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THE EFFECTS OF PARENTAL HEALTH SHOCKS ON ADULT OFFSPRING SMOKING BEHAVIOR AND SELF-ASSESSED HEALTH

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Abstract

An important avenue for smoking deterrence may be through familial ties if adult smokers respond to parental health shocks. In this paper, we merge the Original Cohort and the Offspring Cohort of the Framingham Heart Study to study how adult offspring smoking behavior and subjective health assessments vary with elder parent smoking behavior and health outcomes. These data allow us to model the smoking behavior of adult offspring over a 30-year period contemporaneously with parental behaviors and outcomes. We find strong “like father, like son” and “like mother, like daughter” correlations in smoking behavior. We find that adult offspring significantly curtail their own smoking following an *own* health shock; however, we find limited evidence that offspring smoking behavior is sensitive to parent health, with the notable exception that women significantly reduce both their smoking participation and intensity following a smoking-related cardiovascular event of a parent. We also model the subjective health assessment of adult offspring as a function of parent health, and we find that women report significantly worse health following the smoking-related death of a parent.

Keywords

I1; I12; D8; D84; Smoking; Intergenerational Transmission of Health Investment; Learning; Panel Data

1 Introduction

Economists define intergenerational transmission as the transfer of individual abilities, traits, behaviors, and outcomes from parents to their children (Lochner, 2008). 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 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.

Recently, economists have examined how smokers formulate health expectations in order to evaluate whether changes in expectations, induced perhaps by policy (such as information campaigns), may influence smokers to quit. Viscusi (1990) and Viscusi and Hakes (2008) find that smokers overstate the risk of lung cancer, and the over-stated 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, but also find that an individual's smoking behavior is not influenced by health shocks to a spouse.² They 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., blood pressure, 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 sensitivity of offspring smoking behavior 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 increased attention to this relationship may be an untapped avenue for effective anti-smoking policy. 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

¹More 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 (Black *et al.*, 2005), 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.

²Christakis and Fowler (2008) show that smoking behavior is influenced by the smoking behavior of those within the same social network. They also show that the social networks of smokers are increasingly *defined* by the fact that they smoke.

³A notable exception is Parkes *et al.* (2008), who find that informing smokers of their "lung-age" - the age of the average healthy individual who 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.

(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, fuzzy expectations of one's own future health may not impact smoking behavior of adolescents and young adults today. Yet, if a parent's current health shock impacts expectations of an offspring's future health enough to overcome both own preferences for smoking and discounting, then current smoking behavior may be curtailed. To our knowledge, no paper has studied the extent to which adult offspring smoking behavior and own health assessment is sensitive to parental smoking and/or health outcomes.

In this paper, we present descriptive results from a novel intergenerational dataset that relate to two questions on the intergenerational transmission of smoking behavior and health. First, we examine how adult offspring smoking is influenced by lifetime and contemporaneous parent smoking. Second, we address the extent to which adult off-spring change their smoking behavior and their subjective health assessments when an elder parent experiences a health shock - smoking-related or otherwise. Furthermore, we examine both of these questions along gender lines to assess the potential for "like father, like son" effects. Finally, to relate our work to the literature on health, information, and smoking, we examine the extent to which smoking behavior and subjective health assessments are influenced when an individual herself experiences a major health shock.

To empirically address these questions, we construct a novel panel dataset of parents and adult offspring from the Framingham Heart Study (FHS). Indeed, we are among the first social scientists to merge the FHS Original Cohort (initially interviewed in 1948 and followed to the present) with the FHS Offspring cohort (initially interviewed in 1971 and followed to the present).⁴ In our final estimation sample, we observe 2,402 adult offspring through seven detailed health exams/interviews over a 30-year period, and we model their smoking and subjective health assessments as a function of their Original Cohort parents' contemporaneous health exam/interview results. We interpret our results as associations which could plausibly be interpreted as causal; we control for unobserved heterogeneity with individual and time fixed effects and focus on within-individual deviations from a common trend.

From 1970 to 2001, the percentage of adults in the United States who smoke cigarettes declined from 37.4% to 22.8%.⁵ Christakis and Fowler (2008) use FHS 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 non-smokers, those authors show that by 2001, smokers became increasingly isolated in 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

⁴Christakis and Fowler (2008) use these FHS cohorts to examine behaviors within social networks, which include parent-child interactions.

⁵http://www.cdc.gov/tobacco/data_statistics/tables/trends/cig_smoking/index.htm.

