Parental Health as Foreshadowing? The Effect of Parental Health Shocks on the Smoking Behavior of Adult Offspring

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Abstract

A continued policy focus on smoking cessation incentives is a stronghold in U.S. efforts to encourage healthy lifelong habits. In order to understand initiation and habituation of risky behaviors, economic researchers have studied financial disincentives, smoking bans and restrictions, peer reinforcement, and health information campaigns. We examine the role of health expectations and health signals. In particular, if adult smokers view parental health shocks – smoking-related or otherwise – as foreshadowing of their own health outcomes, then there may be room for anti-smoking policy advocates to capitalize on and emphasize this relationship. In this paper, we merge the Original Cohort and the Offspring Cohort of the Framingham Heart Study (FHS) to study how adult offspring smoking behavior and subjective health expectation vary with elder parent smoking behavior and health outcomes. Our data allow us to model the smoking behavior of adult offspring over a 30-year period contemporaneously with parental behaviors and outcomes. We find that women in our sample are significantly more likely to smoke when their mothers smoke. While adult offspring smoking behavior is not found to be responsive to parent cardiovascular shocks or cancer diagnoses, the subjective health assessment of current smokers declines dramatically after a health shock to a parent who smokes. This finding suggests that smokers internalize the health effects of cigarette smoking, but do not quit smoking as a result. We also find adult offspring are much less likely to smoke and more likely to report worse subjective health when they themselves experience a cardiovascular shock or cancer diagnosis.

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Lochner (2008) defines intergenerational transmission as the transfer of individual abilities, traits, behaviors, and outcomes from parents to their children. We recognize that a measured transmission effect may reflect correlation in behavior due to non-causal or causal influence of parents. The abilities, endowments, or preferences of offspring, which determine their observed behaviors, may be genetically related to those of their parents and, hence, exogenously correlated with parents' behavior. Alternatively, these abilities, endowments, or preferences of offspring that explain their actions may be shaped by parents through related actions and financial and non-financial investment. Such causal mechanisms suggest a role for policy incentives that encourage positive parental behaviors.[1]

To motivate our own research, we want to emphasize the temporal, or distance (measured in time), characteristics of intergenerational transmission. While some parental behaviors shape the eventual outcomes of offspring when these offspring are young, it is possible that parental behaviors may influence adult offspring behaviors contemporaneously (or close in time). In other words, an observed correlation in behaviors may be explained by parental influence that was experienced when the child was young or explained by parental influence as an adult.

A long literature exists on the question of how smokers formulate their expectations and, to the extent that changes in expectations may influence smokers to quit, which policies that may shift expectations. Viscusi (1990) and Viscusi and Hakes (2008) find that smokers overstate the risk of lung cancer, and the overstated risk acts as a tax on cigarette smoking. Other papers define discrete changes in health (e.g., a heart attack) as informational events from which a smoker may learn about the health consequences of smoking. Smith *et al.* (2001) find that smokers update their subjective probability assessments of living to age 75 more harshly than do nonsmokers and former smokers in the event of a significant health shock. Sloan *et al.* (2003) and Khwaja *et al.* (2006) find similar results,

[1]More broadly, a large literature exists on the intergenerational transmission of economic preferences, risk attitudes, and economic outcomes. Examples of behaviors or outcomes that have been studied by economists in an intergenerational context include educational attainment, permanent income (Solon, 1992; Bjorkland and Jantti, 2009; Lefren *et al.*, 2012), savings patterns (Knowles and Postlewaite, 2005), welfare participation (Corcoran *et al.*, 1988), fertility (Booth and Kee, 2006), volunteerism (Mustillo *et al.*, 2004), and charitable giving (Wilhelm *et al.*, 2008). The correlations in these behaviors are as high as 0.40 in some cases.

but also find that an individual's smoking behavior is not influenced by health shocks to a spouse.[2] Those authors conclude that heavy smokers, perhaps with an "it won't happen to me" attitude, require personalized information – often in the form of own-health shocks – to induce smoking cessation. However, for a health shock to induce a heavy smoker to quit, the literature has found that the shock often must be extreme. Indeed, Darden (2010) finds that an individual's smoking behavior does not respond to cardiovascular biomarker changes (e.g., cholesterol), but does respond to larger cardiovascular shocks and cancer diagnoses. Randomized controlled trials have generally found little evidence that biomarker information significantly encourages smoking cessation.[3]

The extent to which smoking behavior is sensitive to the health shocks of parents – smoking-related or otherwise – has received comparatively little attention. Given the genetic link between parents and offspring, if smokers view the health outcomes of their parents as foreshadowing of their own health, then there may be a role for policy to emphasize this relationship. There is considerable evidence that children of smokers are more likely to become smokers themselves. Many health economists have studied smoking initiation among teens as a function of parental smoking behavior (Green *et al.*, 1991; Jackson and Henriksen, 1997; de Vries *et al.*, 2003; Loureiro *et al.*, 2006; Bantle and Haisken-DeNew, 2002; Gohlmann *et al.*, 2010; Melchior *et al.*, 2010). However, the long-term health implications of cigarette smoking are not realized until individuals reach their 50s and 60s (Doll *et al.*, 1994, 2004; Darden, 2010); therefore, teen smoking behavior is unlikely to be influenced by the health of parents. To our knowledge, no paper has studied the extent to which adult offspring smoking behavior is sensitive to parental smoking and/or health outcomes.

[2] Christakis and Fowler (2008) shows that smoking behavior is influenced by the smoking behavior of those within the same social network. Those authors also show that the social networks of smokers are increasingly *defined* by smoking.

[3] A notable exception is Parkes *et al.* (2008), who find that informing smokers of their "lung-age" - the age of a health individual that would perform similarly on lung function tests - does significantly encourage smoking cessation. See McClure (2001), Bize *et al.* (2009), and Lancaster and Stead (2004) for reviews of the epidemiological literature.

In this paper, we exploit a novel intergenerational dataset to address several questions that relate to the intergenerational transmission of smoking behavior and health. First, using reduced-form, family specific fixed-effects models, we examine the empirical relationship between adult offspring smoking and subjective health assessment and a.) lifetime and contemporaneous parent smoking and b.) parent health. We examine these questions along gender lines to assess the potential for "like father, like son" effects. Next, we formulate and estimate a dynamic stochastic model of adult offspring smoking behavior that makes explicit the assumptions and mechanisms associated with changing offspring smoking behavior and expectations in the face of parental health shocks. Under the structure of the model, we separately identify the effect of a parental health shock on an offspring's utility of different smoking alternatives (e.g., depression, anxiety, altruism, etc.) from a shift in offspring expectations of own health transitions associated with different smoking decisions.

