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ABSTRACT

This paper studies how health behaviors and investments are shaped through intra- and inter-generational family spillovers. Specifically, leveraging administrative healthcare data, we identify the effects of health shocks to individuals on their family members' consumption of preventive care and utilization indicative of health-related behaviors. Our identification strategy relies on the timing of shocks by constructing counterfactuals to affected households using households that experience the same shock but a few years in the future. We find that spouses and adult children immediately increase their health investments and improve their health behaviors in response to family shocks, and that these effects are both significant and persistent for at least several years. Notably, we find that these spillover effects in consumption of healthcare are far-reaching and cascade to siblings, stepchildren, sons and daughters in-law, and even "close" coworkers. Using different strategies we show that while a variety of mechanisms seem to be at play, including learning new information about one's own health, there is consistent evidence in support of salience as a major operative explanation, even when the family shock was likely uninformative. Our results have implications for models of health behaviors, by underscoring the importance of one's family and social network in their determination, and are potentially informative for policies that aim to improve population health.

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1 Introduction

Health behaviors, broadly defined as any action, investment, or consumption choice that can affect health and mortality risk, are a key input in the production of individuals' health. These behaviors take a variety of forms including both adverse habits, such as smoking and drinking, and positive actions, such as the consumption of risk-reducing preventive care.

A long tradition of economic research has underscored the importance of family interactions in determining individual behavior, particularly in the context of sharing financial resources for the consumption of goods and of labor supply choices. Similarly, there is a potentially significant role for the family in forming health behaviors over the natural course of the life cycle, through a variety of channels such as the flow of information, attention and awareness, belief formation, and norms. Yet, identifying causal relationships of family spillovers that can affect health behaviors is challenging due to the possibility of correlated unobservables across and within generations of the family. Moreover, it requires large data sets on health and consumption of healthcare with linkages across family members.

In this paper, we study how health behaviors are shaped through family spillovers both within and across generations. Specifically, we estimate the causal effects of health shocks to individuals on their family members' consumption of preventive care and utilization indicative of health-related behaviors, focusing on spouses and adult children. Our estimation strategy relies on the timing of shocks, by constructing counterfactuals to affected households using households that experience the same shock but a few years in the future. To do so, we leverage a long panel of administrative data with comprehensive and detailed information on health and healthcare utilization. The data, which cover the entire adult Danish population from the years 1980-2011, include all medication prescriptions, contacts with primary-care physicians and medical-care specialists, inpatient and outpatient hospitalizations, and death records. An important advantage of our setting is the combination of large-scale objective health information with the ability to link across different circles of one's family and social network. Exploiting these different layers of family and social connections further enables us to investigate the scope of health behavior spillovers within one's network.

Put together, our findings identify intra- and inter-generational family spillovers as a prevalent causal channel through which health behaviors evolve. Spouses and adult children immediately increase their health investments and improve their health behaviors in response to shocks. We show that these effects are both economically significant and exhibit a high degree of persistence. We find that these spillovers in consumption of healthcare are far-reaching across one's network with a meaningful implied multiplier: the effects cascade to siblings, stepchildren, sons and daughters in-law, and even "close" coworkers, who exhibit responses of the same order of magnitude as spouses. Notably, we are additionally able to show that this is likely where they approach their limit, as there are no effects (precisely estimated) on "distant" coworkers with likely weaker social ties. Overall, our analysis reveals spillovers in consumption within the context of healthcare that are significant in magnitude, far-reaching, and long-lasting.

Besides the implications for the spillovers' breadth, studying the different layers of one's network allows us to probe into mechanisms, as different network members are subject to different sets of channels. We conceptualize two major classes of mechanisms by which network shocks can draw one's

attention and trigger changes in health behaviors. The first channel is potential learning and revelation of new information that pertains to one's *own* risk. This could work through an "exogenous" informational shock, as in the case of biologically-linked family members such as siblings and children, for whom the shock carries direct risk-related new information. But it could also work through "endogenous" learning, as in the case of spouses who may be prompted by the shock to gather data on their own preexisting health risks through, e.g., medical testing. The second channel we hypothesize is general salience, by which one's attention and awareness may be drawn to the health domain even when shocks are unlikely to reveal new information. Note that we state here two key potential mechanisms to fix ideas, guided by the literature (ex-ante) and the empirical patterns (ex-post). But, of course, there are potentially many other alternative mechanisms that could be at play. Also, a variety of micro-founded channels could be captured by the second channel of salience, including changes in individuals' decision making processes, perceptions or subjective beliefs, and preferences (e.g., time discounting). As probing into mechanisms is secondary to our primary analysis of the main effects of shocks, their further investigation is beyond the scope of this paper, though this would be an intriguing direction for future work.