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 results show several interesting trends emerging from the data. First, we find strong “like father, like son” and “like mother, like daughter” correlations in lifetime smoking. Our results suggest that a woman is 27.8% more likely to ever smoke if her mother ever smoked. The similar effect for men and fathers is 23.2%. We find limited evidence of changes in adult offspring smoking - either smoking participation or intensity - when a parent experiences either a smoking-related or general cardiovascular or cancer diagnosis; however, a notable exception is the smoking behavior of women following a smoking-related cardiovascular event to a parent. Indeed, at the mean of female smoking, we find that smoking prevalence declines by 33.3% and smoking intensity conditional on past smoking declines by 25.8% following a father’s smoking-related cardiovascular event. Unsurprisingly however, when adult offspring themselves experience the health shock, our results suggest that both smoking participation and intensity decline dramatically for both men and women.⁶

We also model an adult offspring’s subjective health assessment as a function of own and parental health. We find limited evidence that adult offspring significantly change their subjective health assessment following a health shock to a parent, but again an exception is with respect to women. Our results suggest that female adult offspring report significantly worse subjective health following the smoking-related death of a mother or father. Unfortunately, our sample is not large enough to further disaggregate our subjective health regressions by the smoking status of the offspring.

This paper proceeds as follows. Section 2 describes mechanisms for the intergenerational transmission of behavior and the literature on responses to health information. Section 3 describes the Framingham Heart Study. Section 4 presents our empirical model and our main results. Section 5 concludes.

2 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. These mechanisms may explain, for example, an observed correlation between mother’s smoking and adult offspring smoking of 0.14 found in the FHS cohorts.⁷

⁶See Smith *et al.* (2001); Sloan *et al.* (2003); Khwaja *et al.* (2006); Arcidiacono *et al.* (2007) for evidence that an individual responds to an own health shock by changing her smoking behavior.

⁷This raw correlation between maternal smoking and adult offspring smoking varies with whether or not the mother (and father) is still alive; correlations between paternal smoking and adult offspring smoking, although smaller, are similarly divergent depending on the survival state of both parents.

2.1 Transmission at Birth: Genetics

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 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).

2.2 Transmission in the Early Years: Consumption Externality or Role-Model

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 home 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.⁸ 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).

2.3 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

⁸This statistic and other similar information about smoking initiation can be found in CDC Fact Sheet (2012) and USDHHS (2014).

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.⁹ 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 adult offspring smoking behavior and own health assessment addresses the transmission mechanisms that may exist contemporaneously as adults. We examine a unique dataset that follows both parents and their adult offspring over 30 years. In this paper, we provide a descriptive exploration of existence of the information/altruism mechanism. Using panel data on concurrent outcomes of parents and offspring, we seek evidence on whether parental health shocks impact adult

⁹Most of the changes in behavior are quits and reductions in smoking. Initiation of smoking and transition from occasional smoking to daily smoking are quite rare (i.e., <1.5% and <4.3%, respectively) after age 26 (USDHHS, 2014).

offspring smoking behavior and own health assessment. Our subsequent work explicitly models dynamic smoking decisionmaking over a lifetime and allows for parental influence through observed (simultaneous or recent) health outcomes and smoking behavior.

3 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 whom consented for their health exam results to be released. Our sample construction hinges on two criteria. First, only 3,350 of the 4,989 baseline Offspring Cohort participants has a match in the parent data.¹⁰ Therefore, for those participants, we do not have data on parent smoking or health outcomes, and we drop them from our estimation sample. Second, the FHS asked questions about subjective health assessment in Offspring exams five through seven. Thus, we drop Offspring participants who left the sample - either through death or attrition - prior to exam 6.¹¹ Finally, because our identification strategy exploits the within individual variation in smoking and subjective health assessment, we keep only Offspring participants who are observed at least twice between exams five through 12 seven.¹²

As shown in Table 1 our final estimation sample includes 2,402 Offspring participants for a total of 15,843 person/exam observations. Of the 2,402 individuals we study, 2,047 have a matched mother, 1,992 have a matched father, and 1,637 match both parents; for those with one parent missing, we create a missing parent indicator rather than drop this person from the analysis. For the merged parents, we keep information on smoking behavior and the specific years and ages of cardiovascular shocks,¹³ cancer diagnoses,¹⁴ and death.

¹⁰An 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.

¹¹Smoking behavior is measured in all Offspring exams; however, to maintain a consistent sample between our smoking and subjective health models, we focus on individuals in the sample through at least exam six.

¹²FHS 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. However, we do recognize the potential for bias stemming from endogenous sample selection. Given that we want to estimate the smoking response and the subjective health assessment response to parental health shocks *on the same group of people*, our sample restrictions are by necessity given our data. The main results of our paper are robust to different sample definitions. Results from models estimated with a less restricted sample are available upon request.

