groups vary in cessation probabilities. Conditional on the genotype and treatment method, better-educated smokers are more likely to quit. We see that for H1 smokers, the quit rate in a three months' treatment (without considering relapse) is 58.6% for the college educated, and 43.1% for the non-college educated, regardless of the type of treatment. For H2 smokers, these two rates are 43.1% (college) and 30.8% (non-college) when they receive counseling only, and increase to 61.3% (college) and 43.8% (non-college) when they receive counseling plus medication. For H3 smokers, these are 29.0% (college) and 20.7% (non-college) when they receive counseling only, and 61.3% (college) and 43.8% (non-college) when they receive both treatments.

## 2.2 On Smoking Cessation Treatment

Extensive research provides support for the effectiveness of counseling and pharmacologic interventions, alone or in combination, in increasing smoking-cessation rates among patients who are willing to attempt to quit.

Successful counseling boosts the motivation to quit by personalizing the costs and risks of the patient's tobacco use (e.g., tying it to the patient's health, economic status, and family situation). Counseling also provides an opportunity to warn the patient about obstacles or hurdles to quitting

thus, we do not explore the correlation between smoking and other addictive behavior in this study. In addition, while e-cigarettes are becoming popular, the health effects of their long term use and their relation with genotypes are yet to be researched; thus, they are also beyond the scope of this study.

and encourages the patient to plan to use coping strategies for avoiding and resisting temptations or urges to smoke. Seven medications are approved by the Food and Drug Administration (FDA) for smoking cessation to treat symptoms of nicotine dependence such as craving and withdrawal, and all of the approved medications have been shown in randomized trials to be more effective than placebo (Fiore et al., 2011; Tobacco Use and Dependence Guideline Panel, 2008).

Table 2. Population shares and Smoking/quitting behavior of 3 genotypes

| | H1 | H2 | H3 |
|---|---|---|---|
| Population shares (%) | | | |
| all | 25 | 44 | 31 |
| whites | 21 | 44 | 35 |
| blacks | 51 | 43 | 6 |
| CPD | 20.7 | 21.1 | 22.5 |
| Quit rates (%) | | | |
| counseling only | 44.5 | 31.9 | 21.4 |
| college | 58.6 | 43.1 | 29.0 |
| noncollege | 43.1 | 30.8 | 20.7 |
| counseling and medication | 44.5 | 45.4 | 45.4 |
| college | 58.6 | 61.3 | 61.3 |
| noncollege | 43.1 | 43.8 | 43.8 |

# 3 The Model

To properly model smoking behavior and its health consequences over an individual's life course, we develop a lifecycle framework with endogenously determined life expectancy. In contrast with many previous studies on endogenous life expectancy where health or life protection simply reduces mortality (e.g., Rosen, 1988; Ehrlich, 2000; Ehrlich and Yin, 2005; Jones, 2016), we allow better health to improve the quality of life and hence lifetime utility (which is in line with studies by Ehrlich and Chuma, 1990, Chakraborty, 2004, Murphy and Topel, 2006, Hall and Jones, 2007, and Chen, 2010). This addition is important because QALYs is a standard effectiveness measure in cost-effectiveness analysis in medicine and health (e.g., see the guidelines provided by Gold et al., 1996). Following the health capital literature pioneered by Grossman (1972a,b), we allow health capital to evolve endogenously over time. While smoking can be enjoyable to an addict, it has a detrimental conse-

quence for health by accelerating health deterioration. Departing from the literature, we consider heterogeneity in individuals' tastes for smoking. This is consistent with the medical literature that has identified genetic markers associated with smoking behavior (e.g. Chen et al., 2015a,b; Chen et al., 2018). Such a consideration is essential because it permits us to evaluate precision medicine with personalized treatments based on an individual's unique characteristics. Individuals are also heterogeneous in work efficiency and the natural health deterioration rate and hence their labor income and health evolution. Preference, income and health evolution heterogeneities subsequently affect their differential smoking behavior, health capital and life expectancy. Below we proceed with explaining the model setup in detail.

Time is continuous. An individual's life starts at $t_0$ when she acts as a decision-maker (who may be referred to as a young adult). Her life ends at $T$ which is endogenous and will be discussed later. Throughout her life course $[t_0, T]$, she values consumption $c$ and health $h$ and may also value smoking $s$ depending on her genotypes. The flow utility accrued at time $t \in [t_0, T]$ from such enjoyment is assumed to take a simple log linear form:

$$u(c, s, h, t) = \ln(c(t)) + \alpha \cdot I_\alpha \ln(s(t)) + \beta(t) \ln(h(t)) \tag{1}$$

where $\alpha \geq 0$ and $\beta(t) > 0$ measure, respectively, her preferences for smoking (given that she is a smoker) and health relative to general consumption; $I_\alpha$ is an indicator function which equals 1 if one is a smoker and 0 otherwise.

For simplicity, we assume $\alpha$ takes discrete values and individuals with different genetic markers will have different values of $\alpha$. Importantly, $\alpha > 0$ is a behavioral parameter that captures one's addiction to tobacco, which depends not only on genetic markers, but also on environment and social comparison in the spirit of neuroeconomics (Grygolec, Coricelli, and Rustichini, 2012; Gul and Pesendorfer, 2001; Ogaki and Tanaka, 2017). Specifically, this behavioral aspect of the addict is viewed as due to peer, home and workplace influences, particularly for adolescent smoking when a teenager (at age prior to $t_0$) faces stress from family conflict and/or school work or any other forms of anger, anxiety and depression, has curiosity or rebelliousness, or believes that smoking reflects a cool image or a mature appearance (Kobus 2003; Tyas and Pederson 1998). Under such influences, a teenager may become a smoker, being addicted to it and unable to quit despite lacking full enjoyment. Thus, while $\alpha > 0$ is the observed preference for smoking (at and after age $t_0$) due to path-dependence as a result of addiction influenced by the environment of peer, home and workplace, it would not reflect an adult's "true" taste. As such, government intervention may help

individuals to exert self-control by altering the "choice menu" in neuroeconomics through incentivizing smokers to receive cessation treatment and thus induce quitting (i.e., switching $I_\alpha$ from 1 to 0). In this way, incentivizing a grown-up addict to quit smoking is not only social but also individual welfare improving. Moreover, we note that quitting can be viewed as a Poisson jump process, and its duration, or persistence of smoking upon quitting efforts, depends on genetic markers. Specifically, smokers with the genotype of stronger tobacco addiction tend to delay cessation for a longer period.

Denote $\bar{\beta}$ as the (constant) preference parameter for a fully rational individual. We then consider that a boundedly rational individual tends to pay limited or no attention to future events and thus inprecisely values her health when making lifetime choices, as stressed by behavioral macroeconomists (e.g., Gabaix, 2017). Specifically, we propose: $\beta(t) = \bar{\beta}(1-\chi(t))$, where $\chi'(t) \leq 0$ captures the fact that individuals undervalue health when young while overvalue health when older. As to be seen below, we will model the evolution of both nonhuman wealth and health capital as well as the wage-tenure profile. Thus, to avoid further complication, we elect not to include the habits of addiction stock in the utility function.[8]

Letting $\rho$ be the subjective time discount rate, we can then write one's lifetime utility as:

$$U = \int_{t_0}^{T} e^{-\rho(t-t_0)}[\ln(c(t)) + \alpha \ln(s(t)) + \beta(t) \ln(h(t))]dt \tag{2}$$

where we have plugged in the flow utility function given in (1). Thus, health raises an individual's lifetime utility directly via two channels: higher quality of life (through $\beta(t) \ln(h(t))$) and longer life expectancy (through $T$).

Two remarks are in order. First, in order for longevity to raise lifetime utility, it is necessary to ensure the flow utility to be positive. Given the log functional form, this is met when we choose the scale of health and income properly so that their logged values are sufficiently greater than zero (to be further elaborated in Section 4.1 below). This strategy in essence follows Murphy and Topel (2006), by assuming consumption bundles to be above their "subsistence" levels (which are, in our case, $c = h = 1$ and $s = 1$ for smokers).[9] Second, although consumption and health has the same

[8] The reader is referred to Becker and Murphy (1988) for developing a habit-based rational addiction framework. This literature generates valuable insights including better understanding of the short versus long run price elasticity of addictive consumption demand, albeiting price-insensitive initiation of smoking by the youth, as documented by Decicca et al. (2002), and potential bias due to endogenous response in smoking intensity, as elaborated by Adda and Cornaglia, 2006. It is nonetheless beyond the scope of our paper.

[9] One alternative is to follow Hall and Jones (2007) and Jones (2016) to add a constant $\overline{u} > 0$ in flow utility to

income elasticity under the simple log functional form, the positive health effect on life expectancy makes health a luxury good relative to consumption, which is consistent with the literature on health and life (Hall and Jones, 2007; Jones, 2016).

Consider now an individual with work efficiency $\theta$, facing a market wage in efficiency unit denoted by $w$ (needless to say, all dollar measures are in real terms). In addition to her work efficiency, her flow labor income at time $t$ depends critically on her labor market experience as well as her health status. While the former is standard in the labor economics literature, the latter captures the labor productivity effect of health emphasized by Grossman (1972b). Regarding these as two additive components of human capital, we can now specify the labor income obtained by an individual with work efficiency $\theta$ as:

$$y(h(t),t) = \begin{cases} \theta w(t) \left[h(t) + \kappa \left(e^{\phi_1(t-t_0)+\phi_2(t-t_0)^2} - 1\right)\right] & 19 \leq t \leq 65 \\ 0.5 \cdot y(65) & t > 65 \end{cases} \tag{3}$$

In this formulation, we conveniently align the measurement of human capital with health capital, so $\kappa > 0$ captures the relative importance of experience in labor income generation. Moreover, the parameter $\phi_1 > 0$ measures the return to experience, $\phi_2$ measures the curvature of this return, and $\kappa \left(e^{\phi_1(t-t_0)+\phi_2(t-t_0)^2} - 1\right)$ captures the experience-driven wage-tenure profile that is equal to zero at time $t_0$ (when the individual is fully inexperienced). Note that $\theta$ takes different values for individuals with different gender, race and levels of education – thus, although we refer to an individual as "she" for convenience, we do allow for gender differences in earning as observed in the real world. We assume retirement age to be 65 for all individuals and set the retirement replacement ratio to be 0.5 in line with the literature.[10]

In addition to labor income, an individual can also earn income from savings, which is in forms of holding an asset $a$ that provides a yield at the market (real) interest rate $r$. This intertemporal

ensure positive values. Another is to add a constant to the log function such as:

$$u = \ln(1 + c(t)) + \alpha \ln(1 + s(t)) + \beta \ln(1 + h(t))$$

The latter setup would, however, generate extra income effects on smoking which is inconsistent with the empirical evidence (Chaloupka and Warner, 2000).

[10] Our model is essentially an endowment economy in which efficiency wage $w(t)$ is exogenously determined. If one considers a production economy instead with wages pinned down by productivity, then there are two offsetting effects on smoking and health. On the one hand, when policy causes more people to quit, labor supply increases due to improved health, which lowers equilibrium wage and the opportunity cost of smoking on health, encouraging more smoking. On the other, lowered wage generates a negative income effect which discourages smoking. The overall effect of the general equilibrium effect on policy effectiveness is ambiguous.

decision is crucial for an individual to save for the "rainy days" of old ages when health deteriorates and medical spending rises. In each point in time, her flow (labor and interest) income is allocated to consumption, cigarette purchase and health-related spending that will be referred to as health investment $x$. Given the price per unit amount of smoking $p$, an individual's asset accumulation over her life course is governed by,

$$\dot{a} = r(t)a(t) + y(h(t), t) - c(t) - p(t)s(t) - x(t) \tag{4}$$

where $\dot{a} \equiv da/dt$ and labor income is given by (3). That is, asset is accumulated when flow income $(ra + y)$ exceeds flow expenditure $(c + ps + x)$.

We next turn to the evolution of health capital where we highlight three important features. First, health can be improved with more investment in health, but such improvement is subject to diminishing returns. More specifically, given the current state of health $h$, health improvement measured by $\dot{h} \equiv dh/dt$ rises less than proportionately with either the current health status $(h)$ or health investment per unit of health capital $(x/h)$. Second, the health deterioration rate rises with the amount of smoking $(s)$ but falls with the current health status. Third, the marginal return to health investment falls with age, as documented in the life and growth literature such as Hall and Jones (2007), while the smoking effect on the health deterioration rate is rising with age, as documented in medical studies such as Jha et al. (2013). To fulfill all these features, we propose the following health evolution process:

$$\dot{h} = \left[\Phi(t)x(t)^{\epsilon} - (\delta + \gamma(t)(s(t) + q\overline{s}))\right] h(t)^{\mu} \tag{5}$$

where we also incorporate the second-hand smoking effect through $q\overline{s}$, with $\overline{s}$ reflecting the society-wide level of smoking and $q$ measuring the degree of exposure to passive smoking. To each individual, the society-wide level of smoking is taken as given.