With these two channels in mind, we investigate responses to shocks by the different family layers along a variety of heterogeneity dimensions that aim to capture variation in underlying mechanisms. We do so, for example, by proxying for the degree of exposure to a shock, the scope of pre-shock knowledge of one's own health risks, and the extent to which the shock may bear new information. Employing different strategies, we find that though a variety of mechanisms seem to be at play, including learning information about one's own health, the evidence points to salience as a key operative explanation, even in cases when shocks are likely uninformative. While this salience channel appears to generally lead to heightened awareness of health, the findings suggest that agents' attention is specifically drawn to the local nature of the shock so that they take actions particular to that risk's domain.

Overview. To study how health behaviors are shaped through family spillovers, this paper is composed of two complementary parts.

Part I. In the first part of the paper, we study the effects of severe non-fatal health shocks, specifically, heart attacks and strokes, on family members' utilization of preventive care. We focus on heart attacks and strokes for two key reasons. First, this application is particularly well-suited for our empirical strategy, since cardiovascular shocks are commonly studied as sudden and severe events whose particular timing is likely unpredictable (e.g., Chandra and Staiger 2007; Doyle 2011). Cardiovascular shocks also naturally fit our research question since they are directly tied to a disease-specific risk-reducing medication, i.e., the cholesterol lowering medication statins, the consumption of which we study as our main outcome. Second, the prevalence of these health shocks and of statin consumption as preventive care render this application directly relevant for a large share of the population. Cardiovascular shocks are among the leading causes of morbidity and mortality in the developed world (WHO 2014), and statins are among the most widely prescribed and best-selling medications.[1]

[1]Cardiovascular shocks account for 1 in every 3 deaths among adults in the U.S. (800,000 annually), and every year more than 1.5 million Americans experience either a heart attack or a stroke (with estimated annual costs of roughly $320 billion). Globally, cardiovascular deaths represent a similar share of approximately 30 percent of all deaths. As for statins, this class of medications accounts for a significant share of healthcare spending. Revenues from sales of the statin Lipitor alone totaled $7 billion in 2010, Medicare spending on the single statin Crestor amounted to $2.9 billion in 2015, and the global market for statins has been estimated

We begin with intra-generational analysis of spouses, to identify family spillovers that cannot be attributed to biological channels. We find that prime-age individuals (of ages 25-55) immediately increase their consumption of statins in response to their spouse's cardiovascular shock, and that this increased take-up persists for the duration of our analysis horizon of four years. By the fourth year after the shock, spouses' statin consumption responses amount to a significant increase of 15%. Similar patterns are found even for older spouses (of ages 55-85), who we show to have much more frequent interactions with the medical system so that they are presumably more informed of their personal risk. Since, once taken, statins should be consumed indefinitely for reducing cardiovascular risk, the displayed degree of adherence in the impact on spouses' statin consumption is a key element of their response pattern. Correspondingly, the effects on statin use are accompanied by immediate responses in blood tests for cholesterol levels that determine cardiovascular risk and precede statin consumption, which exhibit a large increase of 30% in the year of the spousal shock.

To understand the nature of these spousal spillover responses, we employ different sources of variation. We first analyze response heterogeneity by spouses' underlying risk based on their predicted probability of a cardiovascular shock. We find that while even low risk spouses are induced to increase their take-up of statins as preventive care, high risk spouses are much more likely to increase their statin consumption. That is, in line with economic intuition, greater investments in health (through consumption of preventive treatments) are made by spouses whose expected returns are higher. Interestingly, however, we find no such risk gradient in spouses' "information-seeking" behavior through cholesterol testing. That is, spouses across risk types are uniformly induced to take actions related to cardiovascular risk (through data gathering), but those with higher predicted risk end up having larger statin responses in practice, consistent with a learning channel of the family shock. We provide additional evidence consistent with the learning conjecture, by showing that individuals who are more likely to learn from spousal shocks, due to more similar risk profiles, exhibit stronger statin consumption responses.