¹³These include coronary heart disease, myocardial infarction, angina pectoris, coronary insufficiency, stroke, intermittent claudication, and congestive heart failure.

¹⁴We have information on the specific site of the cancer diagnosis, but for sample size reasons, we aggregate these to a simple binary cancer indicator.

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 1975.¹⁵ Age at the first offspring exam ranges from 13 to 62 years, with an average age of 33 for men and 34 for women. While slightly more women than men smoke at the first exam (39.5% vs. 38%), male smokers smoke on average 4.1 cigarettes more per day. Men are also more likely than women to have ever smoked in their lifetime. Roughly, 13% of mothers and 25% of fathers with a record in the FHS Original Cohort died prior to the first Offspring exam. Of offspring participants with a matched father, approximately 87% of fathers smoked at some point; the similar number for mothers is about 52%. At the first Offspring exam, roughly 24% (15%) of fathers (mothers) previously had a cardiovascular event and 5% (4%) had a history of cancer.

Subsequent offspring exams occurred at roughly five-year intervals. Table 3 provides summary statistics for exams two through seven. Our sample is constructed such that individuals must have completed at least two exams between exams five and seven; however, conditional upon this restriction, individuals could be observed to a.) miss exams and/or b.) die or attrit after completing exam six. 91.4% of the 2,402 Offspring individuals in our sample miss at most one exam; of the 2,308 individuals that are observed to take exam six, 40 are observed to die and 86 are observed to attrit following exam six.¹⁶

We code a health shock at a particular exam if it occurred prior to the current exam but after the previous exam. In our sample, cardiovascular events occur in any given exam period, on average, for 3% of men and 1.5% of women. After an event, a time-varying variable indicates that a person has experienced a shock. For example, 5.6% of men and 3.2% of women have a history of cardiovascular events at any given time. We code parental health shocks similarly. As an example of the prevalence of shocks, approximately 10% of fathers (who are nonmissing and who remain alive) are diagnosed with cancer between any of the offspring exams two through seven.¹⁷

Smoking prevalence over exams two through seven declines from a high (at exam one) of 38.04% for men and 39.55% for women to 14.10% and 13.78%, respectively. Although a variety of objective health measures are provided at each exam, the FHS records a participant's subjective health assessment in exams five through seven only. Women are less likely to report excellent health than are men (42% versus 44.9%).

4 Empirical Model and Results

We begin our empirical analysis by examining the relationship between whether an 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).¹⁸ We estimate the following linear probability model:

¹⁵FHS did not disclose to us 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).

¹⁶Individuals could be included in our sample if they miss exam six but take exams five and seven.

¹⁷Recall that the Original Cohort participants ranged in age from 30 to 60 in 1948. By 2001 (i.e., the end of our data for both cohorts), only 588 of the initial 5,079 Original Cohort participants remain alive.

$$E_i = X_i'\beta + Z_i'\gamma + \epsilon_i \quad (1)$$

where E_i is an indicator for whether offspring i ever smoked; X_i is a vector of offspring controls including age, cohort, and education indicator variables; Z_i is a vector of mother and father variables; and ϵ_i is the error term. Because we observe data on only one parent (rather than the mother/father pair) for some offspring, we include missing and deceased parent indicator variables in Z_i rather than estimate separate models to identify the impact of each familial relationship. Our analysis in estimating Equation 1 is descriptive: we investigate the conditional correlation between parent and offspring smoking.

The results, summarized in Table 4, suggest strong “like father, like son” and “like mother, like daughter” correlations in lifetime smoking patterns. Indeed, at the mean level of offspring male smoking, an offspring man is 23% more likely to have ever smoked if his father also smoked. Similarly, an offspring woman is roughly 28% more likely to have ever smoked if her mother ever smoked. Interestingly, the cross-gender effects are positive but statistically insignificant and much smaller in magnitude.