We now check the required properties. The benefit of health investment is $\Phi x^{\epsilon} h^{\mu} = \Phi \left(x/h\right)^{\epsilon} h^{\mu+\epsilon}$, where $\Phi > 0$ measures the extent to which health investment benefits health. To have diminishing returns in the beneficial effects of current health and health investment per unit of health capital on future health evolution, it is required that $\epsilon > 0$, $\mu > 0$ and $0 < \mu + \epsilon < 1$ for all $t$. To capture diminishing returns to health investment with age we also assume $\Phi'(t) < 0$. We then examine the health deterioration rate, measured by $\left[(\delta + \gamma(t)(s + q\overline{s}))h^{\mu}\right]/h$, where $\delta > 0$ is the natural health deterioration rate which will be allowed to differ by gender in calibration. Thus, the health deterioration rate rises with smoking and falls with the current health status (because $\mu < 1$),

without additional assumptions. In order for the smoking effect on the health deterioration rate to rise with age, we further restrict that $\gamma(t)$ is an increasing function of $t$.[11] In summary, under these parametric assumptions, the above functional form offers a parsimonious setting that governs the evolution of health capital satisfying all the required properties.

We further note from the empirical health literature that many individuals have incomplete knowledge about health which affects their health-related behavior such as smoking. We thus allow individuals' belief in $\gamma(t)$ to be different from its true value. Based on the evidence in Kenkel (1991), one's belief in the detrimental health effect of smoking at age $t$, denoted $\widetilde{\gamma}(t)$, is generally lower than its true value, i.e., $\widetilde{\gamma}(t) < \gamma(t)$ . Given the lack of health knowledge, individuals may form wrong belief in their health status, so their "believed" health evolution process is given by

$$\dot{\widetilde{h}} = \left[\Phi(t)x(t)^{\epsilon} - (\delta + \widetilde{\gamma}(t)(s(t) + q\bar{s}))\right]\widetilde{h}(t)^{\mu} \tag{6}$$

Intuitively, incomplete health knowledge leads smokers to smoke more than they would have done optimally, and causes them to form wrong belief in their health status (i.e., $\widetilde{h}$ differs from $h$). However, given that the true health status $h$ can be observed by the individual herself, she would think that the lower realized health is caused by some shock to $\delta$ rather than by smoking, and thus would not correct her biased knowledge about smoking and smoking behavior.

Finally, the individual dies when her health reaches a threshold level $\underline{h}$. That is, the terminal date of one's life $T$ is reached when $h \leq \underline{h}$. This setting is natural based on the medical literature, where $\underline{h}$ may be viewed as the biological requirement for a human body to function. Because the evolution of an individual's health capital depends on her decisions, her life expectancy is thereby endogenous depending particularly on smoking behavior and health investment. Thus, our framework is in spirit consistent with health capital model of Grossman (1972a,b) and the endogenous life expectancy model of Rosen (1988) in which the value of saving a current life is the expected present value of consumer surplus at that age.

The dynamic optimization problem can therefore be divided into two steps. In the first step, we view the terminal date $T$ as given. We then solve the lifecycle model in which an individual of type $(\alpha, \theta)$ decides on consumption/saving, smoking, and health investment, all as functions of $T$, to maximize her lifetime utility given by (2) subject to asset accumulation and health capital evolution equations (4) and (5). In the second step, we substitute the smoking and health investment functions

[11] There is lack of medical evidence on differential health effects of smoking by gender, race, or health condition (Jha, et al., 2013; Reiner, et al., 2019); thus, we assume $\gamma(t)$ to be the same for all individuals at a given age.

obtained in the first step, denoted $S(t,T)$ and $X(t,T)$, into (5) to generate the path of health capital, denoted $H(t,T)$. Then we pin down life expectancy by solving

$$H(T,T) = \underline{h} \tag{7}$$

Following these procedures, we can solve numerically the entire dynamic path of consumption, smoking, health investment, health capital and asset, and the endogenous life expectancy.[12] To do so, however, we must first calibrate the model to fit the data to which we now turn.

# 4 Calibration and Numerical Solution

We are now prepared to calibrate the model to fit the data. We begin by calibrating the full model, taking all behavioral considerations, under which we evaluate various policies on smoking cessation. To better understand how behavioral bias and externality influence policy effects, we also evaluate the same policies in the absence of incomplete health knowledge, bounded rationality, or second-hand smoking. To check the robustness of our benchmark results, we further extend the benchmark model to various settings in Section 6, including no relapses, quitting at age 50, no quitting delays, and net deadweight cost of medical treatment. Our calibration exercises consist of three steps. In the first step, we calibrate all parameters for a representative individual, who may be regarded as the weighted average of all smokers and nonsmokers. In the second step, we allow heterogeneity in preferences for smoking; in particular, we calibrate the three $\alpha$'s for smokers with three genotypes associated with smoking addiction. In the third step, we allow heterogeneity in work efficiency to calibrate $\theta$'s for individuals in different demographic groups as well as the difference in the natural health deterioration rate $\delta$ by gender. We discretize the life span so that one year corresponds to one period of time, and assume life starts at 18 years old (i.e., $t_0 = 18$).[13]

## 4.1 Step 1: Calibration to fit the average

In the first step, we assume that individuals are homogeneous in all aspects except that they are either smokers or nonsmokers, so we have a single value for $(\alpha, \theta, \delta)$, which allows us to calibrate all the common parameters to fit the average of each of the targeted data. There are twelve

[12] See details of the model characterization in Appendix.

[13] Most smokers start smoking before age 20 (Jha et al., 2013). One might easily allow $t_0$ to be lower and adjust the experience accumulation formulation in (3) if teenager smoking behavior were a major research focus.

parameters, $\rho$, $\bar{\beta}$, $\alpha$, $\epsilon$, $\delta$, $\mu$, $q$, $\theta$, $\phi_1$, $\phi_2$, $\kappa$, and $\underline{h}$, and three functional forms, $\Phi(t)$, $\gamma(t)$ and $\chi(t)$, to be determined. All the pre-set and calibrated parameter values are summarized in Table 3.

Given homogeneous work efficiency, we conveniently normalize $\theta w$ to be one. We set the subjective time discounting rate as $\rho = 0.02$ to fit the average US wealth-income ratio of about 7 (Glover et al., 2019). Then the growth rate of consumption ($g$) is set to equal that of output per capita in the US (1.8%), so the real interest rate becomes $r = \rho + g = 0.038$. We set initial wealth to be 15% of initial labor income as permanent flow income converted to capitalized value, that is, $a_0 = 15\% \cdot y_0/r$. Then as a terminal condition, $a_T$ is computed by multiplying $a_0$ and the compound interest rate over the life course to assure intergenerational growth of bequests.

We set initial stock of health at age 18 as $h_0 = 100$, and calibrate the threshold health level at death to be $\underline{h} = 38.77$ to match the average mortality rate and the average life expectancy of the US population.[14] These provide natural boundaries of the health stock within which the dynamic path of health can be computed to fit real world data. We set curvature of health investment to be $\epsilon = 0.3$ which falls within the range of estimates by Hall and Jones (2007) (0.042-0.4). We further choose the functional form of $\Phi(t)$ to be $\Phi(t) = \Phi_0(B + e^{-\eta(t-t_0)})$ to fit the pattern of increased health expenditure at older ages, and $\Phi_0$, $B$ and $\eta$ are to be calibrated.

In the labor economics literature, the wage-tenure profile estimation usually yields an experience return at $3-5\%$. We thus take the average to set $\phi_1 = 0.04$. Then to align health-based and experience-based human capital, we jointly calibrate $\kappa$ and $\phi_2$ to match income peak year around 52 and relative peak to initial income about 2.27, as documented in Guvenen et al. (2015).

To be consistent with medical evidence that smoking cessation at different ages has different impact on life expectancy (Jha et al., 2013), we specify $\gamma(t)$ as a step function:

$$\gamma(t) = \begin{cases} \gamma_0 & \text{for} \quad t \leq 30 \\ \gamma_1 & \text{for} \quad 31 \leq t \leq 40 \\ \gamma_2 & \text{for} \quad t \geq 41 \end{cases} \tag{8}$$

where the three $\gamma$'s are yet to be determined.

[14] More specifically, we first compute the average mortality rate $\overline{d}$ over the life course from the average life expectancy of the U.S. population (78 years old). Then we pin down the average health deterioration rate $\overline{\delta}$ over the life course using $\overline{d} = \frac{1}{T-t_0}\int_{t_0}^{T} d(t)dt = \frac{1}{T-t_0}\int_{t_0}^{T} \frac{1}{h(t_0)e^{-\overline{\delta}(t-t_0)}}dt$, where we assume that health evolution is an exponential process and that the mortality rate at each point of time is inverse to health status following Hall and Jones (2007). Finally, we obtain health capital at death using $h(T) = h(t_0)e^{-\overline{\delta}(T-t_0)}$.

Table 3. Parametrization of the model

| parameter | value | target |
|---|---|---|
| subjective time discounting | $\rho = 0.020$ | wealth-income ratio in literature |
| health stock boundaries | $h_0 = 100, \underline{h} = 38.77$ | normalized, computed |
| experience return | $\phi_1 = 0.04$ | literature |
| experience scaling factor, curvature | $\kappa = 355$<br>$\phi_2 = -5.9 \times 10^{-4}$ | joint targets of peak income year and relative peak income to initial |
| initial wealth | $a_0 = 395$ | preset |
| health investment curvature | $\epsilon = 0.3$ | literature |
| health cost of smoking | $\gamma_0 = 0.0010$<br>$\gamma_1 = 0.0040$<br>$\gamma_2 = 0.0408$ | joint targets of $\frac{c}{Y}, \frac{ps}{y}, \frac{x}{Y}, \frac{x_{38}}{x_{18}}, \frac{x_{60}}{x_{18}}, T$, life year reduction due to smoking and incremental life saving for quitting smoke at age 30, 40, 50 |
| natural health deterioration rate | $\delta = 1.270$ | same as above |
| preference for health quality | $\bar{\beta} = 0.598$ | same as above |
| health curvature | $\mu = 0.009$ | same as above |
| health investment efficacy | $\Phi_0 = 0.030$<br>$B = 4.156$<br>$\eta = 0.160$ | same as above |
| tastes for smoking for H1 | $\alpha_1 = 0.397$ | CPD of 3 genotypes |
| tastes for smoking for H2 | $\alpha_2 = 0.407$ | |
| tastes for smoking for H3 | $\alpha_3 = 0.426$ | |

Turning to boundedly rational decision making, we consider in the spirit of the recent contribution by Gabaix (2017) that individuals younger than 50 undervalue health by 10% whereas those at or above 50 overvalue health by 10%; that is, $\chi(t) = 0.1$ for $t < 50$ and $-0.1$ for $t \geq 50$. To capture individuals' incomplete knowledge about health, we use the empirical finding by Kenkel (1991) that for 7 illnesses that can be caused by smoking, respondents surveyed (including both smokers and nonsmokers) are aware of 5.5 on average. This implies individuals' belief of $\gamma(t)$, i.e., $\widetilde{\gamma}(t)$, to be 21% lower than its true value. Moreover, recall that $q\overline{s}$ measures the detrimental health effect resulting from second-hand smoking. We set $q$ to be 0.125 based on the finding in Pickett et al. (2006) that even in U.S. counties with extensive smoke-free law coverage the exposure to

second-hand smoking for nonsmokers remains to be 12.5%.[15]

Thus, the main parameters that remain to be calibrated are related to preference, $\alpha$, $\bar{\beta}$, and to the effectiveness of health investment, $\Phi_0$, $B$, $\eta$ and $\mu$, together with the natural health deterioration rate $\delta$ and three $\gamma$'s related to the age-dependent consequences of smoking for health. These ten parameters are calibrated jointly based on five targeted ratios, the average life expectancy, the life year gap between smokers and nonsmokers, plus three age-dependent incremental life saving figures as a result of smoking cessation.

Denote $y$ as labor income and $Y$ as (total) income (labor plus interest earnings). There are three targeted ratios: (i) the consumption-to-income ratio ($\frac{c}{Y}$), (ii) the health–investment-to-income ratio ($\frac{x}{Y}$), and (iii) the tobacco-expenditure-to-labor income ratio ($\frac{ps}{y}$), all based on the U.S. data averaged across individuals and over time. Using data from Penn World Table 6.3 (1998-2007, US average), we compute $\frac{c}{Y} = 0.614$.[16] Employing data from World Bank 1999-2011, we compute the average healthcare cost-to-GDP ratio to measure the health investment-to-income ratio $\frac{x}{Y} = 0.149$. That is, an average individual (weighted average of addicts and nonaddicts) invests about 15% of her income in health. Also using data from PSID 1999-2011, we compute the tobacco-expenditure-to-labor income ratio $\frac{ps}{y}$ as an average household head's smoking expenditure over labor income.[17] More specifically, smoking expenditure $ps$ is computed by multiplying (tax-included) price of per pack of cigarettes (normalize cigarette price in 2009 to be one and compute price in other years correspondingly with CPI adjusted, which results in the average price during 1998-2010 0.79 per pack), cigarettes per day from PSID, and 365 days. We get $\frac{ps}{y} = 0.015$. That is, an average individual (weighted average of addicts and nonaddicts) spends about 1.5% of her labor income on smoking. Two other targeted ratios are related to health expenditure over the lifecycle. Based on Hall and Jones (2007), the average relative health expenditure at age 38 to age 18 ($\frac{x_{38}}{x_{18}}$) is 1.73, and that at age 60 to age 18 ($\frac{x_{60}}{x_{18}}$) is 4.62. Intuitively, these lifecycle health expenditure ratios can be used to pin down the health investment efficiency function $\Phi(t)$.