Next, we further exploit the information on cholesterol testing. The medical literature asserts that the risk information relevant for receiving cholesterol-reducing treatment is a combination of one's LDL ("bad") cholesterol levels and the predicted cardiovascular risk which is based on observables. Hence, spouses who had been tested for cholesterol levels in the pre-shock period, have already gained access to the entire own-risk information set by which their statin consumption should be governed.[2] Notably, however, we find that even in households in which spouses had access to their relevant own-risk information set prior to their partner's shock, there are similar-magnitude spillovers in spousal consumption of statins. That is, while the evidence has pointed to a likely role of family shocks in inducing learning about one's own health, it also simultaneously suggests the salience of the specific health condition as a likely channel, since responses are present even when the pertinent information is already available to the agent.

We then turn to analyze households in which adult children are present at the time of the shock, to study how parental shocks spill over to health behaviors in the next generation. We begin by analyzing

to be $20 billion annually in the last decade (Redberg and Katz 2016).

[2]This stands in contrast to first-degree biological family members whose cardiovascular risk score is a function of family medical history.

the statin consumption responses of adult biological children to parental heart attacks or strokes at different stages of the life cycle. For both younger adult children (of ages 25-40) and older children (of ages 40-65) we find large and persistent responses. We show that their increases in statin consumption are immediate and grow stronger over time, so that by the end of the analysis period they amount to 36% and 16% for younger and older children, respectively. We find comparable patterns and response magnitudes by biological siblings, who are likely subject to impact mechanisms similar to those through which biological children can be affected.

The causal spillovers to health investments by biological children can involve different routes, including revelation of information about biological risk, revelation of risk information attributed to the environment shared with the parent while growing up, as well as salience of health and increased awareness. To take a step toward isolating potential mechanisms, we proceed to study family and social circles for whom different channels may be at play. First, to abstract from the biological-risk channel, we move on to analyze potential spillovers to stepchildren. We find that the spillover in health behaviors cascades also to non-biological children and averages to an 11% increase in their consumption of statins, an effect that is half as large as that on biological children. To additionally abstract from the spillover channel by which children share the same environment as their parents in childhood or adolescence, we study the potential spillovers to sons and daughters in-law. We show that following a cardiovascular shock, children in-law exhibit an average increase in their consumption of statins that amounts to about a quarter of that of their spouses, i.e., the biological children. Interestingly, consistent with exposure intensity and salience of family shocks, we find that these effects on sons and daughters in-law are entirely driven by those who live closer to their in-laws. Lastly, we move on to study individuals who are connected only through social ties. To proxy for one's peers, we use matched employer-employee data to construct a sample of coworkers. We find that "close" coworkers, as measured by similarity of ages and occupations, exhibit prompt responses with spillover patterns that are very similar in their dynamics and magnitude to that of prime-age spouses.

Together, the estimated effects on multiple circles of the family and the social network underscore the far reach of spillovers in health investments and behaviors. We find that the spillovers of health shocks to the consumption of preventive care by family members and peers are immediate, economically meaningful, and display a high degree of adherence, which points to a role of one's network in forming health habits. To further gauge the economic importance of the estimated spillover effects, we illustrate that they account for significant shares of individuals' evolution of health behaviors over time. Specifically, within our studied time horizon (of nine years), we show that a single shock within one's network explains 28%, 15%, and 12% of the life-cycle growth in consumption of preventive care for younger adult children, prime-age spouses, and peers, respectively. We also show that these responses can potentially close a meaningful portion of the underlying gap in preventive care utilization.

Part II. Following our investigation of the scope and nature of spillovers by studying non-fatal health shocks, we then turn to the second complementary part of the paper, in which we study the effects of fatal family shocks. The advantages of doing so are the opportunity and power to explore a variety of utilization margins indicative of changes in behaviors (where to this point we focus on responses particular to cardiovascular risk); as well as the ability to test additional mechanism-related hypotheses by leveraging detailed codes of causes of death. In this part we focus on spouses with the

aim to identify spillovers that cannot be explained by biological channels as before; as well as to gain increased power that allows for clear estimation of dynamics for any given level of relative response (since spouses exhibit higher baseline levels of healthcare utilization compared to their children due to age).[3] The analysis proceeds in two steps.