Economic theory suggests that the smoking decision is a function of a stock of addictive capital (which depends on one’s history of smoking) as well as current cigarette prices, demographic, and socio-economic characteristics. Additionally, this forward-looking decision process is a function of self-assessed health, realizations of current health shocks, and the expectations regarding future health shocks.¹⁹ In this paper, we take a reduced-form approach with the fully structural model in mind. We exploit the panel nature of the FHS data and model contemporaneous cigarette smoking as a function of individual characteristics, individual health shocks, and parental health shocks. We hypothesize that the observation of a parent health shock may influence perceptions of one’s own health, and the resulting *shift* in expectations regarding one’s own health may induce changes in smoking behavior. While we do not know whether these parental health shocks are directly caused by the smoking behavior of the parent or not, we interact them with the last measured smoking status of a parent prior to the health event.²⁰ We estimate linear probability models of the following form:

$$S_{it} = \alpha_i + X'_{it}\beta_1 + A'_{it-1}\beta_2 + H'_{it-1}\beta_3 + H'^P_{it-1}\beta_4 + \sum_{j=3}^7 e_j + \epsilon_{it} \quad (2)$$

where S_{it} is a binary variable for smoking in exam t ; X'_{it} is a vector of individual-specific characteristics such as age, cohort, and education indicators; A'_{it-1} is a vector of smoking

¹⁸At the first offspring exam, the FHS collects retrospective smoking information. We combine these data with responses each exam thereafter to construct an indicator of whether the individual ever smoked.

¹⁹See Becker and Murphy (1988) for the original rational addiction model. See Darden (2010) for a dynamic, life cycle model of the smoking decision process where future health uncertainty and the role of current and past smoking behaviors are made explicit and estimated using FHS data.

²⁰Cigarette smoking causes several forms of both cancer and cardiovascular disease (United States Department of Health and Human Services, 2004, 2010). Recent findings suggest smoking causes many additional diseases (Carter *et al.*, 2015).

duration variables that capture individual i 's smoking history and that are updated through exam $t - 1$; H'_{it-1} is a vector of binary *own* health shocks that may have occurred between exam $t - 1$ and exam t and *own* health histories that capture the individual's past health shocks; and H'^P_{it-1} is a vector of binary *parent* health shocks that may have occurred between exam $t - 1$ and exam t and *parent* health histories that capture the specific parent's past health shocks. We allow for individual permanent unobserved heterogeneity through an individual-specific fixed effect α_i , and we include exam indicator variables, e_j , capturing aggregate time (i.e., small window of months) or exam-related unobservables.²¹ We define the first instance of a parental cardiovascular, cancer, or mortality event occurring in between two adjacent offspring exams as a parental health shock. We continue to control for the parent's history of such shocks (including those that occurred prior to the initial offspring exam) to capture any long-term influence of the shock on current offspring behavior. A parent mortality shock prior to exam t is reflected by the deceased indicator in subsequent periods.²²

Results summarized in Table 5 reveal the marginal effects of own and parent health on adult offspring smoking. Columns (1) and (3) present impacts on current smoking behavior unconditional on previous smoking behavior, for men and women respectively. Columns (2) and (4) focus on the current behavioral impacts for those offspring who smoked in the previous exam interval. First, consistent with the literature, we find negative and significant effects of own health shocks on smoking. For example, among men, a cardiovascular health event experienced in the previous period (and conditional on surviving the event), reduces smoking prevalence on average (and at the mean level of male smoking) by 28.7%. We observe larger effects (i.e., reductions of up to 34.8%) for cancer diagnoses. The effects are typically larger for women than for men.

We find some statistical evidence that adult offspring respond to the health shocks of parents by changing their smoking behavior. Our results suggest that women are less likely to smoke when they observe a cardiovascular shock experienced by a mother or father who was a smoker at the time of the shock; the similar 33% reductions at the mean smoking prevalence is also similar in magnitude to those following a health shock of one's own. Furthermore, a male smoker is significantly less likely to smoke when his smoking mother receives a cancer diagnosis. One surprising finding is that male smokers are significantly less likely to quit smoking following a non-smoking father's cardiovascular shock.

Because we include individual- and exam-level fixed effects, our identification strategy relies on within-individual variation in smoking. That is, our research design is valid if time-varying unobserved heterogeneity that is not common to all individuals (and independent of the i.i.d. error term) does not exist. To check for the presence of time-varying unobserved heterogeneity, we re-estimate our model in Equation 2 with interaction terms that allow the time effects (e_j) to differ by whether or not an offspring's parents are *ever* observed to have a

²¹We estimate Equation 2 on data from exams two through seven.

²²Although we control for a parent's history of particular health shocks, we indicate the first shock only (rather than include indicators each time a parent experiences a subsequent health shock of the same type. Khwaja *et al.* (2006) claim that the first instance of a particular major health shock is the informative shock.

cardiovascular, cancer, or mortality event. Our intuition is that the presence of time-varying unobserved heterogeneity would show up as differential trends in smoking behavior, conditional on the observed heterogeneity included in the model. At the bottom of Table 5 we present the p-values for hypothesis tests of equal trends. While we cannot rule out time-varying unobserved factors, we find no evidence of significantly different trends in smoking behavior.