We construct a novel panel dataset of parents and adult offspring from the Framingham Heart Study (FHS), and we use these data to estimate both our reduced-form and structural model. We are the first social scientists of which we are aware to merge the FHS Original Cohort – begun in 1948 and followed to the present – with the FHS Offspring cohort – begun in 1971 and followed to the present. In our final estimation sample, we observe 2,075 adult offspring through seven detailed health exams/interviews over a 30-year period, and we model their smoking and subjective health expectations as a function of their Original Cohort parents' contemporaneous health exam/interview results.

From 1970 to 2001, the percentage of adults in the United States who smoke cigarettes declined from 37.4% to 22.8%.[4] Christakis and Fowler (2008) use Framingham Heart Study data to study declining smoking rates in the context of social networks of friends, colleagues, and family. While a typical social network in 1971 included both smokers and nonsmokers, those authors show that by 2001, smokers became increasingly isolated in

[4] *http://www.cdc.gov/tobacco/data_statistics/tables/trends/cig_smoking/index.htm*.

specific social networks largely defined by cigarette smoking. Thus, while overall smoking rates have declined, current smokers may face increasingly strong social peer effects, in addition to the biological addiction to nicotine. Furthermore, recent evidence from Chaloupka *et al.* (2012) and Callison and Kaestner (2012) shows that, while cigarette taxes have played an important role in the observed decline of smoking, middle-aged and heavy smokers are much less price sensitive. In this paper, we test the hypothesis that, through a foreshadowing effect, strong, personalized information in the form of a parental health shock, may encourage middle-aged smokers to quit.

Our reduced-form results show several interesting trends emerging from the data. First, fixed effects regressions suggest that women are 22% more likely to have ever smoked in 1971 if their mother has ever smoked through 1971. We find negligible effects of father's ever smoking – both on men and women – because virtually all fathers had some smoking history in 1971. When we study the effect of parental smoking on contemporaneous adult offspring smoking, again we find that women are responsive to their mother's behavior; a mother who continued smoking in 1971 implies a 46% increase in the probability of contemporaneous female smoking. These results are consistent with smoking initiation research that finds a "like mother, like daughter" relationship in which current mother smoking, rather than ever smoking, is most important (Gohlmann *et al.*, 2010). We find no significant change in adult offspring smoking when a parent experiences either a smoking-related or general cardiovascular or cancer diagnosis; however, we find that *current* adult smokers are much more likely to report worse health when a father experiences a smoking-related cardiovascular shock, and we find that adults who have ever smoked are much more likely to report worse health when a father experiences a smoking-related cancer diagnosis. These results provide suggestive evidence that individuals are aware of smoking risks, but addiction – or some other rationale – prevents smokers from quitting, even when the health ramifications are quite tangible. Finally, we find that adult offspring *both* smoke less and report worse health when they themselves have a cardiovascular shock or cancer diagnosis.

We have yet to estimate our preferred structural model, but this work is ongoing.

This paper proceeds as follows. Section I describes mechanisms for the intergenerational transmission of behavior and the literature on responses to health information. Section II describes the Framingham Heart Study. Section III presents our empirical model and our main results. Section **??** concludes.

I Background

In our effort to understand intergenerational transmission, we discuss several mechanisms of transmission that may capture non-causal or causal influence of parents. Because we apply our investigation to smoking behavior across generations, our examples relate to smoking and health. Specifically, we want to understand an observed correlation between mother's smoking and adult offspring smoking of 0.14 found in the Framingham Heart Survey cohorts.[5]

I.1 Genetic Transmission

A potential explanation for this correlation is the genetic transmission of abilities and traits that influence individual behaviors. One determinant of smoking behavior that may be genetically passed from one individual to another is health (Thompson, 2012). For example, the tendency to develop asthma is an inheritable trait. Individuals with asthma may be less likely to smoke. Hence, the observed correlation between (non) smoking behavior across generations may be partially explained by the similar health of a parent and child. Additionally, genetic similarities may include an individual's health response to cigarette consumption. That is, the marginal effects of smoking on physical responses (e.g., blood pressure) that predict chronic health conditions (e.g., coronary heart disease and stroke) may vary across individuals (Darden, 2010). Risk and time preferences are additional determinants of smoking behavior that may be genetically inherited and, hence, are a possible

[5]This raw correlation between maternal smoking and offspring adult smoking varies with whether or not the mother (and father) is still alive; correlations between paternal smoking and offspring adult smoking are similarly divergent depending on the survival state of both parents.

explanation for an observed correlation in parent and offspring behaviors (whether separated by time or contemporaneous). An individual's degree of risk aversion significantly predicts her engagement in risky activities. Rates of time preferences significantly influence adoption of behaviors.

Alternatively, or additionally, it may be the case that preferences and attitudes of offspring are taught or shaped by parents. This mechanism of intergenerational correlation includes the direct attempts by parents to model preferences (Bhatt and Ogaki, 2012) or the less direct cultural transmission of beliefs or the power of example (Arrondel, 2009; Dohmen *et al.*, 2012; Paola, 2012).

I.2 Transmission in the Early Years: Consumption Externality or Role-Model Effect.

Moving beyond the genetic transmission mechanism and the purposeful or non-purposeful molding of preferences mechanism, behaviors of parents while a child is young and in the household may explain observed correlation between adult behaviors of adjacent generations. Financial and non-financial educational inputs correlated with parent's educational attainment (e.g., reading material in the household and time spent reading to children) may explain a child's eventual level of education. Similarly, a child's physical access to a smoking parent's cigarettes or the exposure to second-hand smoke may promote smoking initiation (i.e., a parental consumption externality). Children may mimic the behavior of their parents (i.e., a role-model effect). Over 80 percent of adult smokers say they began smoking in adolescence.[6] Hence, a correlation in parental and offspring adult smoking behaviors may have as its root cause the smoking behavior of the parent when the offspring was young. Many health economists have studied smoking initiation among youth as a function of parental smoking behavior (Green *et al.*, 1991; Jackson and Henriksen, 1997; de Vries *et al.*, 2003; Loureiro *et al.*, 2006; Bantle and Haisken-DeNew, 2002; Gohlmann *et al.*, 2010; Melchior *et al.*, 2010).

[6] CDC Fact Sheet, 2012

I.3 Transmission in the later years: information or altruism.