In the first step, we study the effects of fatal shocks on family members' general awareness of health and common health-related behaviors. Studying hospitalizations for suspected conditions that are ruled out upon examination and urgent-care doctor visits as proxies for the degree to which individuals pay attention to health, we find that family members experience a period of several years in which they exhibit heightened awareness regarding their own health following the family shock. Likewise, we find significant behavioral improvements in the form of decreases in harmful health-related behaviors: smoking, excessive alcohol consumption, and medication abuse. Specifically, exploiting the prescription drug data, we find that family members immediately engage in consuming medications that are prescribed for smoking or drinking cessation. We also find that spouses reduce their consumption of addictive harmful medication, by studying the consumption of prescription opioids, which account for the greatest proportion of mortality cases linked to prescription drug abuse (Volkow 2014; Rudd et al. 2016). Notably, these behavioral responses are present even in cases when the shock does not bear new information or knowledge regarding the risk involved in these behaviors.

Unlike the non-fatal shocks we analyze, fatal spousal shocks can have direct health effects.[4] To take a step toward isolating behavioral responses from health effects in the investigation of spousal death, we proceed by analyzing only treated households who experienced a death shock and by focusing on variation in the condition responsible for this death. The basic idea is that all individuals are faced with the main effects of spousal death, but their experience may differ by the exact cause of death. Among different alternative explanations, this analysis reduces the plausibility that potential complementarities between treatment and preventive care may explain the findings of the effects of fatal shocks on spousal health behaviors. Hence, in the second step of this part of the paper, we exploit variation in specific causes of death to study consumption of different condition-specific preventive care, both within and across conditions. This also allows us to explore the directionality of responses and the degree to which they may be local to the domain of the experienced shock.

We do so in the context of two main classes of preventive-care practices that are tied to the two leading causes of death in the developed world. These are cardiovascular disease, for which we study statin consumption as before; and cancer, for which we study expenditure on diagnostic radiologists, who specialize in disease diagnosis and are responsible for screening patients for major types of cancer. Bolstering our findings from non-fatal cardiovascular shocks, we show that, following a fatal shock, individuals whose spouse died of cardiovascular disease persistently consume statins at a higher rate compared to those whose spouse died of any other cause. In the same vein, we find that in the year just after spousal death of cancer, individuals' healthcare expenditure on diagnostic radiology significantly increases compared to spousal death of any other cause. Importantly, we find similar responses in

[3] Nonetheless, we also replicate the main results for the average effects on adult children.

[4] Previous literature and our own investigation within the current application have found immediate direct effects of fatal shocks on *spouses*' physical health (specifically, by analyzing hospital contacts). This is in contrast to non-fatal spousal cardiovascular shocks, as well as fatal or non-fatal parental shocks, where we find no evidence of such effects on family members.

the case of husbands whose wives die of female cancers, where the spouse's cancer type is not likely predictive of own cancer risk (which we verify in the data).

Finally, we study cross-condition responses which show that behavioral changes are directed to the vicinity of the experienced shock. We find no differential expenditures on diagnostic radiology by individuals whose spouse died of cardiovascular disease. In the context of fatal cancer, we even find declines in spouses' consumption of preventive care against cardiovascular disease. Consistent with the notion of limited attention, this result raises the possibility of a crowd out channel. Namely, it is not only that individuals increase their take-up of condition-specific care, even in cases that are likely uninformative, but they may also reduce their take-up of preventive care that pertains to other, non-salient health risks.

These results on the impact of fatal health events strengthen our first set of findings, by showing that family shocks increase individuals' awareness of their own health and induce them to engage in efforts to cease unhealthy behaviors in the context of key harmful habits. Moreover, they reveal that the behavioral and consumption responses are governed by targeting the particular experienced risk. As the findings prevail when likely no information on own risk has been revealed, the results further bolster the hypothesis of salience and attention as operative channels in explaining family members' behavioral responses to shocks.