To further examine the mechanism of information transmission suggested by parental health shocks, we replace offspring smoking behavior as the dependent variable in Equation 2 with offspring smoking intensity measured by cigarettes per day. While parental health shocks can induce offspring to quit (as evidenced above), such shocks may also reduce the cigarette consumption of smoking offspring. We condition estimation of Equation 2 on smoking in the previous period, but not current smoking; a previous smoker who quits at t has an intensity of smoking equal to zero. Columns (1) and (2) of Table 6 display the impacts of own and parent health on adult offspring smoking intensity. As in Table 5, we find large and statistically significant reductions in smoking intensity following own's own health shock. While we find a limited response of smoking intensity to parent health shocks generally, we find further evidence that female smokers respond to the cardiovascular shocks of smoking fathers - in this case by reducing the intensity of smoking. Our results suggest that male smokers smoke *more* cigarettes per day following a cancer diagnosis to a nonsmoking mother, but, while not statistically significant, this effect is almost canceled out if the mother is a smoker.

Columns (3) and (4) of Table 6 replace our smoking dependent variables with an offspring's subjective health assessment (up to three observations per offspring spanning exams 5–7). We model this assessment as a function of the same right-hand side variables in Equation 2 and individual and exam-level fixed effects. 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.²³ Our results suggest that the death of a smoking parent worsens the subjective health assessment of female offspring. At the mean of female offspring subjective health, the death of a smoking parent - mother or father - lowers subjective health by 16% or 17%. We find no significant effects for male offspring, with the exception of an own cancer diagnosis, which reduces his subjective health assessment.

5 Discussion

In this paper, we exploit a novel panel dataset of adult offspring to estimate the influence of parental smoking and parent health shocks on the smoking behavior and subjective health assessments of adult offspring. Indeed, we are the first social science researchers to merge the Original and Offspring Cohorts of FHS for the purpose of modeling offspring behavior. Our data allow us to model adult offspring behavior over a 30-year period as a function of contemporaneous parent behavior and outcomes.

²³We have estimated multinomial logit models of subjective health, but we prefer linear regression models when including fixed effects because of the incidental parameters problem. Additionally, our sample size prevents us from disaggregating the subjective health regressions by smoking status.

Our results suggest the following conclusions. First, with respect to lifetime smoking participation, we find strong “like father, like son” and “like mother, like daughter” correlations that are consistent with the smoking initiation literature (Loureiro *et al.*, 2006). Interestingly, we do not find opposite gender effects; however, this finding may be because of our setting - 87% of the fathers in our sample had ever smoked by 1971 – or because we are considering adult sons. Second, any evidence of a foreshadowing effect in which adult offspring modify their smoking behavior after observing a parental health shock is limited to women. This result challenges the existing literature that finds that heavy smokers do not quit smoking when spouses have smoking-related health shocks (Khwaja *et al.*, 2006). In fact, we find strong effects among women who observe smoking-related cardiovascular shocks to parents (especially fathers). This result is statistically significant in the full sample but not in the sample of current smokers, suggesting perhaps that the margin on which the “information” is salient is the light to moderate occasional smoker. Third, we find evidence that parental mortality does correlate with worse adult offspring subjective health assessments in women. Finally, we find that own health shocks induce offspring to smoke less and report worse subjective health.

While we control for time-invariant unobserved heterogeneity, as well as common trends in smoking, we view our results are ultimately descriptive. Although we cannot reject the null hypothesis of common trends for those offspring that observe different parent health shocks, we cannot rule out that our results are driven by time-varying unobserved characteristics that are not common to individuals - for example, advice from a physician to quit smoking. Furthermore, our data force us to restrict our sample in such a way that endogenous sample selection may be an issue. Finally, while our models do account for the dynamics of offspring smoking through reinforcement, tolerance, and withdrawal controls, our empirical model technically assumes myopic expectations with respect to future health outcomes. Our next step is to explicitly model expectations in a dynamic, structural model of forward-looking smoking decisionmaking that is estimated with merged parental smoking and health information. Capturing the smoking dynamics and future health uncertainty explicitly will allow us to simulate changes in offspring smoking for a variety of informational events.

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Appendix

Table A1:

Additional Estimation Results: Offspring Current Smoker

Variable	Men		Women	
	Unconditional on previous smoking	Conditional on previous smoker	Unconditional on previous smoking	Conditional on previous smoker
<i>Parent Health History</i>				
Mother CVD	0.008	0.098 [*]	-0.025	-0.110 [*]

Variable	Men		Women	
	Unconditional on previous smoking	Conditional on previous smoker	Unconditional on previous smoking	Conditional on previous smoker
	(0.023)	(0.056)	(0.021)	(0.065)
Father CVD	0.044	0.123 *	−0.004	0.011
	(0.028)	(0.071)	(0.021)	(0.08)
Mother Cancer	−0.008	−0.057	−0.012	0.012
	(0.028)	(0.07)	(0.026)	(0.073)
Father Cancer	−0.024	−0.105	−0.001	0.035
	(0.033)	(0.099)	(0.027)	(0.062)
Mother Death	0.004	0.034	0.001	0.022
	(0.018)	(0.052)	(0.015)	(0.049)
Father Death	0.000	−0.031	0.019	0.068
	(0.019)	(0.055)	(0.016)	(0.051)
<i>Offspring Characteristics</i>				
Age	0.013 **	0.151 **	0.003	0.033
	(0.005)	(0.022)	(0.005)	(0.027)
Experience Smoking (years)	−0.036 **	−0.135 **	−0.036 **	−0.036
	(0.002)	(0.015)	(0.002)	(0.026)
Duration Smoking (years)	0.009 **	0.004	0.009 **	−0.008 *
	(0.001)	(0.003)	(0.001)	(0.005)
Duration Cessation (years)	0.002		0.002 *	
	(0.001)		(0.001)	
History of CVD	−0.034	−0.144 *	0.004	−0.176
	(0.03)	(0.077)	(0.039)	(0.114)
History of Cancer	−0.034	−0.145	−0.025	−0.182
	(0.039)	(0.14)	(0.023)	(0.136)
Exam 3	−0.114 **	−0.241 **	0.005	−0.113
	(0.026)	(0.075)	(0.022)	(0.079)
Exam 4	−0.154 **	−0.415 **	−0.027	−0.179
	(0.042)	(0.128)	(0.038)	(0.141)
Exam 5	−0.209 **	−0.564 **	−0.037	−0.225
	(0.061)	(0.191)	(0.054)	(0.205)
Exam 6	−0.271 **	−0.713 **	−0.051	−0.242
	(0.084)	(0.261)	(0.072)	(0.281)
Exam 7	−0.307 **	−0.784 **	−0.063	−0.254
	(0.101)	(0.317)	(0.086)	(0.335)
Constant	0.183	−2.467 **	0.534 **	0.526
	(0.225)	(0.72)	(0.196)	(0.821)

Notes: Table includes estimates and standard errors of the remaining explanatory variables not reported in Table 5. Time invariant controls drop out due to the individual fixed effects. Duration smoking is the number of years smoking since last not smoking.

** indicates p-value 0.05,

* indicates 0.05 p-value < 0.1.

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Table 1:**Research Sample Construction**

Sample size	Description
4,989	Number of participants in FHS Offspring Cohort - Limited-Access Sample
3,677	Sample size after dropping all individuals whose last observed exam is the 5th exam or earlier
3,546	Sample size after dropping those who do not take at least two exams between exams 5 and 7
2,402	Sample size after dropping those with no matched parent record in FHS Original Cohort 1,992 offspring participants have a matched mother record 2,047 offspring participants have a matched father record 1,637 offspring participants have a matched mother and father record

Note: The research sample includes 2,402 unique offspring individuals and yields 15,843 person/year observations.

Table 2:**Research Sample Summary Statistics: Information Entering Sample**

Variable	Men		Women	
	Mean	St. Dev	Mean	St. Dev
Offspring Characteristics				
Age	33.313	(9.797)	34.572	(10.144)
Over Age 50	0.052	(0.222)	0.072	(0.258)
Education				
High School or Less	0.182	(0.386)	0.166	(0.372)
Some College	0.240	(0.427)	0.316	(0.465)
College Graduate	0.377	(0.485)	0.431	(0.495)
Graduate School	0.201	(0.401)	0.087	(0.282)
Current Smoker	0.380	(0.486)	0.395	(0.489)
Cigarettes/Day if Smoker	22.392	(11.787)	18.184	(11.710)
Ever Smoker	0.521	(0.500)	0.461	(0.499)
Mother Characteristics				
Missing	0.149	(0.356)	0.147	(0.354)
Deceased	0.123	(0.328)	0.130	(0.336)
If mother is observed				
Ever Smoker	0.518	(0.500)	0.522	(0.500)
Health State				
CVD	0.171	(0.377)	0.145	(0.352)
Cancer	0.043	(0.203)	0.039	(0.193)
Father Characteristics				
Missing	0.162	(0.368)	0.179	(0.383)
Deceased	0.246	(0.431)	0.274	(0.446)
If father is observed				
Ever Smoker	0.859	(0.349)	0.891	(0.311)
Health State				
CVD	0.232	(0.422)	0.251	(0.434)
Cancer	0.048	(0.214)	0.054	(0.226)

Note: Sample sizes are 2,402 offspring, 2,047 mothers, and 1,992 fathers at exam one. Exam one was administered between 1971 and 1975.

Table 3:

Research Sample Summary Statistics: All person-years

Variable	Men		Women	
	Mean	St. Dev	Mean	St. Dev
Offspring Characteristics				
Age	51.351	(11.571)	52.594	(11.820)
Current Smoker	0.230	(0.421)	0.225	(0.418)
Cigarettes/Day if Smoker	22.453	(14.514)	19.401	(11.042)
Ever Smoker	0.522	(0.500)	0.458	(0.498)
New Health Shock				
CVD	0.030	(0.170)	0.015	(0.121)
Cancer	0.017	(0.128)	0.015	(0.12)
Death (Following exam 6 or 7)	0.013	(0.114)	0.006	(0.078)
Health History				
CVD	0.056	(0.229)	0.032	(0.175)
Cancer	0.019	(0.138)	0.030	(0.171)
Subjective Health Assessment (Exams 5–7)				
Excellent	0.449	(0.497)	0.420	(0.494)
Good	0.480	(0.500)	0.511	(0.500)
Fair/Poor	0.071	(0.257)	0.070	(0.254)
Parent Characteristics (conditional on nonmissing and alive)				
Mother				
CVD Shock	0.096	(0.295)	0.102	(0.303)
CVD Shock $\times$ Current Smoker	0.234	(0.424)	0.220	(0.415)
Cancer Diagnosis	0.068	(0.251)	0.061	(0.240)
Cancer Diagnosis $\times$ Current Smoker	0.220	(0.415)	0.228	(0.420)
Death	0.107	(0.310)	0.109	(0.312)
Death $\times$ Current Smoker	0.156	(0.363)	0.155	(0.363)
Father				
CVD Shock	0.130	(0.336)	0.123	(0.328)
CVD Shock $\times$ Current Smoker	0.348	(0.477)	0.281	(0.450)
Cancer Diagnosis	0.098	(0.297)	0.106	(0.308)
Cancer Diagnosis $\times$ Current Smoker	0.282	(0.451)	0.347	(0.477)
Death	0.111	(0.314)	0.105	(0.307)
Death $\times$ Current Smoker	0.232	(0.423)	0.276	(0.448)

Note: Sample size is 13,441 person/exam observations for exams 2–7.

Table 4:

Selected Estimation Results: Offspring Ever Smoker

Variable	Research Sample	Men	Women
Mother: Ever Smoker	0.089 ** (0.023)	0.035 (0.033)	0.128 ** (0.031)
Father: Ever Smoker	0.068 ** (0.034)	0.121 ** (0.047)	0.021 (0.049)
Mean	0.489	0.521	0.461
Sample size	2,402	1,120	1,282

Note: The dependent variable is whether the adult offspring ever smokes through the end of our sample. Linear probability models include age, education, and cohort effects. Additional parent controls include whether each parent ever smoked and indicators of missing parent.

** indicates p-value 0.05,

* indicates 0.05 < p-value 0.1.

Table 5:

Selected Estimation Results: Offspring Current Smoker

Variable	Men		Women	
	Unconditional on previous smoking	Conditional on previous smoker	Unconditional on previous smoking	Conditional on previous smoker
<i>Own Health</i>				
CVD Shock	−0.066** (0.023)	−0.159** (0.064)	−0.079** (0.037)	−0.274** (0.097)
Cancer Shock	−0.080** (0.028)	−0.252** (0.102)	−0.027 (0.022)	−0.139 (0.103)
<i>Parent Health</i>				
<i>Cardiovascular Shock</i>				
Mother	−0.005 (0.020)	0.076 (0.070)	0.013 (0.021)	0.004 (0.066)
Father	0.039 (0.029)	0.174* (0.080)	0.020 (0.021)	0.017 (0.079)
<i>Cardiovascular Shock × Parent Smoker</i>				
Mother	0.038 (0.036)	0.066 (0.094)	−0.074** (0.034)	−0.068 (0.073)
Father	0.036 (0.037)	−0.086 (0.081)	−0.075** (0.030)	−0.083 (0.084)
<i>Cancer Shock</i>				
Mother	0.039 (0.024)	0.062 (0.062)	−0.044 (0.027)	−0.068 (0.074)
Father	−0.024 (0.034)	−0.225** (0.111)	−0.005 (0.026)	0.025 (0.068)
<i>Cancer Shock × Parent Smoker</i>				
Mother	−0.052 (0.052)	−0.180* (0.098)	0.006 (0.037)	0.021 (0.108)
Father	0.081 (0.056)	0.162 (0.125)	0.017 (0.038)	0.040 (0.095)
<i>Death</i>				
Mother	−0.010 (0.014)	0.022 (0.049)	−0.001 (0.014)	0.008 (0.044)
Father	−0.005 (0.017)	−0.076 (0.050)	0.011 (0.014)	0.058 (0.046)
<i>Death × Parent Smoker</i>				
Mother	−0.001 (0.033)	−0.048 (0.080)	−0.016 (0.035)	−0.048 (0.062)
Father	−0.046 (0.037)	0.052 (0.074)	−0.011 (0.023)	−0.085 (0.067)