The average life expectancy of the US population is 78 years old, based on the World Bank data (1998-2011). From Jha et al. (2013), the gap of life expectancy between smokers and nonsmokers

[15] Because the exposure to second-hand smoking is nonnegligible, government policy may potentially benefit everyone by bringing down $\bar{s}$. Moreover, the personalized medication policy will be more effective in reducing second-hand smoking than the standard polices since it provides coverage for more smokers.

[16] This consumption-to-income ratio has excluded tobacco consumption and private health expenditure.

[17] In computing the average, observations with $\frac{ps}{y}$ ratio greater than one or negative are excluded from the sample. We also include whites and blacks only in the sample for it to be consistent with our policy experiments in Section 5.

is 12 years, and the averaged increases in life expectancy for smokers who quit at the age 30, 40 or 50 are 10, 9 and 6 years, respectively. These smoking-related life years are employed in particular to pin down the functional form of smoking effect on health $\gamma(t)$. We then use the simplex method to search for the set of ten parameters that minimizes the distance between the model and the targeted values. The algorithm for calibrating these parameters are given as follows. We first choose a set of values for the parameters (initial guesses), and then solve the model which results in a set of lifecycle decisions and outcomes for a representative smoker and a representative nonsmoker, respectively. Next, given the share of smokers and nonsmokers in data (24.6% and 75.4%, respectively), we compute the weighted average of the targets from the model and compare them to the corresponding targeted values. If they are not close enough, the guesses are revised. This is repeated until the model solutions and the targeted values are sufficiently close.[18] From our result, while all the targeted ratios and life expectancy are matched well, the increases of life expectancy for a representative smoker who quits at the age 30, 40 or 50 turn out to be 11, 9 and 6, which are very close to the targeted results of 10, 9 and 6 (see Table 4).

Table 4. Fitness: model versus target

| | model | target |
|---|---|---|
| $\frac{c}{Y}$ | 0.663 | 0.614 |
| $\frac{ps}{y}$ | 0.0188 | 0.0153 |
| $\frac{x}{Y}$ | 0.166 | 0.149 |
| $\frac{x_{38}}{x_{18}}$ | 1.708 | 1.730 |
| $\frac{x_{60}}{x_{18}}$ | 4.242 | 4.620 |
| $T$ | 78 | 78 |
| $T_{gap}$ | 12.3 | 12 |
| LY gains at age 30, 40, 50 | 11, 9, 6 | 10, 9, 6 |
| $s$: H1, H2, H3 (in model unit) | 28.2, 28.8, 30.6 | 28.2, 28.8, 30.6 |

Furthermore, we obtain the lifecycle pattern of income, health, health investment and smoking expenditure ratios for an average nonsmoker and an average smoker, respectively, as shown in Figure 2. There are several interesting lifecycle patterns of interest. To begin, we note that labor income

[18] Under each set of parameterization, the second-hand smoking $\overline{s}$ is solved in equilibrium to equal the average amount of smoking.

peaks at around age 52, whereas individuals start to dissave only at later ages. While nonsmokers and smokers do not differ much in labor income, nonsmokers earn substantially higher asset income than smokers and thus accumulate more assets toward later stages of life. As reported in Table 5, by including asset income, smokers at age 35 earn 11.1% less total income than nonsmokers, with the gap widened to 14.7% at ages 50 and 23.2% at retirement. Also interestingly, nonsmokers' total income peak at age 61 but smokers' at age 58, which is consistent with the smokers' shorter life expectancy (68-69 years as compared to 81 years). Meanwhile, the health-investment-to-income ratio rises sharply at older ages, especially for nonsmokers who live for more than a decade longer than smokers. Before retirement, smokers incur a higher the health-investment-to-income ratio, about 0.8% higher than smokers at age 35 and 1.4% and 4.1% higher at ages 50 and 65. These are in line with the literature documenting that many elderly accumulate assets toward very advanced ages as a buffer against the expensive medical care arising from longevity (e.g., De Nardi et al., 2010), and that quitting smoking can bring down healthcare costs in the short run but would raise the costs in the long run as smoking-related mortality declines and people start to age (Barendregt et al., 1997).

Table 5. Lifecycle patterns compared: Smokers versus Nonsmokers at various ages

| At age | 35 | 50 | 65 |
|---|---|---|---|
| Gap in $Y$ | | | |
| nonsmokers | 336.8 | 538.6 | 592.4 |
| smokers | 299.5 | 459.6 | 454.9 |
| gap | −11.1% | 14.7% | −23.2% |
| Gap in $x/Y$ | | | |
| nonsmokers | 8.6% | 9.9% | 16.8% |
| smokers | 9.4% | 11.3% | 20.9% |
| gap | 0.8% | 1.4% | 4.1% |
| Gap in $h$ | | | |
| nonsmokers | 84.4 | 69.0 | 54.0 |
| smokers | 82.7 | 63.2 | 43.9 |
| gap | −2.0% | −8.4% | −18.7% |

Moreover, the smoking expenditure ratio of smokers declines with age, where the zigzag pattern of the ratio is due to the simple step function form to capture the rising effect of smoking on

health (refer to the $\gamma(t)$ function given by (8) and the calibrated values reported in Table 4). To facilitate a comparison of the smoking expenditure ratio between the model and the PSID data, we smooth the ratio computed from our model using exponential approximation (the solid curve in the bottom-left panel of Figure 2).[19] While our model tends to overestimate the smoking expenditure ratio at younger ages and underestimate it at older ages, the two curves representing the model (smoothed) and the data, respectively, show a similar trend over the life course and intersect at age 40. A remark is in order. Recall that the $\gamma(t)$ function is set to match the life year gains from quitting at the respective ages. Also note that, although our smoking preference parameter $\alpha > 0$ is path-dependence as a result of addiction influenced by the environment of peer, home and workplace, it is not allowed to change over time. Should habit formation be incorporated, it is likely that $\alpha$ is rising over time, which would have corrected the problem with overestimation at younger and underestimation at older ages. Unfortunately, we do not have additional data moments to discipline a time-varying smoking preference parameter while at the same time to match the life year gains.

Finally, despite health investment, health naturally deteriorates since age 18 until death. From the bottom-right panel of Figure 2, it is clear that smokers suffer more severe health deterioration. As reported in Table 5, the health capital gap between smokers and nonsmokers are widening through one's life course, from 2.0% at age 35 to 8.4% at ages 50 – by retirement age, such a gap is 18.7%, which explains why smokers suffer a significant loss in life years (about 12-13 shorter).

### 4.2 Step 2: Heterogeneous preferences for smoking

In the second step, we allow for heterogeneity in preferences for smoking in order to capture different genotypes associated with smoking addiction.

In particular, we calibrate the three $\alpha$'s for smokers with three genotypes, H1, H2, and H3, while employing all other parameter values from the first step. We use cigarettes per day (CPD) for three types of smokers and shares of each type from Chen et al. (2012), rescaling the amount of smoking to fit the model units which becomes our targeted value of smoking ($s$) for each genotype.[20] Again,

[19] In particular, we regress log of smoking expenditure ratio on age, i.e., $\log(ps/y_t) = b_1 + b_2 t + \varepsilon$, and then compute the smoothed $ps/y$ ratio using $\widehat{ps/y}_t = \exp(\widehat{b}_1 + \widehat{b}_2 t)$, using the estimated coefficients.

[20] The amount of tobacco intake may be underestimated for H3, as medical evidence suggests that H3 smokers inhale more deeply when smoking, and thus may have higher smoking intensity given the same cigarettes smoked. However, data on smoking intensity of different genotypes is unavailable except for CPD, which limits our capability of providing a more accurate estimate of the smoking level. Even so, this underestimation implies that our estimate of

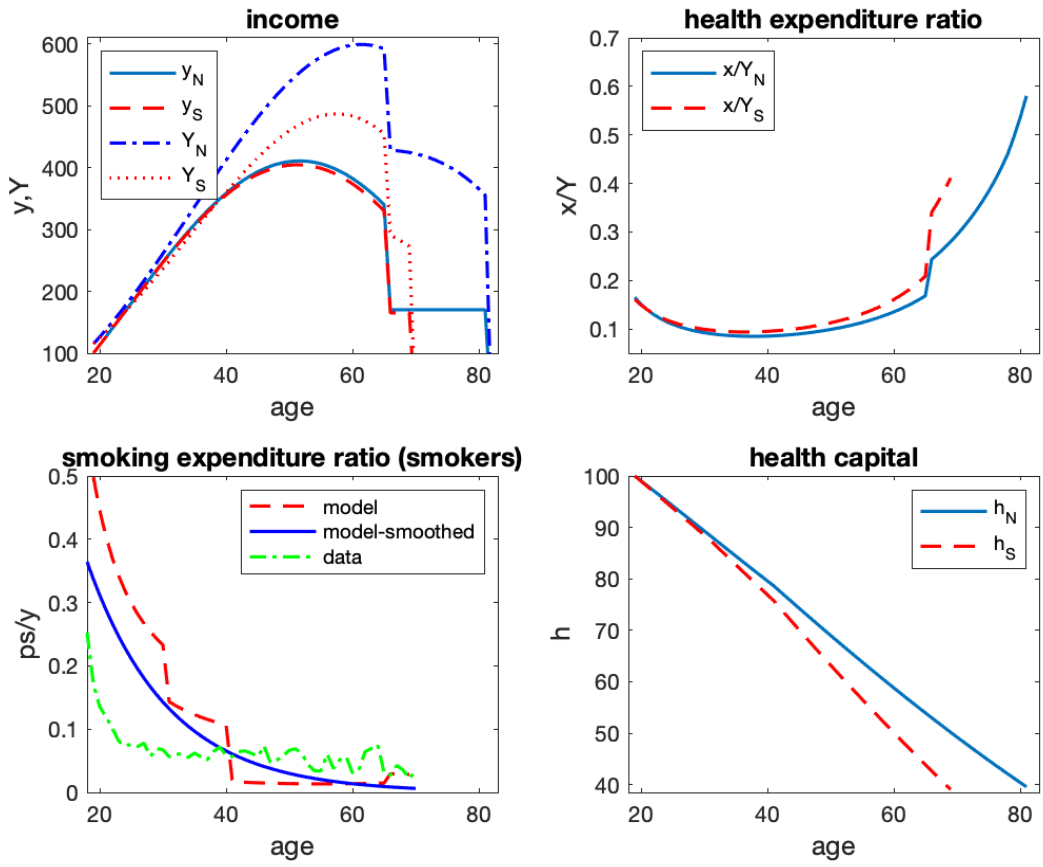


Figure 2: Lifecycle profile of income, health, smoking and health investment

we conduct this task using the guessing-revising-converging algorithm. We obtain $\alpha_1 = 0.397$, $\alpha_2 = 0.407$, and $\alpha_3 = 0.426$, corresponding to H1, H2, and H3 types of smokers respectively.

We are now prepared for utilizing the calibrated model to compute lifecycle profile for the three types of smokers and compare it with nonsmokers. Table 6 presents such a comparison in key indicators including life expectancy $T$, the consumption ratio $\frac{c}{Y}$, the smoking expenditure ratio $\frac{ps}{Y}$, and the medical expenditure ratio $\frac{x}{Y}$ for each of the four groups of population, as well as the respective population weighted averages.

As can be seen, life expectancy of smokers is $12-13$ years shorter than nonsmokers, consistent with medical evidence (Jha et al., 2013). H3-type smokers live slightly shorter than other types, matching with medical evidence that *CHRNA5* is associated with increased mortality in large-scale population studies (Halldén, et al., 2016). Smokers spend about 6-7 percent of income on tobacco;

policy effectiveness (in Section 5) is a conservative measure, since the relative effectiveness of personalized medication policy versus non-personalized medication policy is supposed to be the largest for H3 smokers.

while they spend a similar share of income on normal consumption as nonsmokers, the level of such consumption is lower than nonsmokers' due to a much lower income.

The health expenditure ratio in row 5 may look too low for smokers compared with nonsmokers. This arises from the large life year gap between smokers and nonsmokers, and the sharply increasing health care expenditure at very advanced ages when diminishing returns start to kick in. This increases the average healthcare expenditure ratio of nonsmokers over the life span. If we trim everyone's life span to be the same as the type with the shortest life (i.e., H3-type smokers), then the health investment ratio becomes about 12 percent higher for smokers (see the last row of Table 6), consistent with evidence in literature that health care cost for smokers is about 10 percent higher than nonsmokers at the middle age (Barendregt et al., 1997).

With this node, we now turn to step 3 to calibrate differentiated $\theta$'s and $\delta$'s for individuals in different demographic groups.

Table 6. Lifecycle comparison

| | H1 | H2 | H3 | N | mean |
|---|---|---|---|---|---|
| share of population (%) | 6.2 | 10.8 | 7.6 | 75.4 | |
| life expectancy $T$ | 69 | 69 | 68 | 81 | 78 |
| consumption ratio $\frac{c}{Y}$ | 0.667 | 0.666 | 0.665 | 0.663 | 0.664 |
| smoking expenditure ratio $\frac{ps}{Y}$ | 0.064 | 0.066 | 0.071 | 0.00 | 0.016 |
| health investment expenditure ratio $\frac{x}{Y}$ | 0.141 | 0.141 | 0.138 | 0.175 | 0.166 |
| health investment expenditure ratio $\frac{x}{Y}$, LY fixed | 0.137 | 0.137 | 0.138 | 0.123 | 0.126 |

### 4.3 Step 3: Heterogeneous work efficiency and health deterioration rates

Now we allow individuals' work efficiency to differ by gender, race and education. Normalizing the market wage in efficiency unit as one ($w = 1$), we now calibrate different values of $\theta$ for each group. By construction, the population weighted average of $\theta$ must be one. Thus, $\theta$ is indeed a relative work efficiency measure (relative to the mean).

Specifically, let the gender indicator be $M$ (=1 for male and 0 for female), the race indicator be $W$ (=1 for white and 0 for black), and the education indicator be $C$ (=1 for college educated and 0 for non-college educated). We then categorize all individuals into 8 (i.e., $2 \times 2 \times 2$) groups. We calibrate $\theta$'s of the eight cells of population by gender, race and education using PSID labor income of individuals (household head) aged between 23 and 26, normalized to an average of one. We pick

this age range for three reasons. First, the vast majority of people have completed education and started working by 23. Second, their health may have not been significantly affected by smoking behavior by 26. Third, the experience may have not contributed much to their labor income.

In Table 7, we report the population share of each group among smokers based on PSID data and the calibrated value of $\theta$ for each group. As can be seen, college educated all have higher than the mean work efficiency and are thus expected to have higher labor income upon controlling the age. Black with no college degree, on the contrary, have much lower work efficiency and hence lower labor income within the same age cohort. In addition, there is a gender gap that leads to higher labor income for males compared to females. Such gaps are larger for whites than for blacks. All these implied patterns for labor income are consistent with the labor economics literature. As well documented in the literature, better educated earn higher labor income, have better health knowledge, invest more in health for longevity and are less likely to be smokers (Chaloupkia, 1991; Kenkel, 1991; Grossman and Kaestner, 1997; Lleras-Muney, 2005). As a consequence, this dimension of heterogeneity can lead to an outcome with nonsmokers having larger health investment relative to income.

In addition, it is a well-established fact that women on average live longer than men and the gender life year gap is about 5 years. To capture this biological difference, we allow heterogeneity in the natural health deterioration rate ($\delta$) across genders and calibrate it for men and women separately to match their average life year expectancy (75.5 and 80.5 years, respectively). This results in $\delta_M = 1.34$ for men and $\delta_F = 1.21$ for women, respectively.

Table 7. Work efficiency

| $M$ | $W$ | $C$ | share within smokers (%) | work efficiency $\theta$ |
|---|---|---|---|---|
| 0 | 0 | 0 | 5 | 0.63 |
| 0 | 1 | 0 | 45 | 0.79 |
| 0 | 0 | 1 | 0 | 1.13 |
| 0 | 1 | 1 | 5 | 1.13 |
| 1 | 0 | 0 | 7 | 0.77 |
| 1 | 1 | 0 | 34 | 1.10 |
| 1 | 0 | 1 | 1 | 1.36 |
| 1 | 1 | 1 | 3 | 1.46 |

Finally, it is noted from medical evidence (Chen et al., 2012) that the share of three genotypes

vary substantially across racial groups: whites have a much larger share of H3 type than blacks, while blacks have a much larger share of H1 type than whites (refer to medical evidence in Section 2). This heterogeneity in shares of genotypes will cause differences in policy effects for different racial groups due to a composition effect as will be analyzed later.

### 4.4 Price elasticity of smoking

To further validate our calibrated model, we now compute the untargeted moment of the price elasticity of smoking from our model and compare it to the literature. In particular, we set $\theta = 0.93$ (i.e., the average value of $\theta$ for smokers) and compute the price elasticity at each given age as well as the lifecycle average from our calibrated model. The average price elasticity of smoking from our model is $-0.36$, which falls into the range estimated in the literature that is between $-0.17$ and $-0.56$, and it is very close to most estimates in the literature that center around $-0.4$ (see Chaloupka and Warner 2000 for a comprehensive empirical survey of such estimates). Moreover, the literature documents an inverse relationship between the (absolute value of) price elasticity of smoking and age, which is confirmed by our results (see Figure 3). In particular, the literature shows that youth or young adults have the highest (absolute value of) price elasticity which lies between $-0.58$ and $-1.31$. For example, the estimate from Tauras and Chaloupka (1999) for a sample of individuals in age from 17 to 35 is about $-0.79$, while the corresponding figure from our model is $-0.78$.[21] Thus, our model fits very well with the untargeted moments relating to price elasticity of smoking.

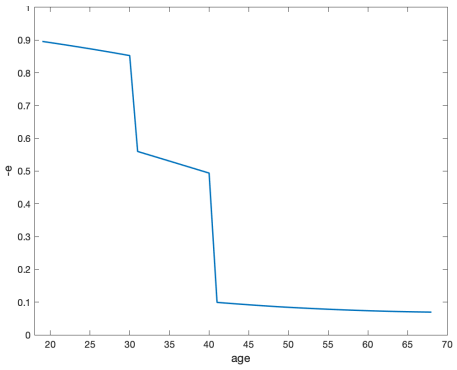


Figure 3: Price elasticity of smoking (absolute value)

[21] Our model also shows that the (absolute value of) price elasticity decreases with $\theta$, generally consistent with the literature that lower income groups (i.e., youth, blacks, or less-educated) are more price elastic in smoking.

# 5 Policy Evaluations

With the theoretical model calibrated to fit the data, we have a running numerical model that can be readily used for policy evaluation. In the interest of this study, we focus on evaluating smoking-cessation policy contrasting personalized with standard medications for smoking-cessation treatment. By doing so, we establish an integrated dynamic framework for a systematic cost-effectiveness analysis on precision medicine in this regard. In the following analysis, we consider policy intervention in forms of subsidizing or rebating smokers to receive cessation treatment which helps reducing addiction to smoking. We begin with policy analysis under the benchmark setup, taking all behavioral considerations. We then evaluate the role of behavioral bias (i.e. incomplete health knowledge about smoking and bounded rationality) and negative externality associated with second-hand smoking played in policy effectiveness with a number of counterfactual experiments.

## 5.1 Method

We compare the effectiveness of the following three government policies including two types of standard medication policies ($\boldsymbol{S}$, $\boldsymbol{S}'$) and one personalized medication policy ($\boldsymbol{P}$), all under the same budgetary cost. Thus, whichever policy yielding greater value of effectiveness is by construction more cost-effective. The three smoking cessation policies are specified as follows.

1. $\boldsymbol{S}$*: subsidize 10% of smokers randomly with a \$600 voucher for both counseling and medication treatment, regardless of genotypes.*

2. $\boldsymbol{S}'$*: subsidize 10% of smokers randomly with two types of vouchers: a \$150 voucher for counseling treatment only, given to 5% of smokers, and a \$1050 voucher for both counseling and medication treatment, given to another 5% of smokers, regardless of genotypes.*

3. $\boldsymbol{P}$*: offer rebate to n% of smokers who have taken genetic test to identify their genotypes associated with smoking addiction. The rebate consists of three parts. R1: \$96 for genetic test; R2: \$72 for counseling; R3: \$432 for medication. All subsidized smokers receive R1 and R2, but only those identified as H2 and H3 genotypes receive R3. The percentage of smokers that are subsidized, n, is determined such that the total budget is identical to that under* $\boldsymbol{S}$ *and* $\boldsymbol{S}'$*.*

Several important points regarding the design of the policies above are in order.

First, the size of the subsidy in the benchmark case under policy $\boldsymbol{S}$ ($600 per person) is reasonable for covering about half of counseling and medication treatment cost (see the detailed breakdown below). The amount (inflation-adjusted) is comparable to the financial incentives ($500) provided for smoking cessation in the randomized controlled trial conducted by Volpp et al. (2009) – their study finds that smokers receiving financial incentives are significantly more likely to participate in and complete a smoking-cessation program.

Second, the size of the budget regarding 10% coverage of smokers in the benchmark case under policy $\boldsymbol{S}$ is also reasonable.[22] The current policy is for Medicaid to pay for all smokers. Since Medicaid covers about 20% of the population, it is expected that more than 20% of smokers may use this benefit. Moreover, the amount of the subsidy is only about half of the actual treatment cost incurred. Even by assuming that only a quarter of Medicaid eligible smokers take advantage of this benefit, the size of the budget under our experiment would not be higher than the budgetary cost of the current policy through Medicaid coverage for smoking cessation.

Third, the three policies are designed on a revenue-neutral base, meaning that the total budget is equalized across all three policies. The shares of three genotypes among smokers are 0.25, 0.44, and 0.31 for H1, H2, and H3 respectively (Chen et al., 2012; Bergen et al., 2013). This enables us to compute $n$ in the next subsection.

Fourth, the value of each type of voucher or rebate in policy $\boldsymbol{S'}$ and $\boldsymbol{P}$ is based on the monetary cost of the corresponding treatment or test. We estimate the cost to be $150 for counseling, $900 for medication, and $200 for a genetic test. The counseling cost ($150) and the total cost of counseling and medication ($1,050) are the base for the vouchers in policy $\boldsymbol{S'}$. The total cost of the three is $1,250. Hence, in policy $\boldsymbol{P}$, the value of each type of rebate is proportional to the cost of the corresponding treatment or test.

Fifth, the motivation behind the policy design is as follows. Policy $\boldsymbol{S}$ is a proxy of the standard policy that recommends all smokers to receive both counseling and medication. Policy $\boldsymbol{S'}$ is a proxy of the real world policy by which some smokers receives counseling while others also receive medication. Neither of the two standard policies takes smokers' genotypes into account. By contrast, policy $\boldsymbol{P}$ is personalized based on genotypes. Smokers receive subsidy only if they have taken a genetic test, and only those with genotypes responsive to medication would receive subsidy for medication treatment.

[22] We could use other numbers without changing the main results of policy comparison.

In our benchmark experiment, we carry out the policies on smokers aged 35 since medical research shows that smokers within age 30-40 gain larger health benefit from cessation than other age groups. We solve the model with each policy for each type of smokers with different genotypes and different demographics in the dimensions of gender (male versus female), race (white versus black), and education (college degree versus no college degree).[23] In addition, when modeling the cost of treatment or genetic test, we also take into account the time cost (i.e., time needed for traveling, waiting, and receiving treatment) besides the monetary cost, and convert it into labor income using hourly wage of each group of population (again, using PSID data). Based on medical practice, we estimate that it takes about 9 hours to receive counseling, 7.5 hours for medication, and 2 hours for genetic test.

Finally, we employ four measures for policy comparison:

1. *Share of smokers that can be subsidized;*
2. *Quit rates of subsidized smokers;*
3. *Consumption Equivalent (CE) of subsidized smokers;*
4. *Income Equivalent (IE) of subsidized smokers.*

While Measure 2 is standard, Measure 1 is an "extensive margin" that has often been overlooked in previous studies. The omission of this extensive margin effect would bias the cost-effectiveness analysis significantly, particularly because of the nature of personalized medical treatment. Measures 3 and 4 require further explanations.[24]

Consumption Equivalent, or $CE$, is defined as the percentage increase in consumption (at each point in time) that is needed, while fixing everything else unchanged, to reach a certain level of lifetime utility if the individual is covered by a policy. Hence, we derive Consumption Equivalent

[23] We also adjust the society-wide level of smoking (i.e., second-hand smoking) under each policy using the share of subsidized smokers, the quit rate and quit age of these smokers, and the amount of smoking over the life course. While the reduction in the second-hand smoking under the proposed policy intervention is very small (due to the relatively low coverage rates and quit rates), it is indeed larger under the personalized policy than the standard ones.

[24] We do not include policy effects on nonsmokers and unsubsidized smokers caused by reductions in second-hand smoke for two reasons. First, such effects depend on the share of smokers subsidized and thus are not robust. Second, under the current coverage rate, the effects on these two groups are negligible compared with that on subsidized smokers. In this regard, our measured policy effectiveness can be viewed as conservative ones.

as (see details in Appendix):

$$CE = \exp\left[\frac{\rho\left(\overline{U} - U_0\right)}{1 - e^{-\rho(T-t_0)}}\right] - 1 \tag{9}$$

where $\overline{U}$ is the lifetime utility when the individual is covered by a particular policy, and $U_0$ is the untreated lifetime utility in the absence of policy intervention. Note that whether cessation is successful is a random outcome and the probabilities are taken from medical data directly. These probabilities vary across individuals, depending on genotypes, education, and the type of treatment (refer to medical evidence in Table 3 of Section 2). Moreover, we regard relapse of smoking cessation as unsuccessful cessation and thus adjust the quit rate by deducting the relapse rate.[25] Thus, by allowing for heterogeneous quitting probabilities across different genotypes and demographic groups based on medical evidence, such probabilities capture the distribution of idiosyncratic characteristics of individuals that lead to differential smoking behavior within the same group as well as systematic behavioral differences across groups. Accordingly, $\overline{U}$ in equation (9) can be regarded as "expected utility" by taking into account the probabilities that smoking cessation may either succeed or fail under a policy, without the need to model individual endogenous respond of quitting to the policy. In addition, cessation often happens with a delay after treatment. Typically, H3 smokers experience the longest delay, followed by H2, and then H1 (cf. Chen et al., 2015a). Based on this evidence, we assume that a successful cessation occurs 2, 3 and 5 years after treatment for H1, H2 and H3 types, respectively.

An alternative measure to $CE$ is Income Equivalent, or $IE$, which is defined as the percentage increase in labor income (at each point in time) that is needed to satisfy the consumption increase as defined in $CE$. That is,

$$IE = CE \cdot \frac{\int_{t_0}^{T} e^{-r(t-t_0)} c dt}{\int_{t_0}^{T} e^{-r(t-t_0)} y dt} \tag{10}$$

Intuitively, $CE$ and $IE$ represent the benefit of a policy to a smoker in terms of lifetime utility, measured as an equivalence of consumption or income increase. Since life expectancy varies across groups of smokers, $CE$ and $IE$ are life-expectancy-weighted to be comparable across individuals.

Notably, both $CE$ and $IE$ are measures of effectiveness. They are more general than the typical measure by QALYs in the literature of medicine and health. In contrast with QALYs, our

[25] We do not model smoking relapse because it is found most likely nonbehavioral, unrelated to typical demographics (age, gender, education), or workplace smoking ban, or perceived costs of smoking or benefits of quitting (see Koçak et al. 2015, Herd et al. 2009 and Longo et al. 2001, respectively). Also, because of the absence of correlation with demographics, we shall take the relapse rate 51.4% from Koçak et al. (2015) and apply it to all demographic groups.

lifetime utility based effectiveness measures account for time discounting and diminishing marginal valuation. Moreover, our measures consider differential utility weights that are calibrated to fit the data. Further, our measures incorporate heterogeneous individuals' dynamic responses. This is important because such responses varies across different individuals at different ages over their life course, as illustrated in the Section 5.3.

### 5.2 Validating the quantitative model: value of statistical life

To validate our key measure of $IE$ for constructing a dollar measure of effectiveness, we first check whether it may produce a reasonable figure of the value of a statistical life, VSL. By normalizing the value of death as zero (which can be simply obtained by setting $c = s = h = 1$ for smokers and $c = h = 1$ for nonsmokers, all at their subsistence levels), one may compute VSL as the dollar value of willingness to pay for a living status on average. This is done by first computing $IE$ for bringing lifetime utility from zero to the calibrated value in the model of an adult. We can then multiply $IE$ by average income of an individual to obtain a dollar measure. We finally convert permanent flow income at age 45 (average age of a licensed driver in the U.S. as we plan to compare our VSL with empirical estimates by Ashenfelter and Greenstone, 2004) to capitalized dollar value based on the real interest rate of 3.8% and the life expectancy of smokers and nonsmokers, respectively.

Assume that an average smoker of any genotype has the same work efficiency $\theta = 0.93$. Then a smoker's average income is 282 efficiency wage units or US\$36, 801 at 2009 constant dollars and the respective life expectancy becomes 69 years. As a result, VSL's for type H1, H2 and H3 can be computed as 1.804, 1.808 and 1.831 million US dollars, largely comparable regardless of genotypes. Given the population weights for the three genotypes (25%, 44% and 31% within smokers), the (weighted) average VSL of a smoker becomes 1.814 million US dollars. In the case of nonsmokers, work efficiency is 1.03 and average income is US\$55, 587 (at 2009 constant dollars), the life expectancy is 82. Accordingly, the VSL of a nonsmoker is computed as 2.146 million US dollars. Given the population weights (6.2% smokers of H1 type, 10.8% smokers of H2 type, 7.6% smokers of H3 type and 75.4% nonsmokers), the (weighted) average VSL in our model is equal to 2.065 million US dollars, almost 14% higher than the comparable figure of a smoker. In the empirical literature, Ashenfelter and Greenstone (2004) estimate an average U.S. driver's VSL at 2.06 million dollars (CPI adjusted to 2009 constant dollars), whereas Costa and Kahn (2004) obtain an average prime age (18-30) US male's VSL as 6.97 million dollars (also CPI adjusted to 2009 constant dollars). Our average VSL falls in between of their estimates and is close to the lower

bound of their range, which is viewed as empirically plausible.

## 5.3 Findings

We begin by computing potential gains in life years for heterogeneous groups of smokers if cessation becomes successful when policy is implemented at age 35, and by contrasting the lifecycle profiles in smoking, health investment, health capital and labor income between a quitter and a nonquitter. We then conduct policy effectiveness analysis and report results from policy comparison using the four measures described in Section 5.1. All the averaged figures reported are based on population weighted measures of the most disaggregated groups (demographics and biomarkers).

### 5.3.1 Gains in life years

Table 8 reports the potential gain in life years (LY) for smokers with the three genotypes and their average if cessation (when policy is implemented at 35 years old) is successful.

Table 8. Gains in life years for three genotypes

| | gain in LY |
|---|---|
| mean | 9.74 |
| H1 | 9.18 |
| H2 | 10.03 |
| H3 | 9.77 |

The results suggest that, across the three genotypes, the gain in LY is the largest for H2 smokers (10.03 years), and is the lowest for H1 smokers.

Table 9 shows life expectancy before and after quitting and LY gains for each of the 8 demographic groups (gender, race and education). As can be seen, all demographic groups gain life expectancy by quitting smoking, ranging from 7.1 to 13.0 years. Given gender, groups with higher $\theta$ (e.g., the college groups) gain more LY than those with lower $\theta$ (e.g., the non-college groups), since the former groups have a higher opportunity cost of smoking; that is, cessation increases lifetime income by more and hence health investment by more. Across the two genders, women gain more LY than men given race and education. This is because women have a lower natural health deterioration rate than men, which means the health effect of smoking is relatively larger for women; thus, cessation benefits women more.

Table 9. Gain in life years for eight demographic groups

| M | W | C | LY-nonquit | LY-quit | gain in LY |
|---|---|---|---|---|---|
| 0 | 0 | 0 | 69.0 | 78.5 | 9.5 |
| 0 | 1 | 0 | 70.2 | 80.6 | 10.4 |
| 0 | 0 | 1 | 72.9 | 85.9 | 13.0 |
| 0 | 1 | 1 | 72.6 | 85.6 | 13.0 |
| 1 | 0 | 0 | 64.9 | 72.0 | 7.1 |
| 1 | 1 | 0 | 66.2 | 75.0 | 8.8 |
| 1 | 0 | 1 | 67.9 | 77.9 | 10.0 |
| 1 | 1 | 1 | 68.0 | 78.2 | 10.2 |

### 5.3.2 Lifecycle profiles: quitters versus nonquitters

We now contrast the lifecycle profiles between a quitter and a nonquitter. In particular, we consider the quitter as to successfully stop smoking at age 35 with a delay. For illustrative purposes, we focus on two demographic groups:

1. white females with no college degree: this is the largest group of smokers (45%), whose work efficiency is far below one (0.79);

2. white males with college degree: this constitutes a small group (3%) of smokers, but has the highest work efficiency (1.46), so informative for comparison purposes.

For both groups, we choose the H2 genotype, which constitutes the largest fraction of smokers in both demographic groups.

In Figure 4, we plot the lifecycle profiles of the smoking expenditure ratio (top panels) and the health investment expenditure ratio (bottom panels); the lifecycle profiles of income (top panels) and health capital (bottom panels) are plotted in Figure 5.

Let us look at the lifecycle profiles of the smoking expenditure ratio. While the white males with college degree have a similar lifecycle pattern (right panels) as the white female with no college degree (left panels), their smoking expenditure ratios are lower due to a higher opportunity cost of health deterioration and their quitting attempts are relatively more resistant (this last point is not reflected in the figure, but can be shown with quit rates, which is 30% for white males with college degree versus 21% for white female with no college degree). For both white females with no

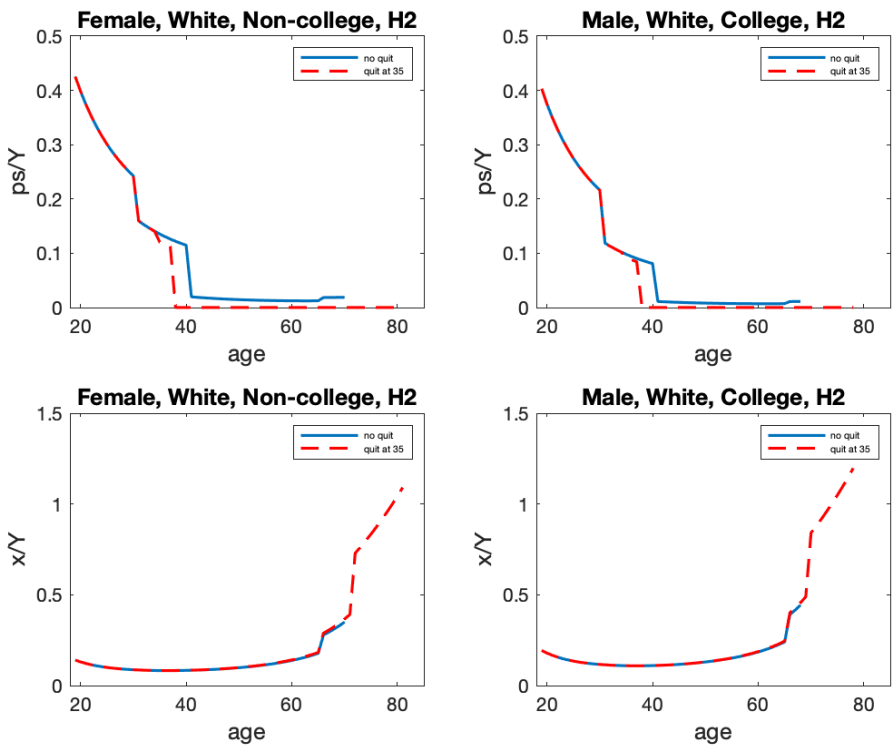


Figure 4: Lifecycle profile of smoking and health investment

college degree and white males with college degree, labor income of nonquitters and quitters (stop smoking at age 35) are peaked at 51 and 52 years, respectively. At the corresponding peak years, the percentage gain in labor income of the former group is 0.93% whereas that of the latter group is slightly higher at 0.98%.

We turn next to the lifecycle profiles of the health investment expenditure ratio. Throughout the life course, the health investment expenditure ratios are larger for white males with college degree, compared to white females with no college degree. This is essentially due to the positive income effect: recall that the positive health effect on life expectancy under our utility function setting makes health a luxury good relative to consumption. While for all groups the health investment expenditure ratios increase after the middle age, that of quitters increases at a higher rate, especially toward the end of life, suggesting a higher demand for investment in health by quitters. Specifically, the intensive margin effect of quitting is to raise the health investment expenditure at age 50 by 1.42% and 0.98% (or, to raise health investment expenditure ratio at 50 by 0.07 and 0.03 percentage points), respectively, for white females with no college degree and white males with college degree. Such an intensive margin effect rises to 5.43% and 4.46% at age 68 for the respective groups (or, 1.30

percentage points for both groups at 68).[26] This result suggests that smoking cessation benefits health not only because it eliminates health deterioration effect of tobacco intake, but increases the level and share of income for health investment.[27]

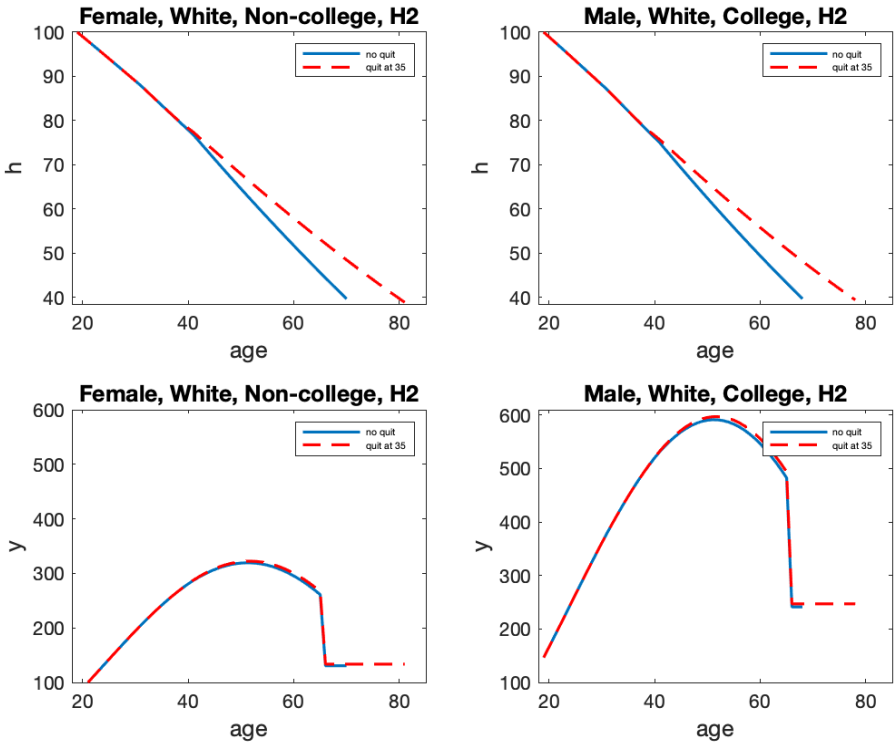


Figure 5: Lifecycle profile of health capital and labor income

We at last examine the lifecycle profiles of health capital. Despite health investment, health naturally deteriorates since age 18. Not surprisingly, successful quitting following treatment at

[26] Age 68 is chosen for calculating the intensive margain effect because the average life expectancy of white male smokers (i.e., nonquitters) with college degree is 68 years. The intensive margin effect of quitting on health investment may look different from estimates in medical literature which show that medical expenditures fall after smoking cessation in the short run (but rise in the long run due to longevity) (Barendregt et al., 1997). This may be explained by the perfect foresightness assumption of our model that implies smoother expendture of health investment over the life course.

[27] The health expenditure ratio may look close for quitters and nonquitters at younger ages because of two offsetting effects of quitting on health expenditure. On the one hand, quitting improves health and thus lowers the healthcare cost; on the other hand, quitters expect to live many years longer, which increases their demand for health investment.

age 35 slows down the rate of health deterioration. Although white males with college degree have stronger incentives to invest in health as elaborated above, their natural health deterioration rate is higher than white females with no college degree; thus, they have a slightly shorter life expectancy than the latter group. The percentage gains of quitting at age 35 in health capital at age 50 are 5.19% and 5.65%, respectively, for white females with no college degree and white males with college degree. The comparable gains in health capital are even larger at age 65, about 16.48% and 17.87%, for the respective groups. Thus, one should not overlook the cumulated detrimental health effect of smoking especially for the older population.

### 5.3.3 Policy Effectiveness

We now report our main findings obtained by comparing the four key measures under the three proposed policies.

**Share of smokers that can be subsidized**

Revenue-neutral policy comparison implies that 12.20% of smokers can be subsidized under policy $\boldsymbol{P}$. This means policy $\boldsymbol{P}$ may cover 22 percent more smokers for treatment than $\boldsymbol{S}$ or $\boldsymbol{S'}$ (Table 10).

Table 10. Share of smokers that can be subsidized under three policies

| | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| % smokers covered | 10 | 10 | 12.20 |

Due to personalized medications, we identify a large extensive margin effect to cover more addicts with differential treatments based on genotypes.

**Quit rates**

Table 11 reports the quit rates of subsidized smokers (weighted average of demographic groups) under the three policies. Note that these quit rates have been adjusted by the relapse rate in one year (51.4%).

Thus, quit rates under $\boldsymbol{S}$ and $\boldsymbol{P}$ are very close since everyone of the H2 or H3 type is treated with both counseling and medication. Quit rates under $\boldsymbol{S'}$ is lower, as some of the H2 or H3 types only receive counseling which is less effective than a combination with medication. Moreover, H1

smokers must have the same quit rates under all policies, because medication does not play any additional role on their cessation outcome. By contrast, H2 and H3 benefit less from policy $\boldsymbol{S}'$.

Table 11. Quit rates under three policies (%)

| | $\boldsymbol{S}$ | $\boldsymbol{S}'$ | $\boldsymbol{P}$ |
|---|---|---|---|
| mean | 22.0 | 18.7 | 22.0 |
| H1 | 21.6 | 21.6 | 21.6 |
| H2 | 22.1 | 18.8 | 22.1 |
| H3 | 22.1 | 16.3 | 22.1 |

**Consumption Equivalent (CE) and Income Equivalent (IE)**

Table 12 reports Consumption Equivalent ($CE$) and Income Equivalent ($IE$) for subsidized smokers.

Table 12. $CE$ and $IE$ of subsidized smokers (%)

| $\boldsymbol{CE}$ | $\boldsymbol{S}$ | $\boldsymbol{S}'$ | $\boldsymbol{P}$ | $\boldsymbol{IE}$ | $\boldsymbol{S}$ | $\boldsymbol{S}'$ | $\boldsymbol{P}$ |
|---|---|---|---|---|---|---|---|
| mean | 8.32 | 7.02 | 8.28 | mean | 6.07 | 5.12 | 6.04 |
| H1 | 6.43 | 6.49 | 6.47 | H1 | 4.80 | 4.84 | 4.83 |
| H2 | 8.82 | 7.53 | 8.75 | H2 | 6.55 | 5.60 | 6.50 |
| H3 | 9.13 | 6.72 | 9.06 | H3 | 6.74 | 4.96 | 6.69 |

On average, $CE$ ($IE$) is about 8% (6%) under policy $\boldsymbol{S}$ or $\boldsymbol{P}$, but about 1 percentage point less under $\boldsymbol{S}'$. The results suggest that the effectiveness of the former two policies is equivalent to increasing consumption by about 8%, or income by about 6%, per year, to a subsidized smoker. Yet, policy $\boldsymbol{S}'$ is not as effective, which is again mainly due to the worse-off of H2 and H3 types under $\boldsymbol{S}'$ as some of them do not receive medication. Accordingly, we observe that H3 smokers receive larger relative gains measured in $CE$ and $IE$ under $\boldsymbol{P}$ than under $\boldsymbol{S}'$ compared with other genotypes, while H1 smokers are almost indifferent between all policies. In addition, H3 smokers receive the largest (absolute) gains measured in $CE$ and $IE$ under $\boldsymbol{S}$ and $\boldsymbol{P}$, suggesting a larger health benefit from cessation for H3 under these policies.

Table 13 shows results for each demographic group by gender, race, and education, where the last six columns report $CE$'s and $IE$'s under the three policies proposed.[28] Notice that in general,

[28] Again, the proportions of three genotypes within each demographic group have been considered when computing the results.

the benefit of a smoking cessation policy increases with work efficiency ($\theta$) given gender. As a consequence, $CE$ under $\boldsymbol{P}$, for example, of white, college, male smokers is highest (16.84%), and of black, non-college, male smokers is the lowest (4.88%). The main reason is the higher opportunity cost of smoking from higher work efficiency. When $\theta$ is higher, income is increased by more as a result of improved health due to quitting, which leads to a larger increase in consumption and health investment, and in turn even better health. Moreover, demographic groups with a relatively larger share of H2 and H3 genotypes (e.g., the white groups) benefit relatively more from $\boldsymbol{P}$ than from $\boldsymbol{S}'$; and women benefit more than men from quitting (given $\theta$) because of a lower natural health deterioration rate.

Table 13. Policy comparison by gender, race, and education (%)

| M | W | C | % | $\boldsymbol{S}(CE)$ | $\boldsymbol{S}'(CE)$ | $\boldsymbol{P}(CE)$ | $\boldsymbol{S}(IE)$ | $\boldsymbol{S}'(IE)$ | $\boldsymbol{P}(IE)$ |
|---|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 5 | 6.63 | 6.20 | 6.61 | 4.98 | 4.65 | 4.96 |
| 0 | 1 | 0 | 45 | 7.82 | 6.55 | 7.77 | 5.83 | 4.88 | 5.79 |
| 0 | 0 | 1 | 0 | 13.77 | 12.59 | 13.76 | 10.18 | 9.31 | 10.17 |
| 0 | 1 | 1 | 5 | 14.63 | 12.13 | 14.60 | 10.80 | 8.95 | 10.77 |
| 1 | 0 | 0 | 7 | 4.89 | 4.50 | 4.88 | 3.67 | 3.37 | 3.65 |
| 1 | 1 | 0 | 34 | 7.63 | 6.44 | 7.62 | 5.65 | 4.77 | 5.65 |
| 1 | 0 | 1 | 1 | 12.36 | 11.30 | 12.36 | 9.14 | 8.36 | 9.14 |
| 1 | 1 | 1 | 3 | 16.87 | 14.55 | 16.84 | 12.05 | 10.40 | 12.03 |

### 5.3.4 Summary

To summarize the results of policy comparison, policy $\boldsymbol{S}$ and $\boldsymbol{P}$ have similar effects in terms of the quit rate and $CE$ or $IE$ for subsidized smokers, but $\boldsymbol{P}$ is able to cover 22% more smokers than $\boldsymbol{S}$, since it saves budget by not subsidizing H1-type smokers for medication while not reducing the effectiveness of treatment. Policy $\boldsymbol{S}'$ is the least effective among the three, as it not only covers fewer smokers, but also results in a lower quit rate, and lower $CE$ and $IE$.

We are now prepared to provide a unified measure of effectiveness. View the 10% or 12.20% subsidy rate as a lottery draw for subsidized treatment with probability $\pi = 0.1$ or 0.122. Then, with probability $1 - \pi$, a smoker would not be subsidized, whose decision remains unchanged and by construction their Consumption or Income Equivalent must be zero. One can therefore compute the expected CE of a smoker as: $\pi \cdot CE + (1 - \pi) \cdot 0 = \pi \cdot CE$. Similarly, the expected $IE$ of a

smoker is: $\pi \cdot IE$. We further construct a dollar measure of effectiveness, multiplying $IE$ by average income of smokers, which is US$36,801 at 2009 constant dollars (or, under $\theta = 0.93$, 282 efficiency wage units). Since average subsidy per smoker is $\$600 \cdot 0.1 = \$60$, the cost/effective (C/E) ratio is simply $60/(\pi \cdot IE)$. For comparison purposes, we also provide expected VSL gains (in 2009 US$ and in %). We report such VSL gain measures and the three unified measures in the last five rows of Table 14.

Table 14. Summary of policy comparison

| | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| % subsidized | 10 | 10 | 12.20 |
| quit rate (%) | 22.0 | 18.7 | 22.0 |
| expected VSL gain per smoker (2009 US$) | 13922 | 11745 | 16894 |
| expected VSL gain per smoker (%) | 0.77 | 0.65 | 0.93 |
| expected $CE$ of a smoker (%) | 0.64 | 0.70 | 1.01 |
| expected $IE$ of a smoker (%) | 0.83 | 0.52 | 0.75 |
| cost/effectiveness ratio (C/E) | 1/3.79 | 1/3.19 | 1/4.59 |

Therefore, under the same program costs (subsidy), personalized treatment provides coverages for more, and maintain high effectiveness. The expected VSL gain under $\boldsymbol{P}$ is $\$16,894$ per smoker or a 0.93% increase from the benchmark value of 1.814 million US dollars (at 2009 constant prices), much higher than the comparable figures under $\boldsymbol{S}$ and $\boldsymbol{S'}$ which are $\$13,922$ and $\$11,745$ (0.77% and 0.65%), respectively. For every dollar of subsidy, it generates 3.79 and 3.19 dollars Income Equivalent in effectiveness under standard treatments $\boldsymbol{S}$ and $\boldsymbol{S'}$, respectively, but 4.59 dollars under personalized treatment $\boldsymbol{P}$. That is, the expected gains from personalized treatment under $\boldsymbol{P}$ are $21-44\%$ higher than standard treatments under $\boldsymbol{S}$ and $\boldsymbol{S'}$. Hence, the results suggest that personalized medication treatment is the most cost-effective.

### 5.4 Counterfactual analyses

As the literature often points to behavioral bias or negative externality created by individual activity as the rationale for government policies (e.g., Chetty, 2015), one might wonder what roles incomplete health knowledge, bounded rationality, or second-hand smoking may have played in the effectiveness of smoking policy here. We thereby conduct counterfactual analyses to reassess our policies when

individuals have complete health knowledge or are fully rational, or when the detrimental health effect of second-hand smoking is absent. We report a summary of results in Table 15, leaving details to Appendix (Table A1-A4).

First, we assume that individuals have complete knowledge about smoking, so that their belief in the detrimental health effect of smoking $\widetilde{\gamma}(t)$ is aligned with the true effect $\gamma(t)$. Row 2 of Table 15 shows that while the relative effectiveness of policy $\boldsymbol{P}$ remains similar as in the benchmark economy (21% and 41% higher than $\boldsymbol{S}$ and $\boldsymbol{S'}$, respectively), the effectiveness of the three policies is lowered by about one-third. In addition, the average life year gain from quitting is 1.7 years less than that in the benchmark economy. In other words, it suggests that incomplete health knowledge has played a role in enhancing the effectiveness of our proposed smoking policies by nearly 50%.

Second, we assume that individuals are fully rational so that they value health consistently over the life course (i.e., $\beta(t) = \overline{\beta}$ for all $t$). In this case, the average life year gain from quitting is similar to that in the benchmark economy while the policy effectiveness is lowered again (by about 13%; see row 3 of Table 15). Thus, bounded rationality has also elevated the policy effectiveness, albeit to a lesser degree than incomplete knowledge.

Finally, we eliminate any detrimental health effect from second-hand smoking by setting $q\overline{s} = 0$; this turns out to increase policy effectiveness considerably (by nearly 60%; see the last row of Table 15). While the average life expectancy is already higher in the absence of second-hand smoking than in the benchmark economy even without quitting (73.3 versus 68.5), the life year gain from quitting is about 4 years more in the counterfacutal scenario, implying a larger gain from smoking cessation policy. This result can be partly explained by the fact that in the benchmark economy, a smoker, even if she herself quits, would still be exposed to second-hand smoke of which the detrimental effect is especially severe when she turns older, mitigating policy effectiveness.[29] Moreover, since a quitter can expect a larger life year gain in the absence of second-hand smoking, she would be incentivized to invest relatively more in health following successful cessation than she would have done in the benchmark case. Nevertheless, while the presence of second-hand smoking seems to dampen policy effects, our results do imply that *given* that second-hand smoking is harmful, it is important to bring down society-wide level of smoking by expanding policy coverage, so that the effectiveness of policy on individual smokers as well as nonsmokers would be improved.

[29] This is related to the fact that the reduction in the society-wide level of smoking (i.e., second-hand smoking) is very small under our proposed policies because the policy coverage rates and quit rates are relatively low.

Table 15. Summary of counterfactual analyses

| cost/effectiveness ratio (C/E) | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| benchmark | 1/3.79 | 1/3.19 | 1/4.59 |
| 1. complete knowledge | 1/2.50 | 1/2.14 | 1/3.02 |
| 2. full rationality | 1/3.25 | 1/2.76 | 1/3.94 |
| 3. no second-hand smoke | 1/5.85 | 1/5.03 | 1/7.11 |

# 6 Robustness analyses

In this section, we conduct several robustness analyses to assure the validity of our policy recommendation. In particular, we consider the cases when policy is implemented for smokers at age 50, or when smokers are able to quit without relapse or without delay. We also reevaluate policy effectiveness when only deadweight cost of treatment is taken into account. Below we discuss the main results while leaving details to Appendix.

## 6.1 Quit at 50

Another critical age for smoking cessation is 50, as smoking becomes much more detrimental to health as one ages. Thus, we examine the effectiveness of policies when they subsidize smokers at age 50. The quit rate is assumed to be unchanged for all groups, whereas cessation delay is set to be 2, 4 and 6 years for H1-H3 types, respectively, based on evidence from Chen et al. (2015a).

The average gain in life years when quitting successfully upon receiving treatment at 50 for all smokers is about 4.5 years, which is about 5 years less than if they receive treatment at 35. It is the lowest for H3 smokers, largely due to the longest delay of cessation. The life year gain varies from 3.0 to 6.9 across demographic groups, and those with higher $\theta$ have slightly higher gain conditional on gender (Table A5 and A6).

In terms of $CE$ and $IE$ (Table A7), now H2 smokers have the largest gains measured in $CE$ or $IE$ under all policies, while H3 still have the largest relative gain from policy $\boldsymbol{P}$ to other policies. That H3 gain less $CE$ and $IE$ than H2 is again due to the longer cessation delay of H3, which turns out to be more detrimental when it occurs at an older age. The relative effectiveness of policy for treating smokers at 50 to treating them at 35 (shown in the last row of Table A8) indicates that all policies are about 40 percent less effective if implemented on smokers at 50 than at 35. Yet the effectiveness of policy $\boldsymbol{P}$ is still 22% and 43% higher than $\boldsymbol{S}$ and $\boldsymbol{S'}$ respectively, very close to the

benchmark value.

### 6.2 Quit without relapse

In the benchmark case, we assume about half of smokers who quit at the beginning relapse and return to smoking in a year. Policy effects may be much larger were there no relapse. Here we reevaluate the same policies but set relapse rates to zero for all groups. While the coverage rates of three policies and life year gains from quitting are the same as in the benchmark, the quit rates are more than doubled now. As a result, $CE$ and $IE$ also more than double the benchmark values, and the C/E ratios are less than half of the benchmark ones (Table A9). The large effectiveness loss due to relapse suggests that a follow-up policy intervention may be necessary to further improve the effectiveness of our proposed policies. Nonetheless, the increase in effectiveness of policy $\boldsymbol{P}$ compared with $\boldsymbol{S}$ and $\boldsymbol{S}'$ is very close to that in the benchmark, which is about 22% and 46%, respectively.

### 6.3 Quit without delay

Another situation worth investigating is that successful cessation happens in the same year of treatment without delay. Cessation delay means smoking for longer years due to addiction. Thus, on the one hand, policy effects on health would be stronger if cessation occurred without delay, especially for H3 smokers who tend to delay for longer. On the other hand, stopping smoking immediately incurs some disutility cost, and, naturally, stronger addiction (measured by $\alpha$) due to genetic markers and/or peer, home and workplace influences make it more difficult to quit, raising disutility cost from quitting. Thus, this disutility cost can be especially high for H3 smokers, and would be even greater if the pre-treatment level of tobacco intake is high. As a result, policy effectiveness measured in $CE$ and $IE$ may either be larger or smaller in the case of no delay. In this regard, we reevaluate the policies assuming there is no delay with successful cessation. In order to compare the effect of delay at different ages, we implement the policies on smokers at age 35 and 50, respectively. We set quit rates to be the same as in the benchmark case.

Table A10 shows that when there is no delay of cessation, the average life year gain from quitting is 10.38 years if being treated (and quit) at 35 and is 5.77 years if being treated (and quit) at 50. These are about 0.6 years and 1.3 years larger than in the cases with delays, respectively. Different from the benchmark case, now among the three genotypes the life year gains are the largest for H3, implying that H3 smokers suffer the largest life year loss from cessation delay. In particular, for H3

smokers cessation delay causes about 1 year and 1.5 years losses in life expectancy if it occurs at 35 and at 50, respectively, larger than the life year losses of other genotypes from cessation delay.

Policy effectiveness measured in $CE$ and $IE$, however, do not all change in the same direction as life year gains. When policy is implemented on smokers at 35, $IE$ is about 5.3% under $\boldsymbol{P}$ for subsidized smokers on average (the first panel of Table A11), which is about 0.8 percentage points lower than the case with delay (see Table 12). The difference is the largest for H3 smokers (5.40% versus 6.69%). This is because the health deterioration effect of smoking at one's 30's is relatively small, while the pre-treatment level of smoking at this age is the among highest over the life course. Thus, strong addiction to smoking outweighs the health benefit from cessation at this age. However, if policy is implemented on smokers at 50, delay reduces $CE$ and $IE$ for all smokers. Comparing the second panel of Table A13 and A9, we observe that $IE$ is reduced substantially for H2 and H3 smokers when there is a delay of cessation. This is because at the older age smoking becomes much more harmful to health and the average amount of smoking is already low even before treatment; health concerns outweigh the enjoyment of smoking.

Table A12 and A13 show that when quitting is not associated with delay, the cost-effectiveness ratio is 1/4.00 and 1/3.35 when policy is implemented at 35 and at 50, respectively. Compared with the delay cases, the effective ratio (defined as the dollar measure of effectiveness in the no-delay case relative to that in the delay case when policy is implemented at the same age) is about 0.87 when smokers are treated at 35, and about 1.24 when smokers are treated at 50, suggesting that cessation delay reduces policy effectiveness only for smokers at an older age. The relative effectiveness of policy $\boldsymbol{P}$ to policy $\boldsymbol{S}$ and $\boldsymbol{S'}$ is very close to the benchmark result in both delay cases, which is about $21-43\%$.

### 6.4 Net deadweight cost

In the benchmark case, the cessation treatment cost includes the monetary cost of treatment incurred to smokers and the government, plus the time cost for smokers. However, the monetary cost is eventually paid to hospitals, pharmaceutical companies and medical professionals; thus, the total cost to the society is not as large. We thereby reevaluate the policies by only considering the deadweight cost. To do this, we add back the total monetary cost of treatment as a lump-sum transfer to subsidized smokers at the subsidized age 35.

Table A14 shows that the results of policy evaluation are very close to the benchmark values. The income effect generated from the lump-sum transfer may either have a positive effect on health

due to a larger health care budget, or a negative effect on health due to more cigarette purchase. The overall effect turns out to be small but positive.

### 6.5 Summary

A summary of the robustness check results is shown in Table 16. Compared with the benchmark case (i.e., policy implemented at 35 and cessation is associated with delay and relapse), policy effectiveness would be lowered by about 40% if it was implemented on smokers at age 50, but would be more than doubled if there was no quitting relapse. The results of the no-delay case depends on the age at which policy is imposed. For smokers at age 35, a delay in cessation increases policy effectiveness since the enjoyment from smoking outweighs health deterioration effects of smoking, while a delay in cessation at 50 has the opposite effect. Considering only the deadweight cost of treatment increases policy effectiveness slightly. In all cases studied, the relative effectiveness of policy $\boldsymbol{P}$ to the two standard policies are very close, which falls within $21-22\%$ to policy $\boldsymbol{S}$ and $42-46\%$ to policy $\boldsymbol{S}'$.

Table 16. Summary of robustness analyses

| cost/effective ratio (C/E) | $\boldsymbol{S}$ | $\boldsymbol{S}'$ | $\boldsymbol{P}$ |
|---|---|---|---|
| benchmark | 1/3.79 | 1/3.19 | 1/4.59 |
| 1. treated at age 50 | 1/2.21 | 1/1.90 | 1/2.70 |
| 2. quit without relapse | 1/8.95 | 1/6.79 | 1/9.91 |
| 3A. quit at 35 without delay | 1/3.28 | 1/2.79 | 1/4.00 |
| 3B. quit at 50 without delay | 1/2.76 | 1/2.35 | 1/3.35 |
| 4. deadweight cost of treatment | 1/3.95 | 1/3.32 | 1/4.79 |

## 7 Conclusions

To address the knowledge gap concerning cost-effectiveness of personalized medicine treatment in smoking cessation, we have developed a lifecycle model with smoking, health investment, savings and life expectancy all being determined endogenously. We have incorporated various aspects of behavioral considerations that justify policy intervention. We have then calibrated the model to fit key economic and medical observations and explored how cost-effective personalized treatment is compared to non-personalized treatments. We have shown that, under the same program costs,

personalized treatment provides coverages for more and maintains high effectiveness. For every dollar of treatment subsidy, personalized treatment generates 4.59 Income Equivalent in effectiveness, $21-44\%$ higher than those under standard treatments. We have also shown that while behavioral bias concerning incomplete health knowledge and bounded rationality can enhance policy effectiveness, the exposure to second hand smoke turns out to mitigate it, highlighting the importance of bringing down society-wide level of smoking by expanding policy coverage. Our finding is robust to various settings regarding no relapses, quitting ages, no quitting delays, and net deadweight cost of medical treatment, where personalized treatment policy yields $21-46\%$ additional effectiveness gain compared to standard policy. We have therefore concluded that personalized treatment is the most cost-effective in smoking cessation by a significant margin.

We would like to acknowledge the limitation of our study, which may also be viewed as a potentially interesting line of future research. Specifically, while we value health and quality of life, it is modeled as a gradual process. Should smoking result in respiratory diseases, especially lung cancer, the quality of life may be lowered sharply and medical cost raised suddenly. Thus, the actual private cost of smoking should be higher than that computed from the model and one may view our effectiveness figure as a conservative measure.

To this end, we note that as genetic tests are increasingly common in medical care and more evidence becomes available to guide medication treatments, this work sets up a study paradigm in gene-guided precision medicine which extends beyond smoking cessation treatments.

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# Appendix
(Not Intended for Publication)

## A. Model: optimization and characterization

To solve the dynamic optimization problem of the model in Section 3, we set up the current-value Hamiltonian for each given value of life expectancy $T$:

$$\begin{aligned}\mathcal{H}(c,s,x,t) &= \ln(c(t)) + \alpha\ln(s(t)) + \beta(t)\ln(h(t)) \\ &\quad +\lambda_1\left[r(t)a(t) + y\left(h(t),t\right) - c(t) - p(t)s(t) - x(t)\right] \\ &\quad +\lambda_2\{\Phi(t)x(t)^{\epsilon} - [\widetilde{\delta} + \widetilde{\gamma}(t)(s(t)+q\overline{s})]\}h(t)^{\mu}\end{aligned} \tag{A1}$$

where $\lambda_1$ and $\lambda_2$ are the costate variables associated with asset accumulation and health capital evolution equations (4) and (5). Note that $\widetilde{\delta}$ can differ from $\delta$ as it contains the individual's "imagined" shock to her health that make her believed health $\widetilde{h}$ different from her true health $h$ (but the shock actually does not exist and the difference between $\widetilde{h}$ and $h$ should be explained by her incomplete knowledge in $\gamma(t)$). For the remainder of the Appendix, we will suppress time index $t$ whenever it does not cause any confusion.

Applying Pontryagin Maximum Principle in optimal control theory, we can obtain three first-order conditions with respect to $c$, $s$ and $x$, and two Euler equations with respect to $a$ and $h$. Straightforward manipulation of these five equations to eliminate the two costate variables leads to the following conditions for the dynamic optimization problem:

$$\frac{s}{c} = \frac{\alpha}{p + \gamma(t)/(\Phi(t)\epsilon x^{\epsilon-1})} \tag{A2}$$

$$\frac{\dot{c}}{c} = r - \rho \tag{A3}$$

$$(1-\epsilon)\frac{\dot{x}}{x} = r + \frac{\dot{\Phi}(t)}{\Phi(t)} - \Phi(t)\epsilon x^{\epsilon-1}h^{\mu}\left(\frac{\beta(t)c}{h} + \theta w\cdot\mathbf{1}_{t\leq 65}\right) \tag{A4}$$

Equation (A2) indicates that smoking relative to consumption increases with the taste for smoking ($\alpha$), but decreases with the price of cigarettes ($p$) and its (relative) health cost ($\gamma(t)/(\Phi(t)\epsilon x^{\epsilon-1})$). When medical treatment cannot effectively mitigate the detrimental effect of smoking on health (i.e., when $\Phi$ is low), it is optimal for an addict to reduce or even quit smoking because her utility enjoyment is outweighed by her health deterioration. Similarly, a rising health cost of smoking as a result of higher medical spending (i.e., higher $x$) also discourages smoking. Furthermore, when one gets older, health investment becomes less effective (i.e., $\Phi'(t) < 0$) and smoking becomes more harmful (i.e., $\gamma'(t) > 0$), thus inducing greater incentives for an addict to reduce or quit smoking.

Equation (A3) is standard in dynamic economic models. It governs the dynamic path of consumption: consumption grows when the rate of return on saving exceeds the subject rate of time discounting. Typically, consumption is expected to grow (that is, $r > \rho$). Equation (A4) governs the dynamic path of health investment. To gain insight from this complicated expression, we note that health capital deterioration when an individual is sufficiently old, together with diminishing returns in health investment with age (i.e., lower $\Phi$ when old) imply that health investment may increase sharply at later ages to maintain health capital above certain threshold. Meanwhile, the

consumption-health capital ratio is expected to increase at least for older individuals. This implies that, from (A4), health investment is likely to decrease eventually when approaching to the end of lifetime, which is consistent with diminishing returns in health investment and biological limitation of its effect on health capital.

Although we cannot solve analytically the entire dynamic paths of consumption, smoking, health investment, health capital and assets, the three expressions above, (A2)-(A4), together with the two evolution equations, (4) and (5), and the boundary condition (7) can be used to solve numerically the five dynamic paths, $\{c(t), s(t), x(t), a(t), h(t)\}_{t=t_0}^{T}$, and the endogenous life expectancy, $T$.

**B. Measures of policy effectiveness**

Mathematically, Consumption Equivalent, or $CE$ is determined by,

$$\int_{t_0}^{T} e^{-\rho(t-t_0)}[\ln(c(1+CE)) + \alpha \ln(s) + \beta(t) \ln(h)]dt = \overline{U} \tag{A5}$$

where $\overline{U}$ is the lifetime utility when the individual is covered by a particular policy. Denote by $U_0$ the untreated lifetime utility in the absence of policy intervention:

$$U_0 \equiv \int_{t_0}^{T} e^{-\rho(t-t_0)}[\ln(c) + \alpha \ln(s) + \beta(t) \ln(h)]dt \tag{A6}$$

we obtain

$$CE = \exp\left[\frac{\rho\left(\overline{U} - U_0\right)}{1 - e^{-\rho(T-t_0)}}\right] - 1 \tag{A7}$$

Mathematically, Income Equivalent, or $IE$ is determined by,

$$\int_{t_0}^{T} e^{-r(t-t_0)}\left[c\left(1+CE\right) + x + ps\right]dt = \int_{t_0}^{T} e^{-r(t-t_0)} y\left(1+IE\right)dt \tag{A8}$$

Applying (A7) and manipulating, we obtain:

$$IE = CE \cdot \frac{\int_{t_0}^{T} e^{-r(t-t_0)}cdt}{\int_{t_0}^{T} e^{-r(t-t_0)}ydt} = \frac{\int_{t_0}^{T} e^{-r(t-t_0)}cdt}{\int_{t_0}^{T} e^{-r(t-t_0)}ydt}\left\{\exp\left[\frac{\rho\left(\overline{U} - U_0\right)}{1 - e^{-\rho(T-t_0)}}\right] - 1\right\} \tag{A9}$$

**C. Counterfactual details and robustness checks**

This appendix provides relevent tables for counterfactual analyses conducted in Section 5.4 and robustness checks performed in Section 6.

Table A1. Gains in life years for three genotypes – counterfactual

| | comp.know. | fully rational | no SHS |
|---|---|---|---|
| mean | 8.02 | 9.65 | 13.95 |
| H1 | 8.04 | 9.93 | 13.41 |
| H2 | 8.19 | 9.64 | 14.15 |
| H3 | 7.78 | 9.44 | 14.12 |

Table A2. Policy comparison – complete knowledge (% except C/E and effective ratios)

| | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| % subsidized | 10 | 10 | 12.20 |
| quit rate | 22.0 | 18.7 | 22.0 |
| expected $CE$ of a smoker | 0.54 | 0.47 | 0.66 |
| expected $IE$ of a smoker | 0.41 | 0.35 | 0.49 |
| cost/effectiveness ratio (C/E) | 1/2.50 | 1/2.14 | 1/3.02 |
| effectiveness ratio | 0.67 | 0.68 | 0.67 |

Note: the effectiveness ratio is the ratio of dollar measure of policy effectiveness under the current experiment to that in the benchmark economy; the same definition applies to all the tables that follow except for Table A13.

Table A3. Policy comparison – full rationality (% except C/E and effective ratios)

| | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| % subsidized | 10 | 10 | 12.20 |
| quit rate | 22.0 | 18.7 | 22.0 |
| expected $CE$ of a smoker | 0.72 | 0.61 | 0.87 |
| expected $IE$ of a smoker | 0.53 | 0.45 | 0.64 |
| cost/effectiveness ratio (C/E) | 1/3.25 | 1/2.76 | 1/3.94 |
| effectiveness ratio | 0.87 | 0.88 | 0.87 |

Table A4. Policy comparison – no second-hand smoke (% except C/E and effective ratios)

| | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| % subsidized | 10 | 10 | 12.20 |
| quit rate | 22.0 | 18.7 | 22.0 |
| expected $CE$ of a smoker | 1.29 | 1.11 | 1.56 |
| expected $IE$ of a smoker | 0.95 | 0.82 | 1.16 |
| cost/effectiveness ratio (C/E) | 1/5.85 | 1/5.03 | 1/7.11 |
| effectiveness ratio | 1.57 | 1.60 | 1.57 |

Table A5. Gains in life years for three genotypes – quit at 50

| | gain in LY |
|---|---|
| mean | 4.48 |
| H1 | 4.53 |
| H2 | 4.63 |
| H3 | 4.24 |

Table A6. Gain in life years for eight demographic groups – quit at 50

| M | W | C | LY-nonquit | LY-quit | gain in LY |
|---|---|---|---|---|---|
| 0 | 0 | 0 | 69.0 | 73.9 | 4.9 |
| 0 | 1 | 0 | 70.2 | 75.2 | 5.0 |
| 0 | 0 | 1 | 72.9 | 79.9 | 6.9 |
| 0 | 1 | 1 | 72.6 | 79.3 | 6.7 |
| 1 | 0 | 0 | 64.9 | 67.9 | 3.0 |
| 1 | 1 | 0 | 66.2 | 69.9 | 3.7 |
| 1 | 0 | 1 | 67.9 | 72.4 | 4.5 |
| 1 | 1 | 1 | 68.2 | 72.9 | 4.7 |

Table A7. $CE$ and $IE$ of subsidized smokers – quit at 50 (%)

| ***CE*** | ***S*** | ***S′*** | ***P*** | ***IE*** | ***S*** | ***S′*** | ***P*** |
|---|---|---|---|---|---|---|---|
| mean | 4.86 | 4.17 | 4.87 | mean | 3.60 | 3.09 | 3.61 |
| H1 | 4.40 | 4.43 | 4.42 | H1 | 3.28 | 3.30 | 3.30 |
| H2 | 5.24 | 4.50 | 5.20 | H2 | 3.89 | 3.34 | 3.86 |
| H3 | 4.69 | 3.50 | 4.78 | H3 | 3.47 | 2.58 | 3.53 |

Table A8. Policy comparison – quit at 50 (% except the C/E and effective ratios)

| | ***S*** | ***S′*** | ***P*** |
|---|---|---|---|
| % subsidized | 10 | 10 | 12.20 |
| quit rate | 22.0 | 18.7 | 22.0 |
| expected $CE$ of a smoker | 0.49 | 0.42 | 0.59 |
| expected $IE$ of a smoker | 0.36 | 0.31 | 0.44 |
| cost/effective ratio (C/E) | 1/2.21 | 1/1.90 | 1/2.70 |
| effective ratio | 0.58 | 0.59 | 0.59 |

Table A9. Policy comparison – quit without relapse (% except the C/E ratio)

| | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| % subsidized | 10 | 10 | 12.20 |
| quit rate | 45.2 | 38.5 | 45.2 |
| expected $CE$ of a smoker | 1.79 | 1.49 | 2.18 |
| expected $IE$ of a smoker | 1.33 | 1.11 | 1.62 |
| cost/effectiveness ratio (C/E) | 1/8.95 | 1/6.79 | 1/9.91 |
| effective ratio | 2.19 | 2.16 | 2.20 |

Table A10. Gains in life years for three genotypes – no delay

| | treated at 35 | treated at 50 |
|---|---|---|
| mean | 10.38 | 5.77 |
| H1 | 9.63 | 5.21 |
| H2 | 10.50 | 6.09 |
| H3 | 10.82 | 5.79 |

Table A11. $IE$ of subsidized smokers (%) – no delay

| treated at 35 | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| mean | 5.34 | 4.55 | 5.35 |
| H1 | 4.61 | 4.65 | 4.65 |
| H2 | 5.69 | 4.87 | 5.72 |
| H3 | 5.45 | 4.03 | 5.40 |

| treated at 50 | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| mean | 4.50 | 3.83 | 4.48 |
| H1 | 3.75 | 3.78 | 3.77 |
| H2 | 5.00 | 4.28 | 4.97 |
| H3 | 4.41 | 3.26 | 4.38 |

Table A12. Policy comparison – quit at 35 without delay (% except C/E and effective ratios)

| | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| % subsidized | 10 | 10 | 12.20 |
| quit rate | 22.0 | 18.7 | 22.0 |
| expected $CE$ of a smoker | 0.72 | 0.61 | 0.88 |
| expected $IE$ of a smoker | 0.53 | 0.46 | 0.65 |
| cost/effectiveness ratio (C/E) | 1/3.28 | 1/2.79 | 1/4.00 |
| effectiveness ratio | 0.87 | 0.87 | 0.87 |

Table A13. Policy comparison – quit at 50 without delay (% except C/E and effective ratios)

| | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| % subsidized | 10 | 10 | 12.20 |
| quit rate | 22.0 | 18.7 | 22.0 |
| expected $CE$ of a smoker | 0.61 | 0.52 | 0.74 |
| expected $IE$ of a smoker | 0.45 | 0.38 | 0.55 |
| cost/effectiveness ratio (C/E) | 1/2.76 | 1/2.35 | 1/3.35 |
| effectiveness ratio | 1.25 | 1.24 | 1.24 |

Note: the effectiveness ratio is the ratio of dollar measure of policy effectiveness under the current experiment to that in the experiment of quitting at 50 with delay; note that this definition is different from that in other tables.

Table A14. Policy comparison – net deadweight cost (% except the C/E ratio)

| | $\boldsymbol{S}$ | $\boldsymbol{S'}$ | $\boldsymbol{P}$ |
|---|---|---|---|
| % subsidized | 10 | 10 | 12.20 |
| quit rate | 22.0 | 18.7 | 22.0 |
| expected $CE$ of a smoker | 0.87 | 0.73 | 1.05 |
| expected $IE$ of a smoker | 0.64 | 0.54 | 0.78 |
| cost/effective ratio (C/E) | 1/3.95 | 1/3.32 | 1/4.79 |
| effectiveness ratio | 1.06 | 1.06 | 1.06 |