Implications. Altogether, our findings of significant spillovers in health behaviors and consumption of preventive care have implications for modeling and analyzing health investments and healthcare demand. The estimated impacts of shocks on different generations of family and peers highlight the importance of incorporating inter-personal interactions into these models. That is, similar to applications such as the sharing of resources and labor supply choices, the results suggest we should study health behaviors, investments, and the consumption of healthcare at the family level, and even at the level of one's social network. The pattern of responses and the suggestive evidence on likely channels could be further informative of the aspects such models should appropriately include. They highlight learning within one's network and, specifically, salience and attention as key components (as in, e.g., DellaVigna 2009),[5] and they also point to event-driven responses, in line with frameworks such as Bernheim and Rangel's (2004) cue-triggered decision making model. More generally, the results can also advance our understanding of the nature and scope of broader network effects in consumption, here through the lens of healthcare utilization.[6]

Lastly, the findings could also have implications for policies that aim to promote population health. The results emphasize that health behaviors are not immutable and suggest the leveraging of family events as a window of opportunity for intervention, as they seem to involve the required intrinsic

[5] As such, our work is consistent with recent findings from interventions in developing economies. In their review of randomized evaluations that involve peer influences on health behaviors, Kremer and Glennerster (2011) argue that while some of the findings are potentially driven by learning, others likely reflect alternative channels such as salience, as in the case of the work by Zwane et al. (2011) who find that the act of being surveyed affects behavior. Similarly, our work is also related to the growing interest in and evidence of how limited attention and salience affect economic behavior in a variety of settings; see DellaVigna (2009) for an earlier review of this work.

[6] De Giorgi, Frederiksen, and Pistaferri (2016) provide a brief review of the work on consumption within the network and study consumption network effects (analyzing total spending) by exploiting interactions between one's household and coworker networks.

motives for behavioral changes that induce persistent responses. Relating to our findings on likely channels, one could exploit family shocks by formulating strategies that provide family members with individual-specific information on risks; or by introducing policies that leverage salience of health to actively offer preventive care, e.g., by "defaulting" family members into checkups or basic treatment regimens. However, the evidence also suggests more broadly that leveraging salience as a policy tool should be done with caution. As individuals' attention can be drawn to specific risks even in the absence of relevant information, their responses could end in excessive consumption of preventive care targeted to that risk; and, at the same time, their attention might be diverted away from conditions of which they may be at higher risk.

The remainder of the paper is organized as follows. The next section discusses related literature. In Section 3 we describe the empirical strategy that we employ for estimating the effects of adverse health shocks on family and network members' consumption of preventive care and health-related behaviors. Section 4 outlines the data sources we use, the analysis sample, and the institutional environment. The empirical evidence on spillovers in health behaviors is presented in Section 5. Specifically, in Section 5.1, we study the effects of severe non-fatal health shocks on preventive care utilization by family members and peers; and in Section 5.2, we study family spillovers in the context of fatal shocks. Lastly, Section 6 discusses the implications of our findings and concludes.

2 Related Literature

In addition to the studies we have mentioned so far, our work relates and contributes to several strands of the literature in economics and in health research. First, within the broader literature that studies behaviors and choices in the framework of the family and social networks (see reviews in, e.g., Browning et al. 2014 and Sacerdote 2014), there is some particular work in the context of health behaviors. A series of papers has documented strong correlations among peers and family members, mostly across spouses, in a variety of health-related behaviors, such as alcohol consumption, smoking, obesity, and preventive medical care.[7]

More recent work offers evidence for network effects in health behaviors, studying a range of outcomes, including weight, smoking, drinking, and substance use. In the context of the family, most relevant to our work, Cutler and Glaeser (2010) have exploited smoking bans at the workplace to study spousal smoking behavior; Christopoulou et al. (2013) have instrumented for parental smoking using exposure to information on health risks (based on magazine articles) to study impacts on children's smoking; Cawley et al. (2017) have studied effects across full siblings in weight and obesity by instrumenting for a sibling's obesity using relevant genetic risk scores; and Fletcher and Marksteiner (2017) have recently found spillovers to spouses' behavior in clinical interventions to reduce individuals' smoking and alcohol consumption. In the context of social ties, studies have analyzed the role of roommates, friends, schoolmates, communities, and neighborhoods by employing different sources of variation.[8] Other papers closely relevant to ours have studied quality of care and health behaviors

[7] These include Franks et al. (2002), Leonard and Mudar (2004), Clark and Etilé (2006), Christakis and Fowler (2007), Cohen-Cole and Fletcher (2008), Falba and Sindelar (2008), Rosenquist et al. (2010), Banks et al. (2013), McGeary (2013), and Cobb et al. (2014). Meyler et al. (2007) offer a review.