Variable	Men		Women	
	Unconditional on previous smoking	Conditional on previous smoker	Unconditional on previous smoking	Conditional on previous smoker
Mean	0.230	0.721	0.225	0.748
CVD p-value	0.907	0.300	0.875	0.972
Cancer p-value	0.301	0.866	0.469	0.440
Mortality p-value	0.466	0.616	0.561	0.139
Sample size	6,265	1,707	7,176	1,939

Note: Sample includes observations from exams 2–7. Linear probability models include age, education, time and cohort controls, years of smoking for current smokers, and years since last smoking for those who ever smoked. Additional parent controls include death prior to t , CVD and Cancer history, whether each parent ever smoked, and indicators of missing parent.

** indicates p-value < 0.05 ,

* indicates $0.05 < \text{p-value} < 0.1$.

Table 6:

Selected Estimation Results: Offspring Smoking Intensity and Subjective Health

Variable	Smoking Intensity		Subjective Health	
	Men	Women	Men	Women
<i>Own Health</i>				
CVD Shock	-4.555** (2.014)	-7.039** (2.657)	0.072 (0.077)	0.107 (0.083)
Cancer Shock	-10.891** (3.072)	-7.037** (2.472)	0.232** (0.077)	0.041 (0.077)
<i>Parent Health</i>				
<i>Cardiovascular Shock</i>				
Mother	3.028 (2.135)	0.386 (1.765)	-0.163* (0.087)	0.088 (0.070)
Father	3.533 (2.178)	1.316 (1.527)	-0.103 (0.125)	-0.017 (0.098)
<i>Cardiovascular Shock × Parent Smoker</i>				
Mother	4.459 (2.842)	-2.578 (2.085)	0.025 (0.134)	-0.072 (0.107)
Father	0.268 (2.413)	-3.895* (2.170)	0.082 (0.143)	-0.107 (0.171)
<i>Cancer Shock</i>				
Mother	4.851** (1.836)	-4.497** (1.915)	0.098 (0.090)	0.103 (0.076)
Father	-7.673** (2.835)	-1.896 (1.392)	0.047 (0.123)	0.106 (0.137)
<i>Cancer Shock × Parent Smoker</i>				
Mother	-3.277 (3.237)	-0.471 (2.61)	-0.073 (0.138)	0.218* (0.122)
Father	5.706 (3.808)	3.340 (2.194)	-0.052 (0.192)	-0.281 (0.188)
<i>Death</i>				
Mother	-0.854 (1.462)	-0.567 (1.094)	-0.068 (0.050)	-0.047 (0.041)
Father	-1.268 (1.803)	1.531 (1.107)	-0.012 (0.060)	-0.029 (0.057)
<i>Death × Parent Smoker</i>				
Mother	-0.461 (2.618)	0.442 (1.696)	0.070 (0.137)	0.263** (0.092)
Father	-0.089 (2.450)	-1.339 (1.664)	0.041 (0.117)	0.282** (0.091)

Variable	Smoking Intensity		Subjective Health	
	Men	Women	Men	Women
Mean	17.792	15.103	1.622	1.650
CVD p-value	0.178	0.591	0.319	0.185
Cancer p-value	0.985	0.140	0.950	0.090
Mortality p-value	0.189	0.151	0.765	0.600
Sample size	1,707	1,938	3,186	3,654

Note: Sample includes observations from exams 2–7. Linear regression models include age, education, and time and cohort controls for adult offspring. Additional parent controls include death prior to t, CVD and Cancer history, whether each parent ever smoked, and indicators of missing parent.

** indicates p-value 0.05,

* indicates 0.05 < p-value 0.1.