While these mechanisms, transmitted at birth or at a young age, may explain observed correlation between adult behaviors across generations, it is important to distinguish them from additional mechanisms that explain contemporaneous correlation between parent and offspring behaviors. That is, do the actions and outcomes of parents today affect the behaviors of their adult offspring today or in the near future? In particular, we explore the role of parental health on the smoking behavior of adult offspring. Realized parental health can be a form of information transmission. An adult offspring may update her own health expectations, or the marginal effects of one's own smoking, when she observes a (smoking) parent's health experiences. The parental (smoking-related) health outcomes that have the potential to influence behavior across generations range from elevated health markers (e.g., cholesterol, blood pressure, body mass) to onset of chronic disease (e.g., heart disease, stroke, cancer, pulmonary problems) to death. Darden (2010) investigates whether individuals respond to their own health events as well as measurements of health markers that predict negative health events. His model of lifetime smoking decision making allows for learning through one's own smoking experience and observation of health markers over time. Smith *et al.* (2001) and Khwaja *et al.* (2006) examine the effects of own health shocks on own subjective longevity expectations. The effect of spousal health shocks on own subjective longevity expectations is also considered (Khwaja *et al.*, 2006). These researchers conclude that individuals do update their health expectations, and that heavy smokers update more than former or non-smokers after health information is received. However, that information needs to be individual specific; at least in the case of heavy smokers, there is little evidence of general health warnings or the health events of their spouses causing smoking cessation.

An observed response among smoking individuals to a spousal or parental health shock, or quit attempts/success, may indicate information transmission, but it may also be explained by the consumption externality mechanism described above or be evidence of

an altruistic mechanism. The latter suggests that an individual adjusts his own smoking behavior because he cares about another individual's health or happiness.

In light of the many potential mechanisms for intergenerational transmission of abilities, traits, behaviors, and outcomes, it is necessary, for policy recommendation purposes, to be able to attribute an observed correlation in parental and offspring adult behaviors to (or to rule out) the different mechanisms. A variety of empirical techniques have been used in the economics literature to disentangle potential mechanisms. Researchers have studied the behaviors of siblings, twins, and adopted children in order to account for or rule out genetic transmission using family fixed effects. They have used instrumental techniques to model endogenous adult behaviors. They have allowed for updating of own subjective health expectations and learning about health transitions based on own experience. Our research on the effect of parental health and smoking behavior on offspring adult smoking behavior and own health assessment addresses these many mechanisms by examining a unique dataset that follows both parents and their offspring over 30 years. We begin, in this paper, by describing the data along these dimensions. Subsequent work will explicitly model dynamic smoking decision-making over a lifetime and allow for parental influence through observed (simultaneous or recent) health outcomes and smoking behavior.

II The Framingham Heart Study

The Framingham Heart Study (FHS) is one of the longest running epidemiological panel studies in the world. The Original Cohort of FHS began in 1948 and consisted of 5,079 individuals aged 30 to 60 in Framingham, Massachusetts. Participants have undergone a cardiovascular health exam and interview at roughly two-year intervals, and we have access to 26 waves of Original Cohort data from 1948 through 2001. In 1971, FHS began conducting health examinations on the offspring and offspring spouses of the original FHS cohort. Offspring and their spouses have participated in health exams/interviews at roughly five-year intervals, and we have access to seven waves of offspring data from 1971 through 2001. For both cohorts, information is available on smoking behavior and a

variety of health outcomes. Importantly, FHS has just begun allowing researchers to merge the two cohorts.

We study the Offspring Cohort as our base sample to which we merge the Original Cohort. Table 1 describes the construction of our final sample. The FHS Offspring Cohort began with 5,124 individuals, 4,989 of which consented for their health exam results to be released. We drop individuals who either missed an exam or who attrit from the sample.[7] The Framingham Heart Study is not a representative sample of the United States population, and we consider the gains from internal validity to outweigh the loss in external validity. After dropping, we are left with 3,012 offspring cohort participants.

TABLE 1

Because we model the behavior of the offspring cohort, we consider all 5,079 Original Cohort participants (2,294 men and 2,785 women) for which we have information. In constructing our final sample, we keep only those offspring who have at least one parent record, reducing our final sample to 2,075 offspring.[8] For the merged parents, we keep information on smoking behavior and the specific years of cardiovascular shocks,[9] cancer diagnoses,[10] and death. 1,381 of the 2,075 offspring have records for both parents; for those with one parent missing, we create a missing parent indicator rather than drop this person from the analysis.

Table 2 presents summary statistics of our research sample at the first offspring cohort FHS exam. There exists variation in the year of the first exam across the Offspring Cohort, but all participants completed the exam between 1971 and 1975.[11] Age at the first offspring

[7]Given that the time gap between health exams is, on average, five years, we prefer to drop those that missed an exam rather than impute smoking behavior.

[8]An Offspring participant may not have an Original Cohort parent if the participant is the spouse or if the parent did not consent to the release of his or her exam/interview results.

[9]These include coronary heart disease, myocardial infarction, angina pectoris, coronary insufficiency, stroke, intermittant claudication, and congestive heart failure.

[10]We have information on the specific site of the cancer diagnosis, but for sample size reasons, we aggregate these to a simple binary cancer indicator.

[11]FHS does not disclose the date of an offspring individual's initial exam. The timing of subsequent health exams and health events is given in days since the first exam. We are able to approximate the year of first exam, and we impute the year of subsequent events with the days information. See Darden (2010).

exam ranges from 13 to 62 years, with an average age of 35 for men and 36 for women. While slightly more women than men in our sample smoke at the first exam (41% vs. 40%), male smokers smoke on average 5.5 cigarettes more per day. Men are also more likely than women to have ever smoked in their lifetime. 15% of mothers and 30% of fathers with a record in the FHS Original Cohort had died before the first offspring exam. Of offspring participants with a matched father, 87% of fathers smoked at some point, and 40% of mothers and 70% of fathers were smoking in the five years prior to the first offspring exam. At the first exam, 26% (20%) of fathers (mothers) had previously had a cardiovascular event and 5% (5%) had a history of cancer.

TABLE 2

Subsequent offspring exams occurred at roughly five-year intervals. Table 3 provides summary statistics for exams two through seven. Given that we have eliminated attrition, Offspring Cohort participants only leave our sample through death. Indeed, of the 2,075 participants, 290 are observed to die prior to their seventh exam. We code a health shock at a particular exam if it occurred prior to the exam but after the previous exam. In our sample, cardiovascular events occur, on average, for 3.4% of men and 1.5% of women. After an event, a time-varying state variable turns to one indicating that a person has experienced a shock. On average, 7.1% of men and 3.6% of women have previously had a cardiovascular event. We code parental health shocks similarly. For example, approximately 10% of fathers that are nonmisisng and that remain alive are diagnosed with cancer per exam for offspring exams two through seven.[12]

TABLE 3

Smoking prevalence over exams two through seven declines from a high (at exam one) of 40% for men and 41% for women to 23.6% and 21.9%, respectively. Finally, FHS

[12]Recall that the Original Cohort participants ranged in age from 30 to 60 in 1948. By 2001, the end of our data for both cohorts, of the 5,079 initial Original Cohort participants, only 558 remain alive.

only asks for a subjective health assessment in exams five through seven, and women are less likely to report excellent health than are men (41.6% versus 44.8%).