[8] For example, papers have exploited variation driven by random roommate or squadron assignments (Carrell et al. 2011;

following bereavement, mainly to test whether they can account for declines in spousal health.[9]

More generally, there is a large literature on a range of other determinants of individuals' health behaviors, which analyzes the impact of, e.g., commitment mechanisms and financial incentives, health insurance coverage, medical information, disease outbreaks, doctor practices, government or workplace policies, and advertisements and the media. Cawley and Ruhm (2011) offer a review of this work and we also list more recent papers within the context of developed economies in Appendix Table 6. Kremer and Glennerster (2011) review evidence from randomized evaluations in developing countries.

3 Research Design

The goal of our empirical analysis is to identify the dynamic causal effects of severe health shocks on family members' consumption of preventive care and health-related behavioral changes. In this section, we describe the empirical strategy that we use to overcome the selection challenges inherent in the identification of these effects and state our estimating equation. We also outline the specifications we employ in the different empirical exercises that analyze heterogeneity in treatment effects, which aim to shed light on the nature of spillovers and their underlying mechanisms.

3.1 Primary Quasi-Experiment

The ideal experiment for identifying the short- and medium-run effects of health shocks would randomly assign shocks to families and track responses in health behaviors over time. Therefore, we need to compare the ex-post responses to shocks of affected households to a counterfactual behavior of ex-ante similar unaffected households. This requires comparing households with similar expectations over the distribution of future paths, but with different realizations, to isolate the unanticipated component of the shock. The access to three decades of administrative panel data on the universe of Danish households allows us to utilize a quasi-experimental research design that mimics this ideal experiment, by exploiting the potential randomness of the timing of a severe health shock within a short period of time.

To do so, we look only at households that have experienced the shocks that we consider at some point in our sample period, and identify the treatment effect from the timing at which the shock was realized. Specifically, we construct counterfactuals to affected households using households that experience the same shock but a few years in the future. As such, our two experimental groups consist of a treatment group, composed of family members in households that experience a shock in year τ, and a matched control group, composed of family members from the *same* cohorts in households that

Eisenberg et al. 2014; Yakusheva et al. 2014), the Moving to Opportunity experiment (Kling et al. 2007; Ludwig et al. 2011), friends' treatment assignment in field experiments (Babcock and Hartman 2010), and instruments such as friends' early life circumstances or their parents' conditions (Argys and Rees 2008; Renna et al. 2008; Trogdon et al. 2008), or have estimated different fixed effects and lagged behavior models (Lundborg 2006; Clark and Loheac 2007). Kremer and Glennerster (2011) discuss a number of examples for randomized evaluations in developing countries in which health behaviors were influenced by peers.

[9] Examples include Zisook et al. (1990), Rosenbloom and Whittington (1993), Jin and Christakis (2009), Shah et al. (2013), and Simeonova (2013). These papers generally compare outcomes before and after bereavement or around the event and make comparisons between widowed and married individuals. Using these comparisons they mostly find no changes or some declines in quality of care or in healthy behaviors, where an exception is Jin and Christakis (2009) who find trends of declines just before and increases just after bereavement (e.g., in mammograms). See also a review in Stahl and Schulz (2014). Relatedly, Khwaja et al. (2006) study associations between smoking and spousal health in the Health and Retirement Study.

experience the same shock but in year $\tau + \triangle$. We then recover the treatment effect by performing traditional event studies for these two experimental groups and combining them into a straightforward dynamic difference-in-differences estimator. That is, we identify the treatment effect purely from the change in the differences in outcomes across the two groups over time.

The trade-off in the choice of $\triangle$, which captures the main limitation of the design, weighs comparability against analysis horizon. On the one hand, we would want to choose a smaller $\triangle$ such that the control group is more closely comparable to the treatment group, e.g., those who experienced the shock a year apart which corresponds to $\triangle = 1$. On the other hand, we would want to choose a larger $\triangle$ in order to be able to identify longer-run effects of the shock, since for each chosen $\triangle$ the estimation strategy provides estimates for up to period $\triangle - 1$. For example, using those who experienced a shock 10 years apart ($\triangle = 10$) will allow us to estimate the effect of the shock for up to 9 years. However, this entails a potentially larger bias since the pre-trend in the behavior of this group might not be tightly parallel to that of the treatment group. Our choice of $\triangle$ is five years, such that we can identify effects up to four years after the shock. We assessed the robustness of our analysis to this choice and found that local perturbations to $\triangle$ provide very similar results.

The identifying assumption is that, absent the realization of the shock, the outcomes of the treatment and control groups would run parallel. The plausibility of this assumption relies on the notion that within the window of time of length $\triangle$ the particular year at which the shock occurs may be as good as random. To test the validity of our assumption, we accompany our empirical analysis with the treatment and control groups' behavior in the four years prior to the shock year (period 0) in order to assess their co-movement in the pre-shock periods. We consistently show throughout the analysis that there are virtually no differential changes in the trends of the treatment and control groups before period 0. This validates the design and alleviates concerns that the groups may differ by, for example, their expectations over the *particular* year of the shock within our chosen five-year window of $\triangle$.

The remainder of this subsection formalizes the research design and provides a formal description of our estimators and econometric models.

Formal Description of the Design and Estimator. Similar to common practice (for example, in the use of matching estimators; see, e.g., Imbens and Wooldridge 2009), our estimation procedure can be broken down into two steps. The first step constructs our treatment and control groups and, in the second step, estimation and inference are conducted using traditional methods. We describe the two steps successively.

Fix a group of cohorts, denoted by Ω, and consider estimating the treatment effect of a shock experienced at some point in the time interval $[\tau_1, \tau_2]$ by individuals whose family members belong to group Ω. We refer to these individuals' family members as the treatment group and divide them into sub-groups indexed by the year in which the shock was experienced, $\tau \in [\tau_1, \tau_2]$. We normalize the time of observation such that the time period, t, is measured with respect to the year of the shock—that is, $t = year - \tau$, where *year* is the calendar year of the observation. As a control group, using only timing, we match to each treated group τ the family members from the same cohort group Ω of individuals who experienced the same shock but at $\tau + \triangle$, for a given choice of $\triangle$. For these households we assign a "placebo" shock at $t = 0$ by normalizing time in the same way that we do for the treatment group,

i.e., $t = year - \tau$ (where, by construction, their actual shock occurs at $t = \triangle$).[10]

Denote the mean outcome of the treatment group at time t by y_t^T and the mean outcome of the control group at time t by y_t^C, and choose a baseline period prior to the shock (e.g., period $t = -1$) which we denote by p (for "prior"). For any period $r > 0$, the treatment effect δ_r can be simply recovered by the difference-in-differences estimator

$$\delta_r \equiv \left(y_r^T - y_r^C\right) - \left(y_p^T - y_p^C\right). \tag{1}$$

The treatment effect in period r is measured by the difference in outcomes between the treatment group and control group at time r, purged of the difference in their outcomes at the baseline period p. Note that the choice of $\triangle$ puts an upper bound on r such that $r < \triangle$ (since the control group becomes "treated" at $t = \triangle$).

Estimating Equation. To study the evolution of household responses, we estimate the regression counterpart of the dynamic differences-in-differences estimator of equation (1). The regression specification allows the inclusion of controls which increases precision, and further balances the treatment and control groups in a systematic way in cases where the studied utilization codes appear in different calendar years (due to institutional changes in reporting). For visualizing our empirical strategy, we also provide within our initial set of results a graphical analysis of the raw data. We do so in the analysis of spouses and biological children, for whom ties are strongest and samples are largest. Our main regression specification is of the form:

$$y_{i,t} = \alpha + \beta treat_i + \sum_{r \neq -1; r=-4}^{4} \gamma_r \times I_r + \sum_{r \neq -1; r=-4}^{4} \delta_r \times I_r \times treat_i + \lambda X_{i,t} + \varepsilon_{i,t}. \tag{2}$$

In this regression, $y_{i,t}$ denotes an outcome for household i at time t; $treat_i$ denotes an indicator for whether a household belongs to the treatment group; I_r are indicators for time relative to the assigned shock year (actual shock for treatment and placebo shock for control); and $X_{i,t}$ denotes a vector of potential controls. The key parameters of interest are δ_r, which estimate the period r treatment effect ($r > 0$) relative to the pre-period $p = -1$.[11] Note that validation of the parallel trends assumption requires that $\delta_r = 0$ for all $r < 0$. Unless we indicate otherwise, we include in $X_{i,t}$ age fixed effects, calendar year fixed effects, gender, and education, and report robust standard errors clustered at the household by experimental-group level.

To quantify mean treatment effects, we estimate the standard difference-in-differences equation of the following form which averages over years before and after the shock:

$$y_{i,t} = \alpha + \beta treat_i + \gamma post_{i,t} + \delta treat_i \times post_{i,t} + \lambda X_{i,t} + \varepsilon_{i,t}. \tag{3}$$

In this regression, $post_{i,t}$ denotes an indicator for whether the observation belongs to post-shock periods. The parameter δ represents the average causal effect of shocks on family members' outcomes.

[10] The same household can appear both in the treatment group and in the control group, but is never used as a control to itself. For example, if treated households that experienced a shock in 1990 (who are matched with households that experienced a shock in 1995 as controls) are included also in the control group, it is only since households that experience a shock in 1985 are included in the treatment group as well. We repeated our main analysis using treatment and control groups that do not overlap, either by including in the treatment group (and matching them with the corresponding control group) households that experience shocks in every other year, or by randomizing overlapping households to only one experimental group. The results remain similar (both qualitatively and quantitatively) and are available from the authors on request.

[11] With no controls and with the same choice of pre-period p, the δ_r estimates of the dynamic treatment effect for $r > 0$ from (2) are mathematically identical to those from (1).

3.2 Response Heterogeneity

There are two related strategies that we employ when analyzing the nature or sources of the main treatment effects.

Heterogeneity Specification 1. We use the first strategy when we are interested in estimating a mean baseline effect and how it varies by some dimension or household characteristic of interest, z_i (which can be a vector). This regression simply augments the baseline difference-in-differences model of equation (3) in the following way:

$$y_{i,t} = \alpha + \beta treat_i + \gamma post_{i,t} + \delta_i treat_i \times post_{i,t} + \lambda X_{i,t} + \varepsilon_{i,t}, \tag{4}$$

where

$$\delta_i = \delta_0 + \delta_1 z_i.$$

We adjust the basic difference-in-differences design by allowing the treatment effect, δ_i, to vary across households and model it as a function of the household's characteristic z_i. Our parameter of interest is δ_1, which captures the extent to which the family member's response correlates with z_i.[12]

Heterogeneity Specification 2. We use the second strategy when we are interested in directly studying how responses vary by some dimension or household characteristic, z_i, among treated households only. Exploiting variation within the treatment group, the regression that follows the dynamics of the heterogeneous responses around the event is of the following form:

$$y_{i,t} = \alpha + \beta z_i + \sum_{r \neq -1; r=-4}^{4} \gamma_r \times I_r + \sum_{r \neq -1; r=-4}^{4} \delta_r \times I_r \times z_i + \lambda X_{i,t} + \varepsilon_{i,t}, \tag{5}$$

Equation (5) is similar to equation (2) but where $treat_i$ is replaced with z_i, so that the treatment and control groups are now defined as households with different values of z_i within the group of treated households. Consequentially, the same identifying assumption is required for these experimental groups, i.e., that absent the shock the outcome of households with different values of z_i would run parallel. As before, an implied necessary condition is that $\delta_r = 0$ for $r < 0$. Similar to equation (2), the key parameters of interest are δ_r for $r > 0$, which estimate how the outcomes for treated households with varying levels of z_i differentially evolve around the event relative to the omitted time category (period -1).[13] Lastly, the corresponding equation that estimates how responses vary on average across treated households with different levels of z_i takes the form:

$$y_{i,t} = \alpha + \beta z_i + \gamma post_{i,t} + \delta \times z_i \times post_{i,t} + \lambda X_{i,t} + \varepsilon_{i,t}. \tag{6}$$

4 Data and Institutional Background

In this section we describe our data sources and analysis sample, and we also provide institutional background relevant for our work. The Danish setting that we study is a particularly well-suited environment for identifying family spillovers in health behaviors in the context of developed economies.

[12] In the estimation of (4) we always include in the vector $X_{i,t}$ the variables in z_i as well as their interaction with $treat_i$ and $post_{i,t}$.

[13] Validation of parallel trends would lend support to our ability to estimate response heterogeneity across households with different values of the characteristic z_i (using either equation (4) or (5)). However, it is important to note that it would not directly imply, of course, that we identify a causal effect of that characteristic.

First, it provides us with the required long panel of detailed administrative healthcare records for linked family members. The exact utilization codes included in the data allow us to identify health investments and behavior proxies, and the large scale provides sufficient statistical power for studying different utilization outcomes. Second, a key institutional feature of the Danish healthