Smoking and Mortality: New Evidence from a Long Panel*

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Abstract

Using longitudinal data over nearly 50 years from the Framingham Heart Study, we provide causal estimates of the expected longevity consequences of different lifetime smoking patterns by jointly modeling individual smoking and health and by allowing for correlated unobserved heterogeneity. We simulate our estimated empirical model, and we compare the resulting mortality differences to the epidemiological literature that treats smoking behavior as random. The unconditional difference-in- means in age of death between lifelong smokers and nonsmokers is 9.3 years in our research sample, while simulations from our estimated dynamic model suggest the difference is only 4.3 years.

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1 Introduction

Smoking is currently considered the leading preventable cause of death in the United States. According to the Centers for Disease Control, smoking causes 480,000 deaths each year and 8.6 million people have at least one serious illness due to smoking. Cigarette smoking is the primary causal factor in lung cancer and is a key risk factor in coronary heart disease.[1] In addition to the obvious negative health consequences of smoking, the medical and epidemiological literatures contend that quitting smoking has significant benefits. For example, ten years after quitting, an individual faces a cancer risk one-third to one-half as large as if he had continued smoking (Doll *et al.*, 2004). If a smoker quits smoking before age 40, she avoids 90 percent of the excess mortality attributed to smoking (Pirie *et al.*, 2013; Jha *et al.*, 2013). Our research suggests that the accepted morbidity and mortality improvements accompanying smoking cessation are overstated by as much as 50 percent.

There is ample biological evidence linking smoking to deleterious health outcomes. Yet, some puzzling aggregate trends demonstrate our concern with the literature's calculations of these impacts. Over the last twenty-five years adult smoking rates for both genders have fallen steadily to about half their initial level, but the incidence of lung and bronchus cancer has doubled for women while declining for men. This variation could stem from heterogeneity — in individual characteristics as well as in smoking history — among the fifty million former smokers. While quitting smoking might suspend additional contributions to poor health, the precise nature of one's smoking history still predisposes that individual to cancers, heart disease, and other diseases.[2] Our research carefully explains contemporaneous smoking and health over one's adult lifetime (ages 30 to 100) while capturing the importance of variation in individual smoking and health histories.

Making accurate assessments of the longevity losses from cigarette smoking and the longevity gains from smoking cessation has proved difficult because each individual is solely responsible for his or her smoking history. Ideally, the gain/loss predictions should be calculated from observed mortality differences following random assignment of lifetime smoking behavior. Because random variation of this kind does not exist, researchers must rely on observational data to measure the effects of smoking on morbidity and mortality outcomes. When using non-experimental data, however, identification of

[1] For a good review of national trends in cigarette smoking and a summary of smoking-attributable diseases, see United States Surgeon General (2014).

[2] An additional explanation for the gender differences in lung cancer rates could be competing risks from other diseases (Honore and Lleras-Muney, 2006), yet mortality rates from cardiovascular diseases also fell more steeply for men during this period.

the causal effect of smoking on mortality is difficult precisely because observed smoking behavior over one's lifetime is *not* random: individuals initiate smoking, may choose to quit smoking, and sometimes fail at quitting (i.e., relapse). These endogenous behaviors, which occur at all different ages, produce very different lifetime smoking patterns. Therefore, the first contribution of this paper is determination of unbiased impacts of the varying histories of smoking through joint estimation of smoking behaviors and health outcomes at frequent intervals over the life cycle (i.e., smoking histories are not exogenous).

An interrelated concern leading to difficulty in assessment of smoking's impact is that morbidity and mortality, while certainly not random, may be attributable to observed and unobserved non-smoking factors. It may be the case that failure to control for heterogeneity that explains correlation in smoking behavior and other behaviors that adversely influence health (e.g., excessive alcohol consumption, drug use, poor nutrition, etc.) leads to an overstatement of the health effects of smoking. Alternatively, ignoring the factors that explain, for example, the inverse smoking/obesity correlation may understate the influence of smoking. While these correlations may be explained by observed individual variation, it is quite likely that unobserved characteristics such as risk-aversion, time preference or self-esteem (which may not vary much over time) and unobserved stress and health (which may be time-varying) influence observed smoking and health patterns over the life cycle.[3] The second contribution of this paper is its generous inclusion of theoretically-justified controls for both observed (in our data) and unobserved (yet econometrically relevant) individual heterogeneity in order to uncover the casual relationship between cigarette smoking and morbidity and mortality outcomes (i.e., confounding factors may influence health outcomes).

To achieve these empirical contributions we leverage a panel dataset of smoking behavior and health outcomes obtained at frequent intervals (via medical exams and survey questions) throughout much of an adult lifetime. Since the early 1950s, the Framingham Heart Study (FHS) has followed three generations of participants in order to identify contributors to heart disease.[4] Since 1948, most of the 5209 subjects of the original cohort have returned to the study every two years (if alive) for a detailed medical history, physical examination, and laboratory tests. The non-death attrition

[3]There is even evidence that differences in the health of certain regions of the brain influence the propensity to quit, and that these neural differences also influence other behaviors (Naqvi *et al.*, 2007).

[4]The original objective of the FHS, directed by National Heart Institute (now known as the National Heart, Lung, and Blood Institute or NHLBI), was to identify the common factors or characteristics that contribute to cardiovascular disease (CVD). Additional cohorts — offspring of the original cohort (1971), a more diverse sample (1994), and a third generation (2002) — have been recruited and are being followed. (`www.framinghamheartstudy.org`).

rate of only three percent mitigates a typical source of selection bias.

This long-term, ongoing study consists of contemporaneous responses; it relies on recall of participants only to identify age of smoking initiation. We use 46 years of longitudinal observations (23 waves) on the original cohort adult participants to construct detailed smoking histories (including duration, quits, and relapse). Among adults, an important smoking transition is quitting (or attempts to quit), as most initiation occurs during adolescence. Often, depictions of individual smoking histories rely on less accurate, retrospective data gathered at a few disperse intervals making accurate identification of quits almost impossible. Our detailed, reliable histories and the modeling of behavior every two years allow us to simulate a range of quitting behaviors — quits at different ages, quits after different smoking durations, and quits with different cessation lengths — in order to evaluate the resulting impact on lifespan and cause of death.

Additionally, much of the available health information in FHS is gathered during frequent (about every two years) detailed medical exams. Health events (e.g., diagnoses of heart disease, cancer, diabetes) are dated and measures of risk factors (e.g., weight, blood pressure, cholesterol) are documented. With such detailed smoking and health data over time, we can model the dynamic effects of smoking histories on health as well as the dynamic effects of health histories on smoking behaviors. To help establish identification, we collect new data on historical cigarette prices and advertising for over a century which we interact with age to get variation across individuals and time. We provide quasi-experimental evidence that these supply shifters are causally related to smoking levels.

These data are combined with an econometric methodology that explicitly addresses several omissions from the literature in a unified framework. First, our empirical model allows for dynamics; smoking behaviors and health outcomes in the past influence smoking behavior and health outcomes contemporaneously. Second, we estimate these behaviors and outcomes jointly and allow for rich observable controls as well as individual unobserved heterogeneity that generates correlation in the observed endogenous variables we seek to explain. Third, we estimate smoking and health over much of the adult lifetime ensuring a well-explained model of life-cycle outcomes for simulation and evaluation.

Figure 1 illustrates the advantages of our methods and these data. Figure 1a depicts the survival curves, by lifetime smoking pattern, generated using the observed age of death and smoking history of original cohort participants of the FHS. Figure 1b depicts survival rates calculated using data simulated from an estimated dynamic model of smoking behavior and health outcomes that includes the heterogeneity discussed above. While both figures indicate that smokers have, on average, higher mortality rates than

non-smokers at all ages, the differences between the two groups are noticeably smaller when we account for non-random selection (Figure 1b) than when we simply examine the raw data (Figure 1a).[5]

Figure 1: Survival Curves by Lifetime Smoking Pattern

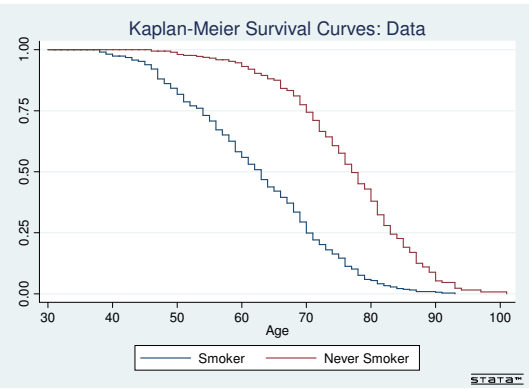


Figure 1a

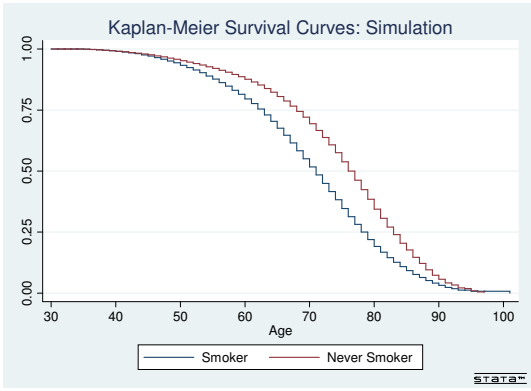


Figure 1b

Notes: Figure 1a depicts the proportion of individuals in our estimation sample who remain alive at each age. Figure 1b depicts the survival rates *of the same individuals as in Figure a.* based on simulations from our preferred model when we impose the two lifetime smoking patterns. Smoker defines an individual who smoked from initiation until death; never smoker defines an individual who never smoked.

We contend that these different findings are explained by the two selection issues highlighted above: 1.) lifetime smoking patterns reflect choices of individuals who differ in observed and unobserved ways (i.e., smoking histories are not exogenous), and 2.) individuals in the two groups may differ in observed and unobserved ways that positively or negatively contribute to mortality (i.e., confounding factors influence health outcomes). For example, the first type of selection is demonstrated if individuals who chose to smoke and those who choose not to smoke are different in observed and unobserved ways that affect mortality differently. Additionally (but not depicted above since the figure reflects survival curves of lifelong smokers and never smokers only), former smokers may have been light (vs. heavy) smokers (and, likely, are in better relative health) who find it easier to quit. Alternatively, individuals in poor health may be more inclined to quit smoking than those in better health. The non-random selection into quitting will result in an observed benefit that also reflects bias. The second type of selection results if smokers are less healthy in ways (other than smoking) that increase the probability of death. The non-random selection into mortality that reflects correlation with other-cause behaviors will overstate mortality of smokers. Hence, observational

[5]The same comparisons among current and former smokers reveal qualitatively similar patterns.

differences (Figure 1a) tend to overstate the mortality consequences of smoking.

Our results suggest that the dynamic specification, which captures the feedback effects of smoking behavior on health and in turn mortality; the rich controls for observed and unobserved heterogeneity; and the joint modeling of smoking and health over the life cycle have sizable impacts on the mortality effects of smoking and smoking cessation. Using the data on smoking and mortality alone, differences in the distribution of age of death by lifetime smoking behavior suggest that continuing to smoke reduces mean life expectancy by nine years relative to those who never smoke. However, simulations from our estimated model, which allows for several sources of individual heterogeneity, show a reduction of just 4.3 years. We find a similar difference when we condition on cause of death. For example, for those who die of cardiovascular disease (CVD), the mean age of death is 9.3 years older for never smokers compared with continual smokers. Our simulations suggest that this difference is only 3.9 years. We also show that unobserved heterogeneity (UH) matters.[6] We find large differences in simulated smoking patterns when we condition on different unobserved types. Overall, our results confirm that smoking does indeed reduce expected longevity; however, we find that failing to control for heterogeneity in smoking histories, health histories, and individual unobservables overstates the magnitude of these reductions. These results are important contributions to public health discussions. For example, our results echo conventional wisdom in some regards: twenty years of smoking experience has little impact on life expectancy if the individual quits by age 40. Additionally, we find that quits of five years followed by relapses have little benefit in terms of life expectancy. The latter suggests that cessation programs without follow-up support for former smokers will not be effective.

The next section discusses the current state of knowledge on the health consequences of smoking, and shows how the approach and rich data that we use advances the literature. Section 3 describes how we constructed our research sample and details the structure of these data. We use Section 4 to introduce notation, to explain the empirical model, and to summarize the variables used in estimation. Section 5 provides results: parameter estimates, model fit, and simulations. We end with a discussion of the policy relevance of our findings.

[6]Throughout the paper we will distinguish between observed and unobserved individual heterogeneity, yet we remind the reader that our emphasis is that "heterogeneity" matters. The variables that we observe are data specific; a researcher cannot possibly control for everything. Yet, our joint estimation procedure allows us to flexibly soak up, or control for, the variation that is unobserved to us and correlated across outcomes. For brevity, we abbreviate unobserved heterogeneity by UH henceforth.

2 Background

2.1 Medical Literature

Research from the epidemiological literature that seeks to understand the impact of smoking/quitting on mortality uses very limited, if any, empirical strategies to address bias associated with non-random selection or confounding behaviors. The heavily cited work of Doll *et al.* (1994) and Doll *et al.* (2004) employs panel data on British doctors over a forty and fifty year period, respectively. The authors compare mortality rates of physicians within 30-year birth cohorts by smoking history. They find that physicians who are current smokers of cigarettes (and who habitually smoked cigarettes) died on average ten years earlier than those who never smoked. Furthermore, quitting smoking at ages 60, 50, 40, and 30, relative to continuing to smoke, implies an increase in longevity of 3, 6, 9, and 10 years, respectively. The researchers condition on birth cohort and age only. There is no attempt to account for endogenous selection into different lifetime patterns of smoking. While the authors acknowledge that their findings may reflect a positive correlation between smoking and alcohol consumption (and hence the smoking effect may be biased upward), they claim that confounding factors reflected in other causes of death "are unlikely to have influenced greatly the absolute difference between the overall mortality rates of cigarette smokers and lifelong non-smokers" (Doll *et al.*, 2004).

Many of the United States Surgeon General's conclusions about the impact of smoking on health (United States Surgeon General, 2004) are based on the Peto *et al.* (2000) study that matches individuals diagnosed with lung cancer (cases) with observationally similar individuals who did not have cancer (controls). The relative risk of cancer from smoking, compared to not smoking, is based on the ratio of smokers among the cases and controls. This case-control approach only accounts for a small set of observed individual-level characteristics, such as gender and age. The restricted observed variation and absent unobserved variation limits our full understanding of the smoking-morbidity-mortality relationship. Furthermore, this approach often relies on information collected at a single point in time (which means smoking histories are reported retrospectively and potentially with recall error) and only allows for analysis of single outcomes (such as a disease or the mortality rate) independently.

2.2 Importance of Observed Individual Heterogeneity

Another approach to minimize bias has been a more thorough inclusion of observed individual-level heterogeneity. For example, some analyses have included objective mea-

sures of health (such as BMI or cholesterol) or behaviors correlated with health (such as exercise or drinking). Others have excluded individuals with pre-existing health conditions (National Cancer Institute, 1997) in order to avoid attributing other-cause deaths to smoking. Treating this additional heterogeneity as uncorrelated with the unexplained health outcome error introduces an endogeneity bias if there are common unobservables that influence both morbidity/mortality and the included variables. Also, it is likely that these additional variables are correlated with smoking behavior or, more importantly, that individual-level UH is correlated across the lifetime smoking categories, the included observed heterogeneity (if not exogenous), and morbidity/mortality.

More recently, economists have begun exploring the health consequences of smoking. One of the early economic analyses used smoking status of individuals (described by non-smoker, current smoker, or former smoker and years since quitting) to quantify the mortality benefit of quitting relative to continuing smoking over the subsequent 14-year period (Taylor *et al.*, 2002). However, smoking behavior was treated as exogenous and was ascertained at enrollment only (so quits are based on retrospective and potentially noisy responses and continuing behavior is assumed of current smokers). Additionally, the empirical specification does not account for differences in smoking duration among smokers. The primary control for heterogeneity was exclusion of individuals who were sick in the initial period. While this sample selection mitigates bias due to poor-health-induced quits, it likely overstates the benefit of cessation since continuing smokers typically engage in less healthy behaviors than do former smokers (United States Surgeon General, 1990).

2.3 Importance of Individual Unobserved Heterogeneity

In order to address the important role of individual UH that influences both smoking behaviors and health outcomes, economists have used different econometric techniques to identify a causal impact. Evans and Ringel (1999) consider the impact of cigarette taxes on the smoking behavior of pregnant women, and the subsequent effect of smoking (and quitting smoking) on birth outcomes. Although the authors are not modeling own health outcomes, their work stresses the importance of accounting for smoking endogeniety as quitting is likely impacted by observables such as taxes as well as unobservables that may be correlated with infant outcomes. Clark and Etil (2002) use data from the first seven waves of the British Household Panel Survey (BHPS) to determine how responsive adult smoking behavior is to health changes. Because they have multiple observations on the same individuals they estimate cigarette demand in first differences using GMM estimation and in levels using twice (or more) lagged variables to

instrument lagged demand. Both approaches address endogeneity bias that results from permanent individual unobservables (in the former case) and unobservables that are not autocorrelated of order greater than one (in the latter case). Their work contributes to the evidence of a dynamic relationship between smoking and health: namely that health changes precede smoking reductions. They suggest that the sample of remaining smokers is, therefore, not random.

Bedard and Deschenes (2006) identify the effect of smoking on premature mortality by studying mortality rate differences between cohorts who were more likely to have served in the United States military and thus to have received subsidized cigarettes. They find that cohorts with higher veteran rates experienced excess premature mortality, and a large proportion of the excess mortality was due to smoking-attributable diseases such as lung cancer.[7] Their analysis, however, does not include individual-level heterogeneity, does not include smoking history such as duration or quits, and relies on linear cohort controls which make it difficult to separately identify cohort, age, and year effects. Balia and Jones (2011) emphasize the important role of UH in reducing bias in the measured effect of smoking on mortality. Their latent factor model uses smoking behavior of parents and other household members to exogenously shift an individual's smoking initiation and cessation while having no direct impact on his own mortality.

2.4 Importance of Smoking and Health Dynamics

By solving and estimating a dynamic model of cigarette consumption and mortality where forward-looking individuals explicitly take into account the health consequences of their smoking behaviors, Adda and Lechene (2001) find evidence that individuals with (observed and unobserved) characteristics that result in a higher risk of mortality (net of tobacco-related mortality) are less likely to quit or reduce smoking. The importance of modeling smoking and quitting decisions over the life course, especially when interested in the impact of smoking on morbidity and mortality, is further emphasized by the work of Khwaja (2010) who estimates a life-cycle model of endogenous health input decisions (including health insurance, medical care utilization, alcohol consumption, exercise, and smoking) using panel data on near elderly individuals as a way to correct for dynamic selection associated with survivorship. In addition to the evidence that smoking behaviors are impacted by health, which necessitates the modeling of both observed and unobserved individual characteristics that may be correlated with quitting

[7] More recently, Carter *et al.* (2015) suggest that much of the excess mortality among current smokers may be due to associations with diseases that have not been formally established as caused by cigarette smoking.

and continuation, Adda and Lechene (2013) demonstrate that individuals in poorer non-smoking-related health are more likely to initiate smoking.[8]

We build on this literature, which emphasizes the importance of smoking endogeneity. Our first addition stems from our data source. The dataset consists of a very long panel that allows us to accurately observe each individual's smoking dynamics, including quits and relapses. Because the time between observations is short there is little issue of recall error. Also, a common medical examination given at each observation provides uniformity in the health variables. The second addition is in our modeling of smoking behavior. We use explanatory factors that vary over time, cohort, and/or individual to model the smoking choices in each period. The modeling of the observed choices each period allows us to estimate unbiased marginal effects of very detailed smoking histories. The estimated dynamic model, in turn, allows us to simulate the morbidity and mortality outcomes resulting from a variety of policy- and behavior-relevant smoking decisions. For example, we quantify how the life expectancy benefit of quitting smoking varies by smoking history (i.e., the return for a long time smoker vs. a smoker with a history of relapse).

2.5 Theoretical Considerations of Smoking and Health

The theoretical approach to smoking framed by economists is one that emphasizes several important aspects of the smoking/health relationship. Models of the demand for health, initially proposed by Grossman (1972) and extended by several others including most recently Kohn (2008) and Galama (2011), emphasize the role of individual health production and the resulting demand for health inputs. Health is valuable because it determines available time for activities that provide either monetary compensation and/or non-pecuniary reward. The rational addiction model and its variants (Becker and Murphy, 1988; Gruber and Koszegi, 2001; Bernheim and Rangel, 2004) suggest reasons why forward-looking individuals continue to smoke despite its impact on health. Namely, one's history of smoking behavior affects the enjoyment one receives from smoking today as well as withdrawal costs. Thus, individual characteristics such as time preference, risk aversion, and health expectations are important for explaining observed behaviors. Economists also explore the role of information in helping individuals form expectations of uncertainties such as future health and the impact of smoking on own health. While there is evidence that expectations about future longevity are relatively

[8]Their findings, both on the relationship between health and smoking initiation and between smoking and mortality, are based on cross-sectional and some very short panel data, an attempt to capture UH with a time-invariant index of health conditions that are not directly caused by smoking according to the epidemiology literature, and no information on quitting or relapse.

accurate (Viscusi, 1990; Smith *et al.*, 2001; Viscusi and Hakes, 2008), economists have also discovered that individuals respond, with changes in their own smoking behavior, to information that is more personal (e.g., own health decline and shocks, own health markers, and parental health shocks) than general (e.g., Surgeon General's reports, spousal health shocks).[9]

2.6 Overview of Our Approach

In light of these theoretical contributions and empirical explorations, our approach is to estimate, using a panel of individuals followed for much of their adult lives, a dynamic model that approximates the forward-looking decisionmaking that results in observed life-cycle smoking behaviors and health outcomes. Importantly, our empirical model explains smoking decisions over time as a function of one's health and smoking histories. In turn, that history of smoking behavior impacts morbidity and mortality. In addition to rich observed variation in individual health characteristics, we model both permanent and time-varying UH using a non-linear latent factor approach, or correlated random effects. Causal impacts of smoking history on health outcomes are identified by time-varying exogenous covariates, theoretically-relevant exclusion restrictions, the dynamic relationships in the set of estimated equations, covariance restrictions on the UH, and non-linear estimators. Using FHS data to construct our research sample, we follow 1,464 men for up to 46 years with censoring determined by either death or the end of the available sample period.[10] The data include a variety of health marker measures such as cholesterol and blood pressure, cardiovascular health measures, cancer diagnoses, and smoking information obtained at each health exam approximately two years apart. Eighty-eight percent of our sample die during the observed time frame. After showing that our preferred random effects specification fits the observed smoking and health data well, we simulate the model under different lifetime smoking scenarios to assess the effects of smoking, and smoking cessation, on morbidity and mortality outcomes.

3 Description of the Research Sample

3.1 The Framingham Heart Study

The original cohort of the FHS is well-suited for our analysis because it follows a group of men and women aged 30 to 62 in 1948 who receive a medical examination every two

[9] See Smith *et al.* (2001); Sloan *et al.* (2002); Darden (2012); Darden and Gilleskie (2015).

[10] We have permission to use the requested set of variables through 1996 only. We were able to obtain death dates of sample participants through 2009.

years (on average) to the present. Longitudinal datasets of this length are rare in health and economics. The U.S. Public Health Service began collecting this information in an effort to identify biological and environmental factors contributing to the rapidly rising rates of cardiovascular death and disability.

The FHS original cohort sample consists of two-thirds of the adult population of Framingham, Massachusetts in 1948. The main drawback of these data, for our purposes, is that the sample is drawn from a single, small town with very little racial and ethnic variation. As a result, geographic and demographic characteristics are limited. Additionally, because the focus of data collection is on health, there is no information on socioeconomic characteristics such as income and only limited information on employment or occupation. Finally, it is challenging to measure the sensitivity of smoking to prices, taxes, or regulations, which in our sample vary over time but not across individuals within a year.[11]

The breadth of health data, however, is overwhelming. Theoretically, measures of height and weight, blood pressure, cholesterol, cancer, diabetes, arthritis, death, and cause of death are available each medical exam (every two years). Realistically, values of these health variables are missing sporadically. Smoking behaviors are also available in great detail. The smoking intensity questions, however, are different over time making it impossible to construct measures of smoking history beyond the extensive margin. Other health behaviors are available at points in time, but may not be ascertained at each exam.

3.2 Determination of Research Sample

To estimate our dynamic model of lifetime smoking and health outcomes we would like to have data at each exam (approximately every two years) for each participant. If smoking behavior is missing for any exam(s) it becomes difficult to construct variables that accurately reflect the history of one's behavior (e.g., years of cessation, duration, and experience). Missing smoking observations require that we either impute behavior or drop individuals from our research sample. Table 1 details our progress with assigning smoking behavior during missing exams by using observed individual responses from other exams. As our sequentially-applied assumptions introduce more potential noise, the resulting sample size increases. Ultimately, we select a sample of men (sample E)

[11] As we explain later, we gathered detailed data on cigarette prices and advertising expenditures from the late 19th century throughout the 20th century in order to explain differences in contemporaneous smoking behaviors as well as initially-observed smoking histories. We use the variation in age each year to examine possible age-related responses to cigarette price variation over time. Additionally, we use the price and expenditure variation at the different reported ages of smoking initiation.

that minimizes this type of imputation while maximizing the number of individuals we can follow.

It remains to purge the sample of individuals with missing important variables. Because individuals enter the study between the ages of 30 and 62, many already have a "positive" history of smoking participation as well as a health history that indicates onset of chronic conditions. We use responses from contemporaneous and retrospective questions administered at the first few exams to construct these initial condition variables. For this reason the initial conditions are represented in our research sample as exam 2 variables and we begin modeling dynamic smoking transitions and health outcomes beginning in exam 3 (around the year 1952). If an individual died before the second exam period or we were unable to construct the necessary initial smoking and health history information, we can not include them in our research sample. Our final research sample for this analysis consists of up to 22 biennial observations on 1464 (i.e., 1754 - 290) men beginning in 1952, providing 21,198 person-year observations.[12]

3.3 Structure of Research Sample

Table 2 characterizes our research sample over time. Recall that we model smoking behavior and health outcomes beginning with exam 2, which was administered to a participant sometime between the years 1950 and 1955.[13] Ages of individuals at this exam range from 32 to 65. Because we have data on the same individuals through 1996 (when ages of those still alive range from 74 to 101), we are able to observe the age and cause of death for 87.8 percent of our sample of men (1285 out of 1464). Cause of death is categorized into cardiovascular related, cancer related, and other. Cardiovascular disease (CVD) includes myocardial infarction, angina pectoris, coronary heart disease, stroke, and heart failure. We know the type of cancer (within classes) that an individual acquired, but we aggregate all cancers in our analysis due to sample size limitations.[14] Note that CVD and cancer account for over two-thirds of all observed deaths in our sample, which is comparable to U.S. death rates for this age group during this time frame. Also, deaths related to CVD appear to occur at a higher rate at younger ages, perhaps linked to the national trend where death from cardiovascular disease has fallen substantially over the last 50 years.[15]

Table 2 confirms that the percentage of current cigarette smokers declines over

[12]Because of likely differences by gender in determinants of smoking and impacts of smoking on

Table 1: Research Sample Selection based on Assignment Method for Missing Smoking Values

Assumptions about smoking behaviors used to determine research sample	Lifetime smoking observed (% of individuals)	Smoking values missing (% of person-years)	Resulting sample size	Research sample name
Begin with original sample of males who have at least two consecutive exams (N=2274). Use reported per-exam smoking info	27.4	22.5	622	A
+ fill in missing per-exam smoking values with data from subsequent retrospective questions	32.5	19.9	739	B
+ fill in up to 4 consecutive missing values when same smoking choice observed before & after missing spell	55.1	13.2	1253	C
+ fill in all missing values when at least 50% of smoking choices are observed and that smoking behavior is the same	55.6	12.9	1265	D
+ fill in missing values before death when at least 3 consecutive exams of the same smoking behavior is observed before death	77.1	8.1	1754	E
+ fill in missing values before death with last observed smoking choice before death	85.1	4.2	1935	F

Table 2: Characterization of Research Sample over Time

FHS exam number	Calendar years	Empirical model period (t)	Sample size at t	Average age at t	Death* by end of t	Cause of death** if died in t		Smoke in t
						CVD	Cancer	
2	1950-55	1	1464	46.6	0.0	0.0	0.0	55.8
3	1952-56	2	1464	48.6	1.7	64.0	20.0	58.8
4	1954-58	3	1439	50.5	2.2	58.1	22.6	59.4
5	1956-60	4	1408	52.3	2.6	62.2	13.5	53.6
6	1958-63	5	1371	54.2	2.7	67.6	24.3	52.4
7	1960-64	6	1334	55.9	3.2	47.6	31.0	52.6
8	1962-66	7	1292	57.7	3.3	65.1	16.3	47.3
9	1964-68	8	1249	59.6	4.4	47.3	27.3	42.1
10	1966-70	9	1194	61.3	4.4	39.6	43.4	37.4
11	1968-71	10	1141	63.1	5.6	46.9	28.1	30.2
12	1971-74	11	1077	64.7	7.1	47.4	18.4	29.2
13	1972-76	12	1001	66.2	7.5	54.7	29.3	27.8
14	1975-78	13	926	67.8	7.1	37.9	34.9	24.4
15	1977-79	14	860	69.3	9.8	40.5	27.4	21.7
16	1979-82	15	776	70.8	10.3	38.6	33.8	19.3
17	1981-84	16	696	72.4	14.8	35.9	23.3	16.0
18	1983-85	17	593	73.7	13.2	32.1	21.8	15.0
19	1985-88	18	515	75.3	16.5	35.3	25.9	13.2
20	1986-90	19	430	76.6	13.0	37.5	33.9	10.5
21	1988-92	20	374	78.2	13.1	38.8	26.5	9.4
22	1990-94	21	325	79.7	17.2	30.4	21.4	7.1
23	1992-96	22	269	81.3	33.5	20.0	22.2	6.7
Total person-observations			21,198	60.6	6.5	42.1	26.3	38.6

Note: $*$ conditional on survival up to t (i.e., the death hazard).
$**$ omitted category is Other.

the sample period.[16] This decline reflects quits as well as selective mortality, and we emphasize both in our empirical approach. It is important to measure well the different smoking histories of individuals that accumulate as they age. Rather than rely on retrospective data, the FHS allows us to observe smoking behavior at frequent intervals over much of adult life. Hence, the specific age one quits smoking, as well as ages of relapse, are observed. In our sample of men, 24 percent never smoke. Smoking initiation typically begins at young ages. In fact, only four percent of our sample had both never smoked before we initially observe them and smoked at some point in our data. (Among those with no observed smoking history in 1952 (28 percent of the research sample), only 13 percent initiate smoking before death or 1996, whichever comes first.) Twenty-seven percent of the sample smoke continuously (i.e., every period they are observed once they begin smoking). Among those men we observe ever smoking, 49 percent quit smoking at least once with a 10.1 percent person-period quit rate. Of the men that quit, 74 percent of them do not restart.[17] Among those who relapse within our period of observation, the mean time of smoking cessation between spells of smoking is 3.3 years. The mean age of relapse is 53.9 years. These figures emphasize the non-random patterns of smoking histories and the importance of a long panel consisting of frequent interviews and limited dependence on recall.

Table 3 details the distribution of age of death and cause of death among those who die during our sample period. By exam 23, 87.8 percent of the individuals have died; the average age of those still alive by the end of this exam period is 81.3.[18] We distinguish deaths by lifetime smoking pattern (i.e., never smoked, smoker in exam before death, and quit smoking before age 50) and by cause (i.e., cardiovascular disease, cancer, and other). The overall mean age of death (conditional on being observed to die) was 72.6

health, we examine males separately.

[13]Note that calendar years may overlap across exam numbers.

[14]United States Surgeon General (2004) reports that smoking raises the risk of many types of cancer, not only lung cancer.

[15]This positive trend has been attributed to both reductions in smoking (and other risk factors, despite increases in obesity and diabetes) and advances in medical care (Prince *et al.*, 2014).

[16]In general, U.S. smoking rates among this cohort have declined over time from 55.8 and 31.8 percent, for males and females respectively, to 6.6 and 7.8 percent among those still alive 45 years later. As discussed above, there were regulatory changes in the cigarette market as well as dissemination of information about cigarettes during this period.

[17]This figure includes quits that may have occurred prior to the beginning of the study. The relapse rate is likely understated because the length of time between reported smoking measures is two years. Among all person-periods where an individual did not smoke last period but had ever smoked, the rate of relapse is 37.5 percent.

[18]Life expectancy of men born in Massachusetts between 1885 and 1918 (i.e., birth years of men in the original FHS cohort) conditional on reaching age forty was 67 to 69 years (Bureau of the Census, 1949).

years of age. Men who die of cardiovascular disease and cancer die, on average, at ages 70.8 and 71.9 respectively, while those who die of other causes live to age 75.5 on average (not shown in table). For men who never smoke, the average age of death is 75.6 years; and for men who report smoking immediately prior to death, the mean age of death is 66.2 years of age. The difference of 9.3 years is similar to the result reported in Doll *et al.* (2004). Ever smoking is also associated with a higher proportion of cancer-related deaths. Interestingly, the raw data suggest that smoking cessation by age 50 results in a comparable lifespan to those who never smoked (yet they still experience cancer-related deaths in a higher proportion). The Surgeon General Reports use similar life year gains to advocate for smoking cessation programs. We demonstrate with our lifetime model of smoking and health that these figures are biased upward.

Table 3: Distribution of Age and Cause of Death by Lifetime Smoking Pattern

	Age of death distribution (percentile)						Cause of death		
Smoking pattern	Mean	10th	25th	50th	75th	90th	CVD	Cancer	Other
Unconditional on smoking	72.6	58	66	74	80	85	42.1	26.3	31.6
Never smoked	75.6	64	70	76	82	87	44.4	19.9	35.8
Smoked in exam prior to death	66.2	52	59	67	74	79	44.3	28.8	26.9
Quit smoking before age 50	74.1	63	70	75	80	84	39.4	29.2	31.4

Note: Statistics conditional on death by exam 23 (87.8 percent of sample).

4 Empirical Framework

Our goal in this section is to describe an empirically-implementable model that captures the dynamic considerations of forward-looking individuals making optimal smoking decisions in light of uncertain health evolution. The key features of the theory from which we derive our empirical model are: (i) individuals care about discounted lifetime utility; (ii) individuals derive utility from smoking (and other consumption), (iii) the marginal utility of smoking may depend on past levels of smoking, (iv) utility also depends on current health, and (iv) smoking histories, health histories, and current smoking behavior impact the distribution of future health outcomes.

4.1 Theoretical Foundation and Derived Empirical Model

To be more specific and to define notation, we write down the individual's optimization problem using a Bellman formulation.[19] From this problem we derive an estimable equation for smoking demand. The lifetime value of each smoking alternative depends on information known by the individual when entering each decision-making period. The information set, denoted Ω_t, includes the vector H_t^S representing an individual's history of smoking decisions up to period t; the vector H_t^D representing his history of diseases up to period t; the vector X_t of exogenous demographic variables in t; and the vector P_t of exogenous period t prices and supply-side characteristics related to the consumption/health input goods. The individual also has private information about his preferences for smoking and his expectations about disease and mortality transitions, denoted by the vector $u_t = [u_t^S, u_t^D, u_t^M]$. Conditional on being alive to make a smoking decision, the lifetime value of smoking alternative $s_t = s$ is

$$\begin{aligned} V_s(\Omega_t, u_t) \;=\; & \sum_{d=0}^{D} p(d_t = d|H_t^D, H_t^S, X_t)[U(c_t, s_t = s; H_t^S, d_t = d) + u_t^s \qquad (1) \\ & +\beta(1 - p(m_{t+1} = 1|H_{t+1}^D, H_{t+1}^S, X_t)V(\Omega_{t+1})] \quad \forall t, s = 0, 1 \end{aligned}$$

where utility is constrained by the per-period budget, $c_t = y_t - P_t s_t$; the price of general consumption c_t is normalized to one; and y_t measures income in period t.[20] We capture uncertain health transitions by $p(d_t = d|\cdot)$ where d_t represents a vector of disease states taking on the value d. Current utility depends on the disease state. Contemporaneous utility also depends on one's smoking history, H_t^S, to capture tolerance, reinforcement, and withdrawal effects that vary with an individual's past smoking behaviors. To characterize future utility (line 2 of equation 1), we define β as a measure of how forward-looking an individual may be (i.e., the discount factor); we allow for an absorbing mortality state stochastically with $p(m_{t+1} = 1|\cdot)$ where the value of death is normalized to zero; and we describe the maximal expected value of future utility (unconditional on the future smoking alternative) by $V(\Omega_{t+1}) = E_t[\max_s V_s(\Omega_{t+1}, u_{t+1}^s)]$. The dynamic optimization problem allows smoking and disease histories to impact expected current utility and allows those histories and current behavior and health to affect expectations about future utility.

Optimal smoking decisionmaking requires backward solution from a final period

[19] Individual subscripts n are dropped to simplify notation.

[20] For simplicity, we take income as given and do not model employment, marital, or savings decisions nor the effect of health on these behaviors. This decision is predicated by the fact that the FHS data do not contain this information.

characterized by certain death. Analytic solution also requires functional form assumptions for several components of the problem including the utility function, the disease production function, the mortality function, and the utility error term distribution. Theoretically, the optimization problem can be solved to obtain a decision rule for smoking of the form

$$p(s_t = s) = f(s_t^*) \text{ where } s_t^* = s(H_t^S, H_t^D, X_t, P_t, u_t^S),\ s = 0, 1 \quad \forall t. \tag{2}$$

Notice that the demand (for smoking) equation is a function of all information available to the individual at the beginning of the decision-making period. Specifically, the vector H_t^S (capturing smoking history up to period t) includes previous period smoking status, s_{t-1}; the length of smoking cessation up to t, C_t,; the length of smoking duration up to t, D_t; and the length of smoking experience up to t, E_t.[21] The smoking history is updated at the end of period t (i.e., H_{t+1}^S) to reflect smoking choices made at t. Importantly, the specification also includes exogenous supply-side characteristics of the cigarette market, P_t (e.g., prices, advertising), that vary over time.[22]

An individual faces uncertain health outcomes each period. We model the health production function as

$$p(d_t = d) = f(d_t^*) \text{ where } d^* = d(H_t^D, H_t^S, X_t, u_t^D),\ d = 0, \ldots, D \quad \forall t \tag{3}$$

where the "disease" variable may take on several discrete values. In practice, we estimate the number of cardiovascular disease (CVD) events in period t; the probability of cancer diagnosis (by the end of period t conditional on no cancer diagnosis up to t); the probability of diabetes diagnosis (conditional on no diabetes diagnosis up to t); and body mass in period t.[23] The vector H_t^D (capturing disease history up to period t) includes variables constructed from the health outcomes that are modeled (i.e., any CVD events entering period t, the number of CVD events entering period t, cancer diagnosis ever, diabetes diagnosis ever, and BMI in period $t-1$) as well as variables that we treat as exogenous that also describe one's health (i.e., per-period systolic blood pressure, diastolic blood pressure, cholesterol levels, and an indicator of arthritis).[24]

[21] It would be computationally infeasible to include indicators of smoking behavior at each age to explain smoking behavior and health outcomes at the current age.

[22] We return to discussion of these theoretically-important variables when we discuss initial conditions and identification below.

[23] Body mass is modeled as a continuous distribution of the body mass index (BMI), which is a normalized function of height and weight.

[24] The medical literature tells us quite plainly that blood pressure and cholesterol are impacted by smoking. For the sake of parsimony, we have chosen to present results from a smaller model where these outcomes are not jointly modeled with the set of equations defined below. Conclusions about the

Mortality, or the probability of death at the end of period t (i.e., an individual dies before making it to the next exam) is

$$p(m_{t+1} = m) = f(m^*_{t+1}) \text{ where } m^* = m(H^D_{t+1}, H^S_{t+1}, X_t, u^M_t),\ m = 0, 1 \quad \forall t. \tag{4}$$

Because death is an absorbing state, decision rules and health production are conditioned on being alive in period t. Non-random mortality, therefore, creates important selection into the sample of (remaining) individuals whose characteristics explain the modeled smoking behaviors and health outcomes. Note that the probability of survival to the next period $(t + 1)$ depends on the updated disease and smoking histories.

As should be evident, the period t demand for smoking is identified by variation in period t supply-side conditions (P_t) that, according to an economic theory of individual optimizing behavior, impact the smoking decision but do not independently impact health production or mortality conditional on period t smoking behavior. In other words, equations 2, 3, and 4 form a set of structural (demand and production) equations that can be empirically identified and estimated. These dynamic equations explain smoking choices, s_t, and health outcomes, d_t and m_{t+1}, from periods $t = 2$ to $t = 22$, where t denotes the two-year period between exams in our data.

4.2 Initial Conditions

Because we first observe individuals between the ages of 32 and 65 (in period $t = 2$), we must account for the endogeneity of initially-observed smoking history [denoted by E_2 (smoking experience entering $t = 2$ where $E_2 = 0$ implies never smoked and $E_2 > 0$ implies ever smoked), s_1 (smoking status in $t = 1$ conditional on ever smoking prior to $t = 2$), and D_2 (years of smoking duration entering $t = 2$ conditional on smoking in $t = 1$)] as well as disease history [denoted by CVD_2 (any CVD events entering $t = 2$) and BMI_1 (body mass index in $t = 1$)].[25] For simplicity we denote the three initial smoking variables entering period $t = 2$ by the vector I^S_2 and the two initial disease variables entering period $t = 2$ by the vector I^D_2. We specify the initial conditions by

effect of smoking on morbidity and mortality were not appreciably different using the larger model. Arthritis, however, appears to have little association with smoking (to our knowledge), but does impact health transitions.

[25] We do not include equations for initially-observed diabetes or cancer because very few individuals in our sample enter the FHS with these diseases. We also exclude an equation for initially-observed years of smoking cessation conditional on having quit prior to the first health exam because the small number of quitters does not provide enough variation for estimation.

the following non-dynamic equations[26]

$$\begin{aligned} I_2^S &\equiv [E_2, S_1, D_2] = s'(X_1, P_1, Z_1, u_1^{S'}) \\ I_2^D &\equiv [\text{CVD}_2, \text{BMI}_1] = d'(X_1, P_1, Z_1, u_1^{D'}) \end{aligned} \tag{5}$$

The initial condition equations are included in the set of jointly-estimated structural equations. Note that these initial condition equations are static and expressed in their reduced form; they do not contain any lagged endogenous variables. In addition to the exogenous cigarette market characteristics, we also include exogenous shifters, denotes Z_1, to aid in identification (discussed below).

4.3 Individual-level Unobserved Heterogeneity

Unobserved individual characteristics (i.e., latent heterogeneity) also impact smoking demand, morbidity, and mortality (represented by equations 2-5). It is important to model this UH for several reasons. First, it is reasonable to believe that unobserved individual differences impact smoking behavior and health. In fact, the ability of observed variables to explain health outcomes is notoriously low. These differences include permanent unobserved characteristics such as health-related genetic endowments or cohort effects and non-health related personality or preference characteristics. They also include differences such as unobserved health events or stress events that vary over time. Second, the outcomes we model are functions of endogenous explanatory variables and, as such, the error term in the equation of interest is correlated with the explanatory variable, creating endogeniety bias in the estimated coefficients. Third, measurement error cannot be ruled out, so allowing for a source of this error reduces measurement error bias in marginal effects of interest. For example, individuals who smoke may smoke at different intensities. Accounting for these unobserved differences is necessary for obtaining unbiased causal impacts of the variables of interest. To model these potential sources of UH, the composite error term in each equation j, u_t^j, is decomposed into three parts: a permanent individual heterogeneity component (μ), a time-varying individual heterogeneity component (ν_t), and an idiosyncratic component (ϵ_t). More specifically, $u_t^j = \mu^j + \nu_t^j + \epsilon_t^j$. We allow the latent heterogeneity captured by μ and ν_t to be correlated across equations and assume that ϵ_t is a vector of independent and identically-distributed errors (Extreme Value or Normally distributed depending on the estimator).

The permanent heterogeneity, which is correlated across outcomes and over time,

[26]The prime superscript on the functions and the error terms is meant to distinguish them from the previously defined dynamic smoking and disease functions and their unobservables.

is captured by the joint distribution of $\mu = [\mu^S, \mu^D, \mu^M, \mu^{S'}, \mu^{D'}]$. The time-varying heterogeneity is defined by the joint distribution of $\nu_t = [\nu_t^S, \nu_t^D, \nu_t^M]$.[27] We could assume that these multivariate distributions are normal, for example, and estimate the cross-equation correlation coefficients along with the coefficients on the observable covariates. However, we do not wish to impose a specific distribution. Rather, we model the UH as random effects and approximate their unknown distributions discretely, estimating both the discrete mass points along the support of the unobserved components as well as the associated probability weights (termed a Discrete Factor Random Effects (DFRE) method or latent factor method). This flexible estimation technique (Heckman and Singer, 1984; Mroz and Guilkey, 1992; Cunha and Heckman, 2008) does not impose a specific distribution on the error terms as is standard with many maximum likelihood techniques.[28] Additionally, the discrete distributions of the random effects add only a fraction of the additional parameters (and associated loss in degrees of freedom) required by the fixed-effects method (which would be inconsistent in nonlinear models).[29]

The latent factor approach allows individual characteristics that are unobserved by the researcher to impact all jointly estimated equations (in a non linear way) and integrates over their distributions when constructing the likelihood function. The con-

[27]Time-varying heterogeneity does not enter the equations for the variables describing one's initial smoking and health histories entering period two because those variables summarize behavior and outcomes from all periods prior to inclusion in the study.

[28]Using Monte Carlo simulation, Mroz (1999) shows that when the true distribution of the error terms is jointly normal the DFRE method performs as well as maximum likelihood estimation assuming normality. When the simulated distribution is not normal, the DFRE method performs better in terms of precision and bias. Mroz (1999) and Guilkey and Lance (2014) describe the econometric properties of the DFRE estimator using Monte Carlo studies.

[29]While the method we use is called a "random effects estimator," it is important to recognize that the estimated "random effect" is not assumed to be independent of endogenous explanatory variables, provided that we model the dependence of such endogenous explanatory variables and outcome of interest on the random factor. Any explanatory variable that we do not explicitly model as a function of the random factor is assumed to be independent of the random factor.

tribution of individual n to the likelihood function, unconditional on the UH, is

$$
\begin{aligned}
L_n(\Theta,\mu,\nu_t,\rho,\psi) \;=\; & \sum_{k=1}^{K}\rho_k\Bigg\{\prod_{i=0}^{I}p(I_2^S=i|\mu_k^{S'})^{I_{n2}^{S',i}}\prod_{j=0}^{J}p(I_2^D=j|\mu_k^{D'})^{I_{n2}^{D',j}} \\
& \times\prod_{t=2}^{T}\sum_{\ell=1}^{L}\psi_\ell\Bigg[p(s_t=1|\mu_k^S,\nu_{t\ell}^S)^{S_{nt}}\big[1-p(s_t=0|\mu_k^S,\nu_{t\ell}^S)\big]^{(1-S_{nt})} \\
& \times\prod_{d=1}^{4}p(D_t^d=1|\mu_k^{D^d},\nu_{t\ell}^{D^d})^{D_{nt}^d}\big[1-p(\mathrm{D}^d{}_t=1|\mu_k^{D^d},\nu_{t\ell}^{D^d})\big]^{(1-D_{nt}^d)} \\
& \times p(m_{t+1}=1|\mu_k^M,\nu_{t\ell}^M)^{M_{nt+1}}\big[1-p(m_{t+1}=0|\mu_k^M,\nu_{t\ell}^M)\big]^{(1-M_{nt+1})}\Bigg]\Bigg\}
\end{aligned}
$$

where Θ defines the vector of parameters of the model and $p(\cdot)$ represents the logit or multinomial logit probabilities (or densities) of the observed behaviors and outcomes. The vectors ρ and ψ denote mass-point specific estimates of the joint probabilities of the permanent and time-varying heterogeneity, respectively. ρ_k is the estimated joint probability of the k^{th} permanent mass point, which is given by

$$\rho_k = \mathrm{P}(\mu^{I^S}=\mu_k^{I^S},\mu^{I^D}=\mu_k^{I^D},\mu^S=\mu_k^S,\mu^{D^1}=\mu_k^{D^1},\ldots,\mu^{D^4}=\mu_k^{D^4},\mu^M=\mu_k^M\ ).$$

ψ_ℓ is the estimated joint probability of the ℓ^{th} time-varying mass point and is given by

$$\psi_\ell = \mathrm{P}(\nu_t^S=\nu_{t\ell}^S,\nu_{t\ell}^{D^1}=\nu_{t\ell}^{D^1},\ldots,\nu_t^{D^4}=\nu_{t\ell}^{D^4},\nu_t^M=\nu_{t\ell}^M\ ).$$

4.4 Variables used in the Empirical Specification

Table 4 summarizes the dependent variables describing the dynamic smoking behavior and health outcomes that we seek to explain. The morbidity measures of disease that we model over time include number of cardiovascular disease events, cancer diagnosis, diabetes diagnosis, and body mass index, while the mortality measures are death and cause of death. Appendix Table A1 lists all the equations that are jointly estimated, the functional form of each equation, and the endogenous, exogenous, and unobserved variables described in equations 2-5. The specification of each equation (i.e., how these explanatory variables enter the equations) includes higher moments and interactions of some variables if relevant since the equations represent nth order approximations of the non-linear and dynamic demand and production functions.

The equation system of smoking behavior and health outcomes captures the inherent dynamics over an individual's lifetime. Namely, observed smoking and morbidity

Table 4: Dependent Variables in the Jointly-Estimated Set of Equations

Variable	Mean	SD
Smoking behavior, S_t		
Smoke at t	0.386	0.487
Morbidity outcomes, D_t^d		
CVD events at t		
0 CVD events (omitted category)	0.946	0.225
1 CVD event	0.042	0.201
2+ CVD events	0.012	0.107
* Ever had CVD event up to t (person-exams)	0.198	0.399
* Ever had CVD event (persons)	0.484	0.500
* Number of CVD events up to t; [0, 14]	0.339	0.843
Cancer diagnosis at t | no cancer up to t	0.013	0.112
* Ever diagnosed with cancer up to t (person-exams)	0.048	0.214
* Ever diagnosed with cancer (persons)	0.179	0.383
Diabetes diagnosis at t | no diabetes up to t	0.011	0.102
* Ever diagnosed with diabetes up to t (person-exams)	0.061	0.240
* Ever diagnosed with diabetes (persons)	0.145	0.352
Body Mass Index/10; [1.4, 5.4]	2.644	0.350
Mortality outcomes, M_{t+1}		
Death hazard at t	0.065	0.247
Cause of death		
CVD	0.421	0.494
Cancer	0.263	0.440
Other (omitted category)	0.316	0.465
Initial conditions, $I_2^{S'}, I_2^{D'}$		
Never smoked	0.272	0.445
Current smoker | ever smoked	0.863	0.344
Years of smoking/10 | current smoker; [0.2, 5.5]	2.512	0.938
Any CVD	0.034	0.182
Body Mass Index/10; [1.7, 4.0]	2.588	0.338

Note: Starred rows are additional statistics, not dependent variables.
Ranges of continuous variables are in brackets.

outcomes depend on information known at the beginning of the period. Specifically, they depend on one's smoking history and disease history up to the current period. Table 5 provides summary statistics for explanatory variables entering period t (i.e., those that enter the smoking and disease equations). Mortality occurs during the period (or prior to period $t+1$) and depends on the *updated* histories of these variables. That is, the probability of death before period $t+1$ (conditional on not dying prior to period t) depends on one's smoking and disease histories up to period t as well as behavior and disease outcomes in period t. All equation specifications also include polynomials and interaction terms, and a flexible time trend as explanatory variables.

4.5 Identification

Having defined all of the equations in our jointly-estimated system, we can now thoroughly discuss identification. Our main equations for smoking and health are dynamic (i.e., depend on endogenous past outcomes) and we model (estimate) the endogenous smoking behavior and health outcomes for all observed periods spanning up to 46 years of a person's lifetime. This dynamic specification allows previous exogenous covariates to serve as implicit instrumental variables for the lagged endogenous variables (Arellano and Bond, 1991; Bhargava and Sargan, 1983). That is, they directly influence past (but not current) behavior. Recall that these dynamic equations include health markers (i.e., blood pressure and cholesterol levels) and arthritis, which we treat as exogenous. These smoking equations also include theoretically-justified, supply-side, exogenous variables that influence cigarette demand, namely the mean real price of cigarettes at time t (for 5 cartons or 1000 cigarettes in year 2000 dollars) and real per capita expenditures on cigarette advertising (in year 2000 dollars) at time t, denoted by P_t. The cigarette market characteristics do not impact period t health outcomes conditional on observed smoking behavior. Because individuals live in the same community, cigarette advertising and price vary over time but not across individuals. Thus, we interact these time-varying variables with age and previous smoking status. We discuss the cigarette market data below.

Because dynamic equations cannot explain our initially-observed endogenous variables (i.e., smoking and health histories up to the point we first observe someone in our sample), we specify unique static equations that are jointly estimated with the main dynamic equations. Theory tells us that smoking demand in period t is a function of one's smoking history, H_t^S. Dynamic substitution confirms that period t smoking demand is a function of initial smoking history, H_2^S, entering period $t=2$ (the first period we can model smoking behavior dynamically). The equations for each variable defining

Table 5: Explanatory Variables Entering Period t

Variable	Abbreviation	Mean	SD	Min	Max
Time-varying variables					
Endogenous					
Smoker in $t-1$	$\texttt{S}_{t-1}$	0.409	0.492	0	1
Years of cessation entering t	$\texttt{C}_t$	5.035	9.799	0	68
Years of duration entering t	$\texttt{D}_t$	13.238	18.317	0	74
Years of experience entering t	$\texttt{E}_t$	21.208	18.585	0	74
1 CVD event in $t-1$	$\texttt{1[CVD}_{t-1}=1\texttt{]}$	0.037	0.189	0	1
2+ CVD events in $t-1$	$\texttt{1[CVD}_{t-1}>1\texttt{]}$	0.011	0.106	0	1
Ever had CVD event up to $t-1$	$\texttt{E\_CVD}_{t-1}$	0.167	0.373	0	1
Number of CVD events up to $t-1$	$\texttt{N\_CVD}_{t-1}$	0.274	0.742	0	12
Cancer diagnosed in $t-1$	$\texttt{CAN}_{t-1}$	0.010	0.098	0	1
Ever diagnosed with cancer up to $t-1$	$\texttt{E\_CAN}_{t-1}$	0.036	0.187	0	1
Diabetes diagnosed in $t-1$	$\texttt{DIA}_{t-1}$	0.010	0.099	0	1
Ever diagnosed with diabetes up to $t-1$	$\texttt{E\_DIA}_{t-1}$	0.052	0.222	0	1
Body mass index in $t-1$	$\texttt{BMI}_{t-1}$	26.436	3.458	13	54
Exogenous					
Systolic blood pressure in $t-1$	$\texttt{SBP}_{t-1}$	136.422	20.538	80	260
Diastolic blood pressure in $t-1$	$\texttt{DBP}_{t-1}$	81.934	11.595	38	140
Cholesterol level in $t-1$	$\texttt{CHO}_{t-1}$	222.752	39.461	81	551
Arthritis in $t-1$	$\texttt{ART}_{t-1}$	0.268	0.443	0	1
`BMI, SBP, DBP` missing		0.063	0.243	0	1
`CHO` missing		0.131	0.338	0	1
Age (years)		60.597	12.170	32	101
Time-invariant variables (Exogenous)					
Education: grade school		0.274	0.446	0	1
Education: some high school		0.161	0.367	0	1
Education: high school degree		0.282	0.450	0	1
Education: some college		0.087	0.281	0	1
Education: college degree		0.098	0.298	0	1
Education: post college		0.098	0.298	0	1
Born outside U.S.		0.170	0.376	0	1
Italian ancestry		0.233	0.423	0	1
Older cohort: Age 50+ at $t=1$		0.309	0.462	0	1

Note: Table summarizes variables entering period t that explain smoking and disease in t. Mortality at end of t depends on updated endogenous variables that include period t smoking and disease. Equation specifications also contain interactions, polynomials, and time trends.

smoking history entering period $t = 2$, I_2^S, are functions of the initial cigarette market characteristics, P_1, which we observe. Because one's initially-observed disease history also depends on previous smoking behavior, we include cigarette market characteristics in the equations that explain initial cardiovascular disease and body mass, I_2^D, and do not include individual smoking history.

Appendix C provides intuition on why these these supply shifters would causally influence demand. The argument is relatively straightforward for prices, but one could be concerned that advertising simply shifts demand between brands. There is indirect evidence that advertising has a causal impact on smoking rates. In the late 1960s and early 1970s a series of government regulations restricted and then banned cigarette ads on television and radio.[30] Appendix Figure C.2 shows that real advertising spending rose continuously in the decades before and after these regulations, but fell by forty percent in the five-year period after the regulations were introduced. This reduction is almost surely driven by regulations and not changes in demand or new information about the consequences of smoking (i.e., it is several years after the 1964 Surgeon General's Report). Cigarette consumption for adults fell over five percent during the beginning of this period, while it rose for the decades before and just after the ban. (See Table 2 in American Lung Association (2011); Figure 2 in Harris (1979).)

The cigarette market data we use come from a variety of sources discussed in Appendix C and from which we assembled a time series of cigarette prices and industry-wide advertising by cigarette companies from 1893 to 2009. In the main equations, we use the data from 1950-1994 to represent time-varying market characteristics affecting contemporaneous smoking behavior. We use the data through 2009 in simulations, based on our estimated model, until death. We use the data from 1895-1939 to account for variation in the cigarette market that may have influenced smoking behavior early in one's life and, in particular, smoking initiation. Recall that individuals are different ages (i.e., 32 to 65) when we originally observe them. Hence, they were "young" at different points in history. We argue that variation in cigarette prices and advertising *when individuals in the FHS were in their teenage years* may explain propensities to begin smoking. Indeed, we show that real per-capita cigarette advertising expenditures *averaged over the years in which an individual was between 10 and 14 (or 15 and 18)* positively predict the initially-observed smoking history when an individual enters the FHS. Our "advertising expenditure during the ages of 10 and 14 (or 15 and 18)" variable is not perfectly correlated with age or calendar year because ages at the first exam vary and the first exam of each individual in the FHS was administered sometime between

[30]In 1968 the FCC required TV and radio stations to air anti-cigarette commercials if they also broadcast cigarette ads. In 1971 federal law banned all cigarette ads on TV and radio.

1948 and 1953.[31] So, for example, when the Supreme Court dissolved the Tobacco Trust in 1911 cigarette prices fell and advertising rose. These changes are likely to have a different effect on older men of our sample (who were adults at the time of the breakup) relative to younger men. Support for this sort of differential effect is presented in the literature review in Appendix C.

In addition to the time-varying cigarette market characteristics that serve as a source of exogenous variation that impacts the initial conditions, we also include information about sibling structure in the initial condition equations . We argue that smoking initiation, which generally occurs at young ages, may be influenced by siblings or one's ordering among siblings (Gilleskie and Strumpf, 2005; Kelly *et al.*, 2011). These variables include the number of siblings, an indicator of being an only child, a linear birth order variable, and an indicator of being a first-born child. The coefficients on these variables are jointly significant in the initial condition equations, and are not significant in the main equations once we control for smoking and health histories. We provide summary statistics for variables that capture the cigarette market and sibling structure in Table 6. To summarize, our model parameters are (over-)identified using theoretically-relevant exclusion restrictions where appropriate, the entire history of exogenous time-varying variables given the dynamic equation specification, covariance restrictions associated with estimation of the UH, and non-linear estimators.

[31] A historical cigarette prices variable was similarly constructed to reflect average prices during an individual's teenage years. This variable did not satisfy the identification criteria and is, therefore, not used in the initial conditions equations.

Table 6: Variables that Serve in Identification

Variable	Mean	SD	Min	Max
Cigarette market (values in year 2000 $)				
Mean cigarette price for 5 cartons in year t - using years 1996-2009*	212.87	49.65	129.52	283.79
Mean cigarette price for 5 cartons in year t - using years 1950-1994	87.45	13.84	70.07	125.97
Advertising expenditure per capita in year t - using years 1996-2009*	3.07	1.35	1.29	5.10
Advertising expenditure per capita in year t - using years 1950-1994	6.57	1.93	2.83	10.60
Advertising expenditure per capita at ages 15-18 - using years 1899-1939	2.21	1.85	0.01	5.92
Advertising expenditure per capita at ages 10-14 - using years 1895-1935	1.82	1.80	0.07	5.92
Sibling structure				
Number of siblings	4.45	2.89	0	20
Only child	0.04	0.20	0	1
Birth order (up to 5th)	2.75	1.49	1	5
First born child	0.27	0.45	0	1
Sibling information missing	0.18	0.38	0	1

Note: * 1996-2009 values are used when simulating behavior beyond our sample observation period. The price and expenditure time series are depicted in Appendix C.

5 Results

Our empirical analysis begins with estimation of the 12 equation system (see Appendix Table A1) — representing smoking behavior (1 equation), morbidity outcomes (4 equations), mortality outcomes (2 equations), and initial conditions (5 equations) — that allows for individual-level correlation across equations and over time. We discuss parameter estimates from two versions of the model: one that allows for individual-level UH (our preferred model) and one that does not. Having estimated the parameters, we demonstrate the ability of our preferred model to fit the observed data. We also show that the model is able to predict out-of-sample (post-1996) ages of death for the 12 percent of individuals in the estimation sample who had not died by 1996. We then use the model to simulate morbidity and mortality outcomes for a wide array of smoking patterns that could be exhibited by individuals over the life course. These results, compared to those from models typically used in this literature, demonstrate the differences we find regarding the impact of smoking cessation on morbidity and mortality.

5.1 Parameter Estimates

Our preferred model (labeled 'with UH') is the one that explicitly introduces and estimates the UH that might be correlated with smoking behavior and morbidity/mortality in order to capture the selection inherent in smoking history variation and the confounding associated with health outcomes variation. Table 7 presents estimates of and standard errors on coefficients of selected determinants of smoking. It is difficult to infer marginal effects from this table, since the variables are part of a dynamic (e.g., smoking history is defined by lagged smoking as well as years of smoking duration, experience, and cessation) and larger system (e.g., smoking history impacts health history which also influences current smoking behavior).[32]

As expected, the point estimate on lagged smoking is large and significant, suggesting state dependence in smoking. Furthermore, the longer one has smoked the more likely he is to continue smoking (at a diminishing rate). With regard to the endogenous health variables, blood pressure has a significant effect (negative for SBP and positive for DBP) and a CVD event in the previous period reduces the probability of smoking. Body mass and cholesterol levels in the previous period do not impact the probability

[32]We calculate (and discuss in the next sections) marginal effects using simulation techniques to account for the dynamic feedback and large number of polynomials and interactions in the specification. We focus our discussion of the effects of smoking and health histories. Other exogenous determinants included in each equation are age, education, ancestry, origin, cohort, and year trends (and results are available from the authors). Estimates of the UH contributions and their distributions are presented in Appendix Table A2. Appendix Table A3 provides results for the initial condition equations.

of smoking.

For completeness, we also provide coefficient estimates and standard errors from a model with no UH (i.e., the equations are estimated separately and coefficients on endogenous variables reflect bias associated with selection and confounding). The 'without unobserved heterogeneity' model (labeled 'without UH') extends the models often used by practitioners and policymakers to measure the impact of smoking on health outcomes and to calculate the benefits of smoking cessation by including a richer description of observed smoking and disease histories. Yet, it does not attempt to capture potential (permanent and time-varying) individual UH and, therefore, may still produce biased impacts of endogenous smoking and health histories despite providing an improved fit. Indeed, changes in the significance and, for some, signs of endogenous variables is evident in the table. A comparison of the estimates from the model without UH (columns 1 and 2) and with UH (columns 3 and 4) reveals differences in the significance and functional relationship between one's smoking history and his propensity to smoke currently. In particular, notice that when UH is allowed variation in years of smoking cessation no longer significantly impacts contemporaneous smoking, while additional years of smoking duration and experience increase the probability of smoking at a decreasing rate. (The sizes of the coefficients also exhibit differences, but changes in marginal effects are difficult to assess at this point. We further examine marginal effects, and address the role of UH, after we introduce simulations from the models.)

Before proceeding, we note the significance of the cigarette market variables in the smoking equations. These variables, serving as a source of identification, are not significant when included in the morbidity and mortality equations conditional on one's smoking history. The cigarette price and advertising variables impact smoking but appear to do so in an unexpected direction. We estimated the model using prices and advertising expenditures in level terms and got the expected signs: prices have a negative effect on the smoking probability while advertising expenditure has a positive effect. We do not include these level variables in our main specification because it is hard to disentangle these terms, which vary only over time, from temporal effects.

Table 7: Selected Parameter Estimates: Contemporaneous Smoking

	Without UH			With UH		
Variable	Estimate	Std. error		Estimate	Std. error	
Smoker in $t-1$, $\texttt{S}_{t-1}$	0.784	0.337	**	1.631	0.871	*
Years of cessation, $\texttt{C}_t$	0.260	0.061	***	-0.109	0.086	
$\texttt{C}_t^2/100$	-3.146	0.553	***	-0.576	0.653	
Years of duration, $\texttt{D}_t$	0.110	0.010	***	0.151	0.025	***
$\texttt{D}_t^2/100$	-0.134	0.021	***	-0.156	0.041	***
Years of experience, $\texttt{E}_t$	0.105	0.011	***	0.197	0.022	***
$\texttt{E}_t^2/100$	-0.115	0.021	***	-0.207	0.042	***
$\texttt{1[CVD}_{t-1}=1\texttt{]}$	-0.447	0.213	**	-0.538	0.296	*
$\texttt{1[CVD}_{t-1}>1\texttt{]}$	-0.684	0.324	**	-1.216	0.557	**
$\texttt{CAN}_{t-1}$	-0.620	0.452		-0.389	0.674	
$\texttt{DIA}_{t-1}$	0.129	0.495		0.386	0.574	
$\texttt{E\_CVD}_{t-1}$	-0.929	0.285	***	-1.394	0.355	***
$\texttt{N\_CVD}_{t-1}$	0.242	0.112	**	0.404	0.113	***
$\texttt{E\_CAN}_{t-1}$	-0.118	0.487		-0.135	0.540	
$\texttt{E\_DIA}_{t-1}$	-0.228	0.410		-0.578	0.508	
$\texttt{E\_CVD}_{t-1}$ * $\texttt{S}_{t-1}$	0.714	0.249	***	0.893	0.330	***
$\texttt{E\_CAN}_{t-1}$ * $\texttt{S}_{t-1}$	0.232	0.544		0.110	0.662	
$\texttt{E\_DIA}_{t-1}$ * $\texttt{S}_{t-1}$	0.592	0.467		0.376	0.695	
$\texttt{BMI}_{t-1}$	-0.069	0.104		-0.193	0.238	
$\texttt{BMI}_{t-1}^2/100$	0.078	0.193		0.220	0.441	
$\texttt{SBP}_{t-1}$	0.039	0.015	***	0.044	0.020	**
$\texttt{SBP}_{t-1}^2/100$	-0.013	0.005	**	-0.015	0.007	**
$\texttt{DBP}_{t-1}$	-0.068	0.027	**	-0.067	0.033	**
$\texttt{DBP}_{t-1}^2/100$	0.036	0.016	**	0.035	0.020	*
$\texttt{CHO}_{t-1}$	0.006	0.005		0.009	0.007	
$\texttt{CHO}_{t-1}^2/100$	-0.001	0.001		-0.002	0.002	
BMI, SBP, DBP missing	-1.000	1.559		-2.799	3.342	
CHO missing	0.918	0.673		1.327	0.908	
Cigarette price at t * $\text{Age}_t/10$	0.327	0.100	***	0.467	0.151	***
Cigarette price at t squared/100 * $\text{Age}_t/10$	-0.231	0.058	***	-0.314	0.088	***
Cigarette price at t * $\text{Age}_t/10$ * $\texttt{S}_{t-1}$	0.029	0.009	***	0.019	0.012	
Ad expenditure at t * $\text{Age}_t/10$	-0.300	0.083	***	-0.273	0.105	***
Ad expenditure at t * $\text{Age}_t/10$ * $\texttt{S}_{t-1}$	0.098	0.072		0.009	0.093	
Constant	2.281	1.827		4.916	4.232	

Note: Specifications also include controls for age, education, ancestry, origin, cohort, and year trends. Standard errors are in parentheses.
*** indicates joint significance at the 1% level; ** 5% level; * 10% level.

Table 8 presents estimates of and standard errors on the coefficients of the observed determinants of death by the end of period t conditional on being alive in period t. We also examine cause of death conditional on dying in Appendix Table A4. Note that the specification reflects updated values of the endogenous variables (i.e., includes the period t behavior and health events). As with the previous table where we cannot draw firm conclusions simply by examining the coefficients, we nonetheless can discuss some interesting findings. First, while it may appear that current smoking reduces the probability of death, one should note that each year of smoking duration significantly increases the probability of death and current period smokers are likely to have a long history of smoking. Second, disease is an important predictor of death, as expected. Cardiovascular disease events and cancer diagnosis in the current period increase the probability of death by the end of the period. While such events in the previous period specifically do not have a statistically significant effect, having ever had these diseases increases the death hazard. Higher levels of current health markers, such as body mass, diastolic blood pressure, and cholesterol, predict eminent death. Third, it appears that years of smoking cessation has no statistically significant effect on the probability of dying, but this interpretation ignores the indirect channels embedded in the entire system of equations. Continued smoking (i.e., a positive number of years of smoking duration) significantly increases the probability of both morbidity and mortality. Thus, smoking cessation (which sets duration to zero) eliminates an important detrimental impact. These dynamic effects will be clearer when we simulate behavior of the individuals under different lifetime smoking patterns.

Differences in the coefficient signs and significance across the models with and without UH are more apparent in the cause of death equation. Current smoking significantly predicts death due to cancer, conditional on dying, and has a large but imprecise impact on death due to cardiovascular disease. Diagnosis of cancer in the current period or the previous period does explain cancer deaths before the next period and cardiovascular events in the current period explain CVD deaths.

Appendix Tables A5, A6, and A7 present estimates of and standard errors on the coefficients of the observed determinants of endogenous disease events in period t: the number of cardiovascular events; cancer diagnosis conditional on no diagnosis prior to the current period; diabetes diagnosis conditional on no diagnosis prior to the current period; and the continuous health marker body mass index. These per-period disease events define health history variables that explain the dynamic smoking patterns of individuals over a lifetime. Because we allow for correlation between these endogenous events and smoking behavior and mortality outcomes, we reduce bias in the estimated marginal impacts of interest in our study. The results presented in the tables indicate

Table 8: Selected Parameter Estimates: Mortality by end of period

	Without UH			With UH		
Variable	Estimate	Std. error		Estimate	Std. error	
Smoker in t, $\mathtt{S}_t$	-0.624	0.153	***	-1.428	0.506	***
Years of cessation, $\mathtt{C}_t$	0.009	0.010		0.004	0.012	
$\mathtt{C}_t^2/100$	-0.018	0.024		-0.012	0.026	
Years of duration, $\mathtt{D}_t$	0.074	0.013	***	0.101	0.023	***
$\mathtt{D}_t^2/100$	-0.102	0.023	***	-0.132	0.033	***
Years of experience, $\mathtt{E}_t$	-0.014	0.009		-0.008	0.011	
$\mathtt{E}_t^2/100$	0.031	0.018	*	0.026	0.021	
$\mathtt{1[CVD}_t = 1\mathtt{]}$	0.476	0.112	***	0.700	0.273	**
$\mathtt{1[CVD}_t > 1\mathtt{]}$	0.692	0.193	***	0.745	0.278	***
$\mathtt{CAN}_t$	0.874	0.175	***	1.224	0.349	***
$\mathtt{DIA}_t$	0.413	0.275		0.688	0.435	
$\mathtt{1[CVD}_{t-1} = 1\mathtt{]}$	0.021	0.130		0.019	0.139	
$\mathtt{1[CVD}_{t-1} > 1\mathtt{]}$	0.460	0.217	**	0.437	0.242	*
$\mathtt{CAN}_{t-1}$	0.288	0.219		0.342	0.254	
$\mathtt{DIA}_{t-1}$	0.095	0.268		0.099	0.277	
$\mathtt{E\_CVD}_{t-1}$	0.396	0.107	***	0.377	0.116	***
$\mathtt{N\_CVD}_{t-1}$	0.151	0.046	***	0.162	0.047	***
$\mathtt{E\_CAN}_{t-1}$	0.604	0.122	***	0.667	0.144	***
$\mathtt{E\_DIA}_{t-1}$	0.555	0.111	***	0.445	0.123	***
$\mathtt{BMI}_t$	-0.276	0.079	***	-0.350	0.097	***
$\mathtt{BMI}_t^2/100$	0.432	0.143	***	0.497	0.172	***
$\mathtt{SBP}_t$	0.015	0.012		0.015	0.013	
$\mathtt{SBP}_t^2/100$	-0.005	0.004		-0.005	0.005	
$\mathtt{DBP}_t$	-0.053	0.022	**	-0.052	0.023	**
$\mathtt{DBP}_t^2/100$	0.040	0.013	***	0.040	0.014	***
$\mathtt{CHO}_t$	-0.021	0.005	***	-0.019	0.007	***
$\mathtt{CHO}_t^2/100$	0.004	0.001	***	0.004	0.001	***
BMI, SBP, DBP missing	-4.770	1.136	***	-6.164	1.442	***
CHO missing	-0.921	0.579		-0.708	0.740	
$\mathtt{ART}_t$	-0.035	0.070		-0.049	0.074	
Constant	-1.557	1.740		1.285	2.288	

Note: Specifications also include controls for age, education, ancestry, origin, cohort, and year trends. Standard errors are in parentheses.
*** indicates joint significance at the 1% level; ** 5% level; * 10% level.

that the parameters differ in sign as well as economic and statistical significance between the specifications. Marginal impacts are described below using simulations from the estimated model.

5.2 Ability of Empirical Model to Fit the Observed Data

Given the many features of our dynamic model, and the associated inability to fully comprehend a variable's impact by looking only at coefficients, we simulate smoking behavior and health outcomes using the estimated model. To conduct our simulations, we replicate the exogenous variables and initial conditions of each individual in the estimation sample R=50 times. For each replication we simulate smoking behavior and health outcomes (morbidity and mortality) for one period. That is, we use the estimated model and draws from the estimated joint UH error distribution and the i.i.d. error distributions to simulate the endogenous outcomes. We then update the smoking and disease histories and simulate behavior and outcomes in the subsequent period for those who are not simulated to die. Analogously, we simulate outcomes until everyone in the simulated sample has died.[33] Note that our simulations use the parts of the model that are explained by observed heterogeneity (i.e., the estimated coefficients and observed exogenous and endogenous variables as well as unobserved heterogeneity captured by the estimated permanent UH, the estimated time-varying UH, and the random error draw for each outcome each period. Predictions often only capture that explained by the model. All three sources of UH play an important role is our simulations because they determine current behavior and outcomes that impact subsequent behavior and outcomes through our correlated system of dynamic equations. Thus, the fit we capture is one that demonstrates the dynamic, comprehensive ability of our system of equations to explain lifecycle observations.

The top panel of Table 9 displays the distribution of age and cause of death for the observed sample (used in estimation of the model) and the simulated sample (generated from the estimated model). Note that we report averages in this panel using only those replicated individuals whose counterpart is alive in the observed data in order to show that our model fits the observed data. Our preferred model captures the age and cause of death distributions quite well, and correctly simulates that 88 percent of the sample has died by the end of 1996 (or 22 periods of the model).

In the lower panel of Table 9, we evaluate our model's ability to predict age of death

[33] In cases where we simulate someone to survive who, in the data, is observed to die, we must impute values of his exogenous variables. Age is increased by two years every period that the replicated individual is alive. The health markers (i.e., systolic and diastolic blood pressure and cholesterol levels) are imputed based on an individual's last observed values and averages among individuals his age.

Table 9: Age and Cause of Death: Observed and Simulated Data

Sample	Percent died	Age of death distribution (percentile) Mean	10th	25th	50th	75th	90th	Cause of death CVD	Cancer	Other
Deaths observed through 1996 (right-censored; estimation sample)										
Observed	87.8	72.6	58	66	74	80	85	42.6	26.3	31.6
Simulated	88.8	72.0	58	65	73	79	85	43.6	25.8	30.6
Deaths observed through 2009										
Observed	100.0	74.4	59	68	76	82	88			
Simulated	100.0	74.2	59	67	75	82	88			

Note: Observations are right-censored if the individual (observed or simulated) has not died through 1996 (two years after exam 23).

outside of the sample used in estimation of the model. When we began this project, we were granted access to the FHS data from NHLBI through 1996. However, the original cohort of the FHS continued to be followed. We recently acquired age of death (but not cause of death) for the original sample through 2009. Using our estimated model to simulate smoking behavior and health outcomes until death for the replicated sample, we can determine how well our model captures the true observed age of death of individuals used in estimation who had not died through 1996. By 2009, everyone in our estimation sample had died. The average age of death was 74.4 years. When we use our model of lifetime smoking, morbidity, and mortality to simulate the sample until death, we find a simulated average age of death of 74.2. This ability of our model to match the observed out-of-sample death ages gives us additional confidence that the model explains lifetime smoking and health very well.

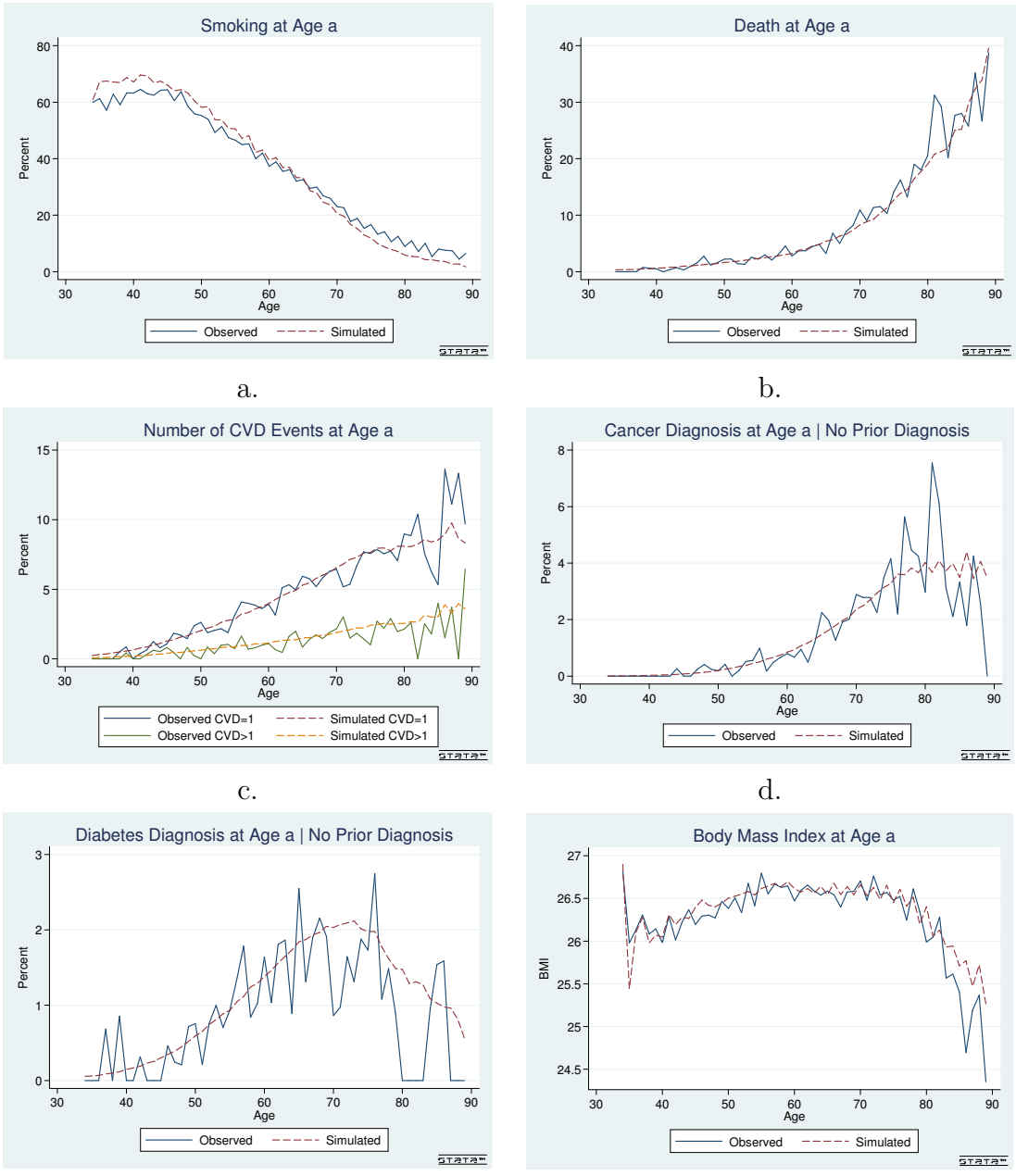
Comparisons of the simulated data to the observed data provide measures of how well our model captures the dynamic behaviors and outcomes of interest. Up to this point, we have described the FHS data over time, with calendar year or period (two years) being the unit of observation per person. However, the main purpose of the empirical model is to explain smoking behavior and health outcomes over an individual's lifetime (while controlling for aggregate variation that affects all individuals over time). From this point forward we discuss the model in terms of its ability to fit lifetime profiles of smoking, morbidity, and mortality by age.

First, we graphically compare the age-specific outcomes of the simulated sample, for those person years when the replicated individual is observed to be alive, with the outcomes of the estimation sample. Figure 2 presents the model fit for each of the

dependent variables (excluding cause of death) by age.[34] We slightly overstate smoking behavior at younger ages and understate it at older ages, but generally capture the overall decline in smoking by age quite well. Our mortality model fits very well even into ages above 70 when observed death rates are less precise due to small sample sizes. The model also accurately predicts, by age, small probability events such as one or more CVD events, cancer diagnosis, and diabetes diagnosis. We also fit body mass as measured by BMI very well. Additionally, chi-squared tests indicate that we cannot reject that the averages of the simulated data from our data generating process are equivalent to those of the observed data.

[34]We do not describe the fit of the five reduced-form (not dynamic) initial condition equations; this information is available from the authors. The initial conditions, which are correlated with the permanent individual UH, are estimated jointly with the per-period equations in order to aid in identification of the UH distributions. When we create the simulated sample, we use the observed initial conditions as our starting point. Appendix Table A3 provides coefficient estimates for the initial condition equations.

Figure 2: Model Fit of Smoking, Morbidity, and Mortality Outcomes

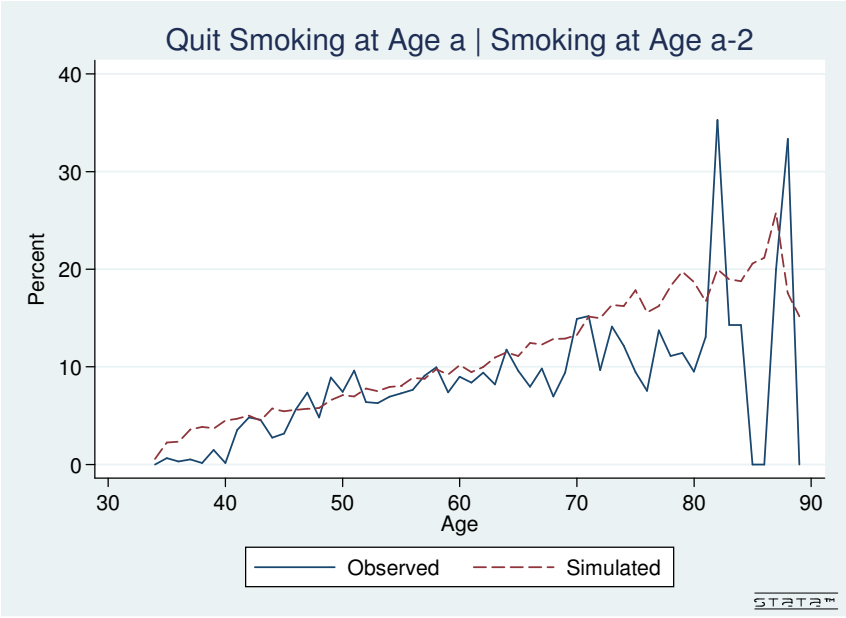


Note: Solid lines indicate averages of observed sample behavior and outcomes. Dashed lines indicate predicted probabilities from simulation of our preferred model with unobserved heterogeneity. Figures depict rates or levels of: a.) smoking, b.) mortality, c.) cardiovascular disease, d.) cancer, e.) diabetes, and f.) body mass.

Second, we use the simulated data to assess the ability of the model to capture

important smoking transitions such as quitting and relapse, rather than simply levels of smoking. Figure 3 displays the probability of quitting smoking at each age conditional on smoking in the previous period (two years earlier on average). Again, our model does an exceptional job of capturing the quitting trend by age.[35] The probability of relapse is more difficult to graph, yet average simulated relapse rates of 44.2 percent among men who quit smoking is not statistically different from the observed relapse rate of 37.5 percent.[36] Among those who relapse, the observed and simulated mean length of smoking cessation between spells of smoking is 3.3 and 4.2 years, respectively; the mean age of relapse is 53.9 and 54.4 years.

Figure 3: Model Fit of Quit Behavior conditional on Smoking in Previous Period



Note: Solid line indicates average of observed sample behavior. Dashed line indicates average of simulated behavior from our preferred model with UH.

Appendix B shows the empirical importance of modeling individual unobserved heterogeneity. In the model UH captures unobservables, both those that are common across a lifespan (like genetics) and ones which vary over time (such as unobserved stress), which would bias the estimated impacts of smoking and health histories. We show there are important differences in smoking behavior, mortality distribution, and the link between smoking and death across the various permanent UH types. In addition to providing a better fit through reduced selection and endogeneity, these results

[35]Note that behavior at the youngest and oldest ages reflects small sample sizes for these groups in our data.

[36]In this calculation, we condition on quits observed after age 30.

highlight the importance of distributional issues which can factor into the choice of policy variables such as cigarette taxes or regulations.

5.3 Simulated Lifetime Smoking Scenarios

Having demonstrated the ability of our estimated model to capture observed behavior and outcomes accurately and to predict mortality well out-of-sample, we now turn to assessment of the impact of smoking on morbidity and mortality. To do so, we conduct several simulations in which we impose different lifetime smoking patterns. For this analysis, we simulate smoking behavior and health outcomes until death. That is, there is no right censoring of the data; every simulated individual is observed until death.[37]

Row 1 of Table 10 presents the age and cause of death distributions when all individuals in the sample (i.e., a sample composed of R (=50) replications of exogenous variables of the N (=1464) sample observations) are simulated to never smoke. Average age of death is 75.6 years. Row 2 describes the mortality outcomes of the sample when everyone is assumed to smoke from age 18 through death (which is determined by the model based on sequential updates of morbidity throughout the lifecycle).[38] Age of death is, on average, 71.3, or 4.3 years earlier than a non-smoker. For comparison, the (biased) unconditional difference-in-means in age of death between lifelong smokers and nonsmokers is 9.3 years using the right-censored FHS estimation sample of males.[39] We contend that the observed and unobserved heterogeneity that we model reduces the bias in estimates of the impact of life cycle smoking behavior and morbidity on mortality. Additionally, CVD accounts for 16.6 percent (or (46.4 - 39.8 =) 6.6 percentage points) more deaths for smokers versus non-smokers, while death from cancer rises 40 percent (or (28.0-20.0=) 8.0 percentage points).

More importantly for policy purposes, we calculate the expected gain (in life years) of quitting smoking at particular ages. Relative to smoking continuously, quitting smoking at ages 60, 50, and 40 implies an increase in longevity of 0.4, 3.0, and 4.7

[37]Simulations are conducted until the year 2024 (or hypothetical exam 42). Recall that individuals are age 30-62 in 1952. Every replicated individual is simulated to die. In the baseline simulation (where we impose no smoking pattern but use the model to simulate smoking histories), only 1.3% of individuals are simulated to die after age 100. In all simulations in which someone is simulated to die after age 100, we assume death occurs at age 100 for summary calculations.

[38]For these simulations, initial smoking duration is set to initial age - 18. We also simulate the smoking scenarios in Table 10 with smoking initiation at age 13 rather than 18. The age of death distribution is shifted to the left slightly (i.e., younger). Results are available from the authors.

[39]Restricting our calculations to death before 1998 (i.e., the period when we observe both smoking and health in our data), the difference in the simulated death ages of never smokers and continual smokers is 3.8 years.

Table 10: Age and Cause of Death: Simulated Data by Smoking Scenario

		Age of death distribution (percentile)					Cause of death		
Smoking scenario	Mean	10th	25th	50th	75th	90th	CVD	Cancer	Other
Never smoked	75.6	61	69	77	83	89	39.8	20.0	40.2
Smoked continuously from age 18	71.3	57	64	72	79	85	46.4	28.0	25.6
Started smoking at 18 and quit at:									
Age 40	76.0	61	69	77	84	90	38.7	28.0	33.3
Age 50	74.3	56	67	76	83	89	39.5	25.4	35.1
Age 60	71.7	57	61	73	80	87	42.1	23.7	34.1
25 Years of smoking:									
Ages 13-48	74.4	56	67	76	83	89	39.3	25.9	34.9
Ages 23-58	72.9	57	63	74	82	88	41.9	23.5	34.6
Continuous smoking with a gap between:									
Ages 30-35	71.1	60	66	71	77	82	52.6	27.6	19.8
Ages 40-45	72.3	62	68	73	78	82	53.2	27.7	19.2
Ages 50-55	73.0	58	69	75	79	83	52.2	27.2	20.6

years, respectively. The heavily cited Doll, *et al.* work finds increases of 3, 6, and 9 years. Our findings suggest that these commonly-used figures are inflated by nearly 50 percent. Note that while quitting by age 40 produces a lifespan distribution that is almost identical to that of never smokers, the likelihood of death by cancer is still proportionately higher. Interestingly, quitting smoking lowers the probability of death by CVD relative to cancer or other causes, yet the history of smoking among former smokers still manifests itself in a higher probability of a cancer-related death. Related, Taylor *et al.* (2002) find that quitting smoking at age 35 extends life expectancy of males by 6.9 to 8.5 years relative to those who continue to smoke. They also find that quitting earlier is more beneficial than quitting later. While their empirical results are based on a larger sample of individuals than ours, it is not a nationally representative sample and only 20 percent of the sample had died during the study period. Our findings using their same specifications are similar to theirs. However, when we evaluate results using our preferred model with additional sources of heterogeneity and more observed deaths, our estimates of the benefits of quitting decrease significantly.

Also detailed in Table 10, we examine differences in age and cause of death for individuals with the same smoking experience but different ages of initiation (and hence also different ages of quitting, conditional on survival). We find that starting smoking

later in life (i.e., age 23 versus 13) leads to a lower life expectancy by 1.5 (=74.41 - 72.88) years. Yet, death by cancer is more likely when smoking is initiated earlier and death by CVD is more likely when smoking occurs at older ages.

Lastly, we examine the impact of smoking cessation followed by relapse. We simulate individuals to have a 5-year reprieve from smoking at the ages of 30-35, 40-45, or 50-55. In all simulations the individuals began smoking at age 18 and smoked until death (following the single 5-year cessation period). A small spell of cessation has no statistically significant difference on life expectancy (from that of continuous smokers) if it occurs at younger ages. If the 5-year cessation occurs later, there is a slight increase in average ages of death. Death attributable to cancer or CVD receives similar weights, relative to other causes, regardless of the age of cessation. Interestingly, CVD deaths are 13 percent more prevalent (about 6 percentage points) for individuals with a 5-year gap in smoking than those who have smoked continuously since age 18.

6 Discussion

Based on our findings, we concur with the universal evidence in the medical and economics literatures that smoking is detrimental to health measured by both morbidity and mortality. Our results suggest, however, that the mortality consequences of smoking typically cited and used by policymakers are overstated by as much as 50 percent (i.e., a difference in age of death of 4.3 years on average versus the existing evidence of 9.3 years). As an example of why accurate estimates are important, consider the U.S. Food and Drug Administration's (FDA) current evaluation of the costs and benefits of smoking cessation in order to determine the appropriateness of a particular regulatory action that will impact smoking behaviors (Chaloupka *et al.*, 2014). The economic analysis has received much attention due to the suggestion that the benefits be discounted to reflect a smoker's lost happiness that would accompany smoking reductions. Less attention has been paid to the calculations of the morbidity and mortality consequences of reduced smoking. Irrespective of the discounting issue, the "inflated" figures currently being used to evaluate policy and regulatory decisions could lead to costly implementation with significantly reduced impacts.

Our results reiterate the importance of quitting at younger ages, with improvements in life expectancy of, for example, 4.7 years versus 3.0 years if the cessation occurs at age 40 versus 50. We demonstrate new findings showing the importance of relapse avoidance; short spells of non-smoking followed by relapse has very little benefit. In addition to policies that encourage quitting, emphasis should be placed on quit maintenance. Cessation programs without follow-up support for former smokers will not be

effective in extending life if relapse occurs. Our model also demonstrates that rates of death attributed to CVD and cancer differ by lifetime smoking patterns. By applying our findings and accurate pecuniary costs and measures of pain and suffering by disease, more comprehensive cost-effectiveness analysis can be used to evaluate smoking policy recommendations expected to result in different smoking behaviors.

Admittedly, the nature of smoking has changed profoundly for recent generations compared to that of the FHS original cohort. Changes include the age of initiation, the modes of smoking, the use of filters (introduced in 1950s), the levels of tar, etc. Some of these changes are technological and might change underlying estimated parameters; others are behavioral and should support our findings. The additional cohorts of the FHS (e.g., offspring and third generation) make it possible for us to follow up our paper using the same techniques with a more recent cohort (Darden, 2012). We emphasize the importance of continued and new data acquisitions, like the FHS, that follow individuals at frequent intervals over a long period of time.

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