III Reduced-Form Model and Results

We begin our empirical analysis by examining the relationship between whether the adult offspring reports having ever smoked (through the offspring's last exam, around 2001) and whether the "linked" parent ever smoked prior to the end of the offspring's first exam (around 1975).[13] Our linear probability model includes indicators of whether the parent observation is available, the parent's gender, and controls for offspring age, education, gender, and cohort.

In addition to identifying the parents of the offspring participants, we have a common family identifier that allows us to identify siblings within the FHS Offspring Cohort. In our linear probability models, we include family fixed effects to account for any permanent family unobservables that explain both parent and offspring behaviors. This model allows us to examine whether intergenerational correlation in smoking behavior exists, even after controlling for correlation transmitted through a genetic mechanism or common, unobserved, permanent family characteristics. A statistically significant effect of a parent ever smoking suggests a causal influence transmitted in the past (during the offsprings youth) or one that occurs contemporaneously. At this point, the model does not include current parental smoking behavior and health outcomes.

Table 4 presents results from our linear probability model with and without family fixed effects. Models with family fixed effects show a positive and significant relationship between mother's smoking and female offspring smoking. At the mean level of female offspring smoking, a women is 22% more likely to have ever smoked if her mother also

[13] At the first exam, we have retrospective smoking information, so we have data on whether the offspring participant has ever smoked through 2001 and the age of initiation.

smoked. Interestingly, the family fixed effects eliminate any significant relationship between mother's smoking and male offspring smoking, as well as any effect of father's smoking.

TABLE 4

We continue our exploration by modeling the smoking behavior of the adult offspring over exams two through seven. Because the matched parents of the offspring continue to be followed over the same time interval, the time-varying smoking behavior of the adult offspring is allowed to depend on whether the parents ever smoked as well as whether they continued smoking through the time of the first offspring exam (if the parent is still alive).[14] Again, we control for permanent family unobservables that may be correlated with both offspring and parent behaviors. Here, we can disentangle the causal correlation as resulting from past parental smoking or recent parental smoking.

Table 5 presents results of linear probability model estimates of offspring smoking in exams two through seven. Again, we find mother's behavior may influence a daughter's smoking behavior, but only significantly when the mother continues smoking into older age. Also, we find no significant effect of father's smoking on either male or female offspring.[15] Furthermore, although the estimate is not statistically significant, our fixed effect estimates suggest that men may in fact smoke slightly less if a parent ever smoked, regardless if he or she continued smoking.

TABLE 5

Economic theory suggests that individual expectations of own current and future health may impact smoking decisions. Exploring the data further, we introduce the time-varying health shocks to the parent (e.g., cardiovascular and cancer) as possible predictors

[14]We focus only on whether the parent was smoking in the first offspring exam to differentiate parents that ever smoked and quit versus those that continued smoking. In 1971, the parents that remain alive are on average 66 years of age.

[15]Recall that 87% of father's had ever smoked at the first offspring exam, and roughly 70% of fathers were smoking at the first offspring exam.

of contemporaneous adult offspring smoking behavior. We hypothesize that observations of parental health may influence perceptions of one's own health. While we do not know whether these parental health shocks are directly caused by the smoking behavior of the parent or not,[16] we interact them with the last measured smoking status of a parent prior to the health event.

Table 6 presents linear probability model estimates of offspring smoking in exams two through seven as a function of parent health. While we consider only the first instance of a cardiovascular shock or cancer diagnosis, we control for past shocks. We also control for whether the shock in question caused the parent to die. While almost none of our fixed effects estimates are statistically significant, we do find that men are statistically *more* likely to smoke when a father has a cardiovascular shock and was not smoking. Khwaja *et al.* (2006) finds a similar result at the death of a parent, a shock that those author's describe as a "stressor." We conclude from Table 6 that adult offspring in our sample are *no less likely* to smoke following a health shock to a parent.

TABLE 6

In order to further examine the mechanism of information transmission suggested by parental health shocks, we estimate linear regressions of the offspring's own subjective health assessment (three observations per offspring spanning exams 5-7) as a function of the same parental health shocks and contemporaneous parental smoking behavior in Table 6. The subjective health dependent variable takes three values corresponding to excellent, good, and fair/poor health. Higher values of this measure imply worse subjective health.[17]

Table 7 presents the results from subjective health regressions. We control for offspring age, education, gender, cohort, and own-health effects. Interestingly, offspring who have a father with a smoking-related cancer diagnosis are significantly more likely to report

[16] Cigarette smoking causes several forms of both cancer and cardiovascular disease (United States Department of Health and Human Services, 2004, 2010)

[17] We have estimated multinomial logit models of subjective health, but we prefer regression models when including fixed effects.

worse subjective health. Indeed, our fixed effects estimates suggest that a smoking-related cancer diagnosis to a father implies a two-thirds standard deviation reduction in offspring's self-reported health.

We also indicate among our offspring sample of ever smokers those who are current smokers and those who smoked in the past but quit. Current smokers who observe a smoking-related cardiovascular shock to a father report significantly worse health. The magnitude of the reduction in self-reported health is large at more than twice a standard deviation. Furthermore, current and ever smokers who observe a smoking-related cancer diagnosis also report significantly worse health.

TABLE 7

As a check of completeness, we also explore whether the offspring's own time-varying shocks to health explain own smoking behavior. Here we estimate linear probability models and linear regressions of offspring smoking and subjective health with and without *individual* fixed effects controlling for offspring age, education, gender, and cohort effects. Tables 8 and 9 present results from these models.

TABLE 8 AND TABLE 9

As expected, and consistent with Smith *et al.* (2001); Sloan *et al.* (2003); Khwaja *et al.* (2006); Arcidiacono *et al.* (2007), we find that own cardiovascular shocks and cancer diagnoses significantly reduce the probability of smoking; however, the effect is larger for cardiovascular shocks. A cardiovascular shock implies a 31% (at the mean) reduction in the probability of smoking. Interestingly, while a cancer diagnosis is statistically associated with worse subjective health (i.e., the estimate of 0.134 is almost one-quarter of a standard deviation), the individual fixed effects dramatically reduce both the magnitude and significance of smoking and cardiovascular health shock effects on subjective health.

IV Smoking Behavior with Health Uncertainty

Our goal is to understand how the health of an elderly parent influences the smoking behavior of an adult offspring. To this end, we model an adult offspring's smoking behavior as a function of her smoking history, her own health, and the health of her mother and father.

The optimization problem of the offspring involves choosing an optimal smoking amount each period that maximizes expected, discounted lifetime utility. The tradeoff faced by the offspring – one that we argue requires more structure to identify – is between the current gratification of smoking and the future (potential) health consequences. Indeed, there are several sources of future uncertainty in the model, each of which requires the econometrician to make assumptions.

First, with regard to own smoking preferences, the offspring observes her current preferences, but does not know for certain what her preferences for each smoking alternative will be in the future. We capture preference uncertainty by making a specific distributional assumption about future preference shocks.

Second, the offspring observes her current health, but faces probabilities of future health outcomes. The standard approach, and the one that we take, is to assume that the offspring knows the technology of health production (i.e., to assume rational expectations).[18] That is, the offspring understands the marginal effects of her smoking history and individual characteristics that define the estimated health transition probabilities.[19] In this case, the offspring's accumulated history of own smoking behavior is modeled (i.e., it captures the decisions of the offspring) and therefore is endogenous and accounted for along every possible future path an offspring can take when evaluating the value of lifetime utility associated with a current smoking alternative.

[18] Alternatively, we could assume that the offspring "predicts" or "assumes" her health transition probabilities (i.e., has subjective expectations) and learns about her true probabilities of transition (case 2). The latter is modeled by Darden (2013). The focus on the role of parental health shocks in this paper render the modeling of learning quite difficult, as we explain below.

[19] Aguirregabiria and Mira (2010) note that in the absence of subjective expectation data, expectations cannot be separately identified from state transitions and conditional choice probabilities without further assumptions.

Third, an assumption must be made about what (and when) the offspring knows about her parents' health. While reduced-form empirical models can capture changes in smoking behavior for changes in parent health, these models assume either that offspring are completely unaware of the *potential* for parent health shocks (i.e., no forecasting) or that offspring have perfect foresight over parent health (i.e., including the lead of parent health on the right-hand side and assume that an offspring anticipates the future health shock of her parent today). Instead, within our dynamic optimization problem, we assume that an offspring forecasts the probability of different parental health shocks.

Fourth, given our third assumption, *how* an offspring forecasts her parents' health requires assumptions about the offspring's knowledge of parents' behavior. We can take two possible paths: assume that the offspring assigns the parents' current health to all future health states and solves her optimization problem accordingly or assume that the offspring knows the determinants of her parents' future health (namely, current health, current smoking behavior, and smoking history of the parents) and predicts future health transitions. The latter path requires, similar to this option facing us regarding knowledge of parents' health, an assumption about what the offspring knows of the parents' future smoking behavior. Again, we can assume she observes today's behavior and assumes it stays the same for the future, or we can have her make predictions about her parents' smoking behaviors. Forecasting future parent smoking boils down to jointly modeling the smoking decisions (i.e., the optimization problems) of both the offspring and the parents using a simultaneous game-theoretic approach. Instead, we assume that an offspring observes the smoking behavior of her parents in the current period, and she assumes that this behavior will continue.

In summary, we assume that the offspring forecasts her own future health and that of her parents. We assume that the offspring knows her parents' current smoking status and history and that she assumes it will persist. These assumptions prevent us from having to model the offspring's expectations of the parents' smoking behavior and therefore does not admit the behavior of the offspring to influence the parents' smoking behavior. That is,

the offspring takes the parents' health and smoking behavior (up to period t) as given and uses this information to predict her own and parents' future health transitions governed by known technologies.

IV.1 The Individual's Decisionmaking Problem

The discrete choice, dynamic model represents the optimization problem governing smoking behavior and health of an adult offspring. Individuals choose among a set of smoking alternatives each period indexed by t. Think of it as an annual decisionmaking problem for now. Let i index an offspring individual in family j at time t. The per period choice indicator of the adult offspring is $d_{ijt} = d$ where

$$d = \left\{ \begin{array}{ll} 0 & \text{Do not smoke} \\ 1 & \text{Smoke} \leq 1 \text{ Pack/day} \\ 2 & \text{Smoke} > 1 \text{ Pack/day} \end{array} \right\}$$

Characteristics of the offspring affect the utility of smoking each period as well as her health evolution. These characteristics include exogenous socio-demographics at time t, X_{ijt}, and a vector of her smoking history up to time t, A_{ijt}. This accumulated history includes the individual's total experience smoking (i.e., number of periods), her tenure smoking (i.e., duration of current smoking), and her tenure not smoking (i.e., duration of current smoking cessation). For the purpose of carefully explaining the role of parental health on offspring smoking, we do not go into detail here about the differential roles of the components of the smoking history vector A_{ijt}. Ultimately, our estimated model will incorporate the legitimate roles of this history that capture (and explain) habitual smoking behavior and quit behaviors. (cite rational addiction literature)

IV.2 Health Uncertainty

Health and eventual death of both the offspring and the parents influence the optimization problem of the offspring in several dimensions. The offspring's current health state may impact the value of each smoking alternative contemporaneously (i.e., per period utility). The smoking behavior at period t will impact the probability of health and

death in the future. In fact, one's history of smoking behavior will impact future health outcomes. Likewise, we hypothesize that the observed health and mortality of parents will influence expectations of one's own health. We do not allow the health of the parent to affect per-period utility of the offspring, nor does the smoking behavior of the parent (past or present) affect the offspring's contemporaneous utility.

We denote the adult offspring's health state as H_{ijt}. The health state of parent k in family j is H_{kjt}, where $k \in \{m, f\}$. Mortality in period t of either parent, and the offspring, is similarly denoted M_{kjt} and M_{ijt}. The health and mortality states are assumed to be known by the offspring entering decision period t. However, when evaluating her smoking alternatives today, she knows only the health transition densities (and hence probabilities) of future health states (of herself and her parents).

An adult offspring understands that her period t smoking decision will influence future utility through several channels. First, the period $t+1$ value of the smoking stock is a function of her current period decision:

$$A_{ijt+1} = a(A_{ijt}, d_{ijt})$$

Second, she must forecast the health of her parents ($k \in \{m, f\}$). Sociodemographic characteristics of the parents, known by the offspring, are denoted Y_{kjt}. Additionally, the offspring knows the smoking history of each parent(if still alive) up to period t, S_{kjt}, and assumes that the parents' period t smoking behavior will be whatever it was in the previous period. (The offspring does not probabilistically forecast her parents' smoking behavior. If the parent changes his or her smoking behavior, this change will be reflected in the histories that are revealed at the beginning of the next period.) The densities of the parents' future health states (both health $h(\cdot)$ and mortality $m(\cdot)$) depend on this knowledge of her parents' characteristics. That is,

$$H_{kjt+1} = h(Y_{kjt}, S_{kjt}, \mu_j)$$

$$M_{kjt+1} = m(Y_{kjt}, H_{kjt+1}, S_{kjt}, \mu_j)$$

These densities are correlated (across health outcomes as well as across parents) through a common family unobservable, μ_j. [Note: this μ_j can't be genetics then. Mom and Dad aren't related genetically.]

Finally, the offspring must forecast the probability of her own health state transition and the probability of her own death. These probabilities depend on her own characteristics; namely her smoking history A_{ijt}, the current smoking alternative d_{ijt}, and exogenous individual characteristics X_{ijt}). We also want to allow these probabilities to depend on parental health and mortality. Rather than simply allow parents' health to shift (particular moments of) the distribution of offspring health, we allow the densities themselves to differ. The superscript denotes that these probabilities shift for parent health shocks. That is,

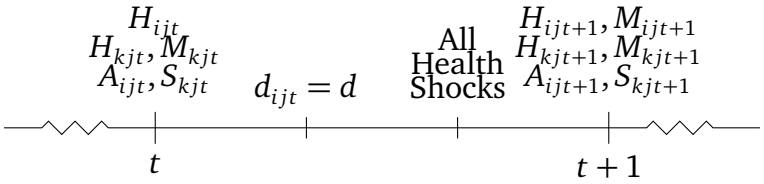
$$H_{ijt+1} = h^{H_{kjt+1}, M_{kjt+1}}(A_{ijt}, d_{ijt}, X_{ijt}, \mu_j)$$

$$M_{ijt+1} = m^{H_{kjt+1}, M_{kjt+1}}(A_{ijt}, d_{ijt}, X_{ijt}, H_{ijt+1}, \mu_j)$$

IV.3 Timing of Information, Decisionmaking, and Health Shocks

Figure 1 shows the timing of a representative period in the model. An adult offspring enters period t with knowledge of her smoking stock A_{ijt}, her own health H_{ijt}, and her parents' mortality and health state M_{kjt}, H_{kjt} for $k \in \{m, f\}$. She also knows the extent to which both parents have smoked in the past S_{kjt}. An offspring i makes a period t smoking decision by evaluating current utility (below) and her expectations about future utility.

Figure 1: Timeline of Information and Decisionmaking



IV.4 Putting it All Together

The period t deterministic utility that offspring i in family j receives from smoking alternative $d_{it} = d$ is:

$$\begin{aligned}\overline{U}^h_{it}(A_{it}, d_{it}, X_{it}, \mu_j) = \alpha_{0h} + \\ +(\alpha_{1h} + \alpha_{2h}A_{ijt} + \alpha_{3h}X_{ijt} + \alpha_{6h}H_{kjt} + \alpha_{7h}M_{kjt}) * \mathbf{1}[d_{it} = 1] \\ +(\alpha_{8h} + \alpha_{9h}A_{ijt} + \alpha_{10h}X_{ijt} + \alpha_{13h}H_{kjt} + \alpha_{14h}M_{kjt}) * \mathbf{1}[d_{it} = 2] \\ +\alpha_{15h} * \mathbf{1}[d_{ijt-1} \neq 0] * \mathbf{1}[d_{ijt} = 0] + \rho^{Uhd}\mu_j\end{aligned} \tag{1}$$

This specification captures the extent to which the marginal utility of smoking is sensitive to past smoking ($\alpha_{2.}, \alpha_{9.}$), exogenous demographics (such as age, gender, and education) ($\alpha_{3.}, \alpha_{10.}$), and the health of a parent ($\alpha_{6.}, \alpha_{13.}$) and ($\alpha_{7.}, \alpha_{14.}$). Note that each parameter is specific to the overall health state of individual i.[20]

The lifetime value of choosing smoking alternative $d_{it} = d$ in period t while in health state $H_{ijt} = h$ is

$$\begin{aligned}V^h_d(A_t, H_t, S_t, \epsilon_t|\mu_j) &= \overline{U}^h_{it}(\cdot) + \epsilon^{hd}_{ijt} \\ &+ \beta \int_{H_k}\int_{M_k}\int_{H_i}\int_{M_k} E_t[V^{h'}(A_{t+1}, H_{t+1}, S_{t+1})|\mu_j, d_{it} = d] m^{H_k,M_k}(\cdot|\mu_j) h^{H_k,M_k}(\cdot|\mu_j) m(\cdot|\mu_j) h(\cdot|\mu_j) \\ &\quad \forall t, d = 0, 1, 2.\end{aligned} \tag{2}$$

The expectation operator is denoted $E_t[\cdot]$ and captures the uncertainty of future smoking preferences of the offspring. The integrals represent uncertainty over 1.) the offspring's parents' health and mortality, and 2.) the offspring's own health and mortality which depends on the health and mortality of her parents. Conditional on the family unobservables, these densities are independent.

The dynamic programming problem can be solved through backward recursion from period $t = T$, where the probability of the offspring's mortality is one (i.e., age 125). The assumption of an additive Extreme-value distributed preference error (ϵ_t) yields multinomial logit probabilities of the smoking alternatives for each period t and every value of

[20] We may need to relax this if, for example, we do not observe many offspring in poor health with a parent in poor health.

the state space entering period t. These probabilities, and the distributional assumptions about the health and mortality transitions, form the likelihood function (conditional on family unobserved characteristics μ_j) contributions of each offspring i and her parents k.

V Discussion

What does estimation of the primitive parameters of the offspring's optimization problem provide us, over estimation of reduced form parameters of an approximation to this problem (i.e., demand equations and health production equations)?

On a fundamental level, we can separately identify the effect of a parent's health shock on utility of different smoking alternatives (e.g., depression, anxiety, altruism, etc.) from a shift in offspring expectations of own health transitions associated with those different smoking decisions.

Furthermore, if we can determine whether these health shocks of parents will alter the smoking behavior of offspring through changes in own health expectations or if an individual's own history of smoking swamps the additional information. That is, the offspring's history impacts his current and future utility of smoking through addictive channels and withdrawal effects. If these effects are large, then an improved understanding of health transition probabilities may not impact the value of smoking sufficiently to overcome the habitual effects of smoking.

Using our model and the estimated parameters, we can simulate offspring smoking behavior over time to examine how smoking changes immediately after a parent's transition to worse health. The key tension here is between the reinforcement (α_{2h}, α_{9h}) and withdrawal (α_{15h}) effects and the effects from parental health: do the parent health shocks convince individuals to quit in spite of the reinforcement and withdrawal effects? *Simulations in Darden (2013) show that the withdrawal effect is really what keeps people smoking.*

The inclusion of a family specific random effect, μ_j, captures common unobservables across family members that may be correlated over time. One interpretation of this family

effect could be genetics. However, we would need to amend our modeling of the correlation between the health outcomes of the mother and father, and that across parents and offspring, in order to differentiate an explanation of genetics (among biological members but not non-biological members) from one of family influence (among all members of a family).

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A Tables

Table 1: Sample Construction

N	Description
4,989	Framingham Heart Survey Offspring Cohort Participants - Limited-Access Sample.
3,730	Sample after dropping all person/year observations of individuals that skipped one or more of the health exams.
3,012	Sample after dropping all person/year observations of individuals who attrit.
2,075	Individuals with at least one matched parent record from FHS Original Cohort
	1,680 with a matched father record
	1,776 with a matched mother record
	1,381 with both a matched mother and father record

Note: 2,075 unique individuals yields 13,456 person/year observations.

Table 2: Summary Statistics: Exam One

	Men		Women	
	Mean	St. Dev	Mean	St. Dev
Offspring - 1,015 Men and 1,060 Women				
Age	35.553	(10.351)	36.145	(10.245)
Over Age 50	0.095	(0.293)	0.098	(0.298)
Education				
High School or Less	0.100	(0.301)	0.078	(0.269)
Some College	0.281	(0.450)	0.357	(0.479)
College Graduate	0.411	(0.492)	0.477	(0.500)
Graduate School	0.208	(0.406)	0.088	(0.283)
Current Smoking	0.403	(0.491)	0.410	(0.492)
Ever Smoker	0.726	(0.446)	0.608	(0.489)
Health State				
CVD	0.200	(0.400)	0.169	(0.375)
Cancer	0.050	(0.218)	0.040	(0.197)
Mother Information				
Missing	0.140	(0.347)	0.148	(0.355)
Deceased	0.153	(0.361)	0.150	(0.357)
Conditional on mother nonmissing and alive				
Ever Smokes	0.512	(0.500)	0.518	(0.500)
Smoking at first offspring exam	0.401	(0.409)	0.387	(0.478)
Health State				
CVD	0.200	(0.400)	0.169	(0.375)
Cancer	0.050	(0.218)	0.040	(0.197)
Father Information				
Missing	0.183	(0.387)	0.197	(0.398)
Deceased	0.291	(0.454)	0.309	(0.462)
Conditional on father nonmissing and alive				
Ever Smokes	0.871	(0.336)	0.893	(0.310)
Smoking at first offspring exam	0.680	(0.467)	0.714	(0.452)
Health State				
CVD	0.262	(0.440)	0.267	(0.443)
Cancer	0.049	(0.217)	0.058	(0.234)

Notes: n=2,075 offspring at exam 1. Less than 1% of offspring in our sample enter with a history of cancer or cardiovascular disease.

Table 3: Per-Period Summary Statistics

	Men		Women	
	Mean	St. Dev	Mean	St. Dev
Offspring - 5,388 male and 5,933 female person/exam observations				
Age	51.760	(11.467)	53.098	(11.736)
Current Smoking	0.236	(0.424)	0.219	(0.414)
Ever Smoker	0.714	(0.452)	0.602	(0.490)
Offspring Health				
Health State				
CVD	0.071	(0.257)	0.036	(0.186)
Cancer	0.016	(0.127)	0.029	(0.167)
New Health Shock				
CVD	0.034	(0.181)	0.015	(0.122)
Cancer	0.020	(0.140)	0.018	(0.131)
Dies	0.037	(0.189)	0.016	(0.126)
Offspring Subjective Health Assessment: Exams 5-7				
Excellent	0.448	(0.497)	0.416	(0.493)
Good	0.479	(0.500)	0.513	(0.500)
Fair/Poor	0.073	(0.261)	0.071	(0.257)
Parent Characteristics conditional on nonmissing and alive				
Mother				
CVD Shock	0.091	(0.287)	0.099	(0.299)
Cancer Diagnosis	0.060	(0.237)	0.060	(0.238)
Father				
CVD Shock	0.122	(0.328)	0.117	(0.321)
Cancer Diagnosis	0.094	(0.292)	0.105	(0.306)

Notes: n=11,381 person/exam observations for exams 2-7.

Table 4: Adult Offspring Ever Smokes

			Group			
Ever Smoked	Full Sample		Women		Men	
Mother	0.111**	0.090**	0.142**	0.136**	0.081**	0.065
	(0.024)	(0.040)	(0.034)	(0.057)	(0.031)	(0.057)
Father	0.080*	-0.006	0.061	-0.021	0.115**	-0.031
	(0.047)	(0.072)	0.060	(0.088)	(0.055)	(0.104)
Family F.E.	No	Yes	No	Yes	No	Yes
Mean	0.666	0.666	0.608	0.608	0.726	0.726
N	2,075	2,075	1,060	1,060	1,015	1,015

Notes: The dependent variable is whether the adult offspring ever smokes through the end of our sample. Linear probability models include age, education, gender, and cohort controls for adult offspring. We also control for missing and deceased mothers and fathers. Whether a parent has ever smoked is defined as any observed smoking through 1975 - the last possible year of the adult offspring first exam. ** indicates p-value$\leq$0.05 * indicates 0.05$<$ p-value$\leq$ 0.1

Table 5: Adult Offspring Smokes

	Group					
	Full Sample		Women		Men	
Ever Smoked						
Mother	0.037	0.031	0.025	0.056	0.044	-0.019
	(0.024)	(0.041)	(0.031)	(0.047)	(0.039)	(0.074)
Father	0.003	0.012	0.004	0.057	0.010	-0.050
	(0.032)	(0.043)	(0.045)	(0.050)	(0.040)	(0.068)
Smoking at First Offspring Exam						
Mother	0.061**	0.075	0.101**	0.101**	0.014	0.061
	(0.032)	(0.051)	(0.035)	(0.043)	(0.043)	(0.086)
Father	0.027	-0.029	0.033	-0.062	0.021	-0.045
	(0.024)	(0.037)	(0.028)	(0.046)	(0.028)	(0.055)
Family F.E.	No	Yes	No	Yes	No	Yes
Mean	0.227	0.227	0.219	0.219	0.236	0.236
N	11,381	11,381	5,993	5,993	5,388	5,388

Notes: Sample includes observations from exams two through seven of the Framingham Heart Study - Offspring Cohort. Linear probability models include age, education, gender, a time trend and cohort controls for adult offspring. We also control for whether the mother and father are missing or dead. ** indicates p-value$\leq$0.05 * indicates 0.05$<$ p-value$\leq$ 0.1

Table 6: Adult Offspring Smokes

	Group					
	Full Sample		Women		Men	
Cardiovascular Shock						
Mother	-0.033	-0.010	-0.020	-0.003	-0.047	-0.020
	(0.024)	(0.024)	(0.034)	(0.031)	(0.033)	(0.033)
Father	0.044	0.060**	0.025	0.055*	0.060	0.076**
	(0.028)	(0.025)	(0.038)	(0.030)	(0.041)	(0.037)
*Cardiovascular Shock*Parent Smoking*						
Mother	0.035	0.007	0.034	-0.029	0.045	0.033
	(0.041)	(0.031)	(0.062)	(0.042)	(0.060)	(0.050)
Father	0.026	0.000	0.042	-0.015	0.004	-0.001
	(0.059)	(0.054)	(0.078)	(0.061)	(0.079)	(0.075)
Cancer Shock						
Mother	-0.055*	-0.018	-0.068*	-0.027	-0.042	-0.011
	(0.030)	(0.030)	(0.038)	(0.029)	(0.044)	(0.045)
Father	-0.014	-0.027	0.064	0.020	-0.103**	-0.053
	(0.027)	(0.024)	(0.040)	(0.030)	(0.037)	(0.034)
*Cancer Shock*Parent Smoking*						
Mother	0.136**	0.044	0.139*	0.028	0.125	0.027
	(0.058)	(0.048)	(0.074)	(0.049)	(0.080)	(0.076)
Father	0.083	0.059	-0.015	-0.006	0.201**	0.145
	(0.062)	(0.049)	(0.087)	(0.063)	(0.089)	(0.099)
Family F.E.	No	Yes	No	Yes	No	Yes
Mean	0.227	0.227	0.219	0.219	0.236	0.236
N	11,381	11,381	5,993	5,993	5,388	5,388

Notes: Sample includes observations from exams two through seven of the Framingham Heart Study - Offspring Cohort. Linear probability models include age, education, gender, time and cohort controls for adult offspring. Parent controls include mortality, CVD and Cancer state variables, whether each parent ever smoked, and whether each parent is missing. ** indicates p-value$\leq$0.05 * indicates 0.05$<$ p-value$\leq$ 0.1

Table 7: Adult Offspring Subjective Health

	Group					
	Full Sample		Current Smokers		Ever Smokers	
Cardiovascular Shock						
Mother	0.014	0.008	-0.168	0.065	0.032	0.035
	(0.056)	(0.063)	(0.106)	(0.175)	(0.065)	(0.076)
Father	0.005	-0.100	-0.098	-0.222	-0.110	-0.157*
	(0.082)	(0.069)	(0.189)	(0.187)	(0.091)	(0.081)
*Cardiovascular Shock*Parent Smoking*						
Mother	-0.072	-0.040	-0.280	-0.039	0.053	0.005
	(0.098)	(0.111)	(0.200)	(0.267)	(0.128)	(0.143)
Father	-0.018	0.056	1.338**	1.379**	0.448	0.591*
	(0.138)	(0.108)	(0.205)	(0.208)	(0.401)	(0.346)
Cancer Shock						
Mother	0.130*	0.134**	-0.227*	-0.167	0.116	0.151**
	(0.073)	(0.063)	(0.138)	(0.210)	(0.085)	(0.075)
Father	-0.020	-0.041	0.040	-0.225	-0.001	0.015
	(0.082)	(0.078)	(0.146)	(0.172)	(0.107)	(0.108)
*Cancer Shock*Parent Smoking*						
Mother	-0.061	-0.201	0.236	0.311	-0.019	-0.206
	(0.166)	(0.124)	(0.296)	(0.281)	(0.204)	(0.170)
Father	0.197*	0.421**	.	.	0.958**	0.776**
	(0.097)	(0.082)	.	.	(0.144)	(0.136)
Family F.E.	No	Yes	No	Yes	No	Yes
St. Dev.	0.612	0.612	0.607	0.607	0.617	0.617
N	5,429	5,429	834	834	3,512	3,512

Notes: Sample includes observations from exams five through seven of the Framingham Heart Study - Offspring Cohort. The dependent variable - self-reported health - takes three values, with higher values indicating worse health. Regression models include age, education, gender, time, health, and cohort controls for adult offspring. Parent controls include mortality, CVD and Cancer state variables, whether each parent ever smoked, and whether each parent is missing. ** indicates p-value$\leq$0.05 * indicates 0.05$<$ p-value$\leq$ 0.1

Table 8: Adult Offspring Smokes

	Group					
	Full Sample		Women		Men	
Own Health Shock						
Cardiovascular Shock	0.025	-0.094**	0.018	-0.103**	0.027	-0.083**
	(0.025)	(0.025)	(0.043)	(0.046)	(0.031)	(0.030)
Cancer Shock	-0.033	-0.040*	-0.067**	-0.032	0.007	-0.044
	(0.022)	(0.022)	(0.031)	(0.031)	(0.033)	(0.031)
Individual F.E.	No	Yes	No	Yes	Yes	Yes
Mean	0.227	0.227	0.219	0.219	0.236	0.236
N	11,381	11,381	5,993	5,993	5,388	5,388

Notes: Sample includes observations from exams two through seven of the Framingham Heart Study - Offspring Cohort. Linear probability models include age, education, gender, health, time and cohort controls for adult offspring. ** indicates p-value$\leq$0.05 * indicates 0.05$<$ p-value$\leq$ 0.1

Table 9: Dependent Variable: Self-Reported Health

	Group					
	Full Sample		Women		Men	
Cardiovascular Shock						
Current Smoker	0.128**	0.016	0.092**	0.002	0.164**	0.034
	(0.029)	(0.049)	(0.039)	(0.063)	(0.043)	(0.077)
Cardiovascular Shock	0.258**	0.084	0.303**	0.128	0.244**	0.061
	(0.058)	(0.066)	(0.094)	(0.101)	(0.073)	(0.085)
Cancer Shock	0.167**	0.134**	0.082	0.034	0.244**	0.217**
	(0.056)	(0.058)	(0.076)	(0.074)	(0.080)	(0.085)
Individual F.E.	No	Yes	No	Yes	No	Yes
St. Dev.	0.612	0.612	0.607	0.607	0.617	0.617
N	5,429	5,429	834	834	3,512	3,512

Notes: Sample includes observations from exams five through seven of the Framingham Heart Study - Offspring Cohort. The dependent variable - self-reported health - takes three values, with higher values indicating worse health. Regression models include age, education, gender, time and cohort controls for adult offspring. ** indicates p-value$\leq$0.05 * indicates 0.05$<$ p-value$\leq$ 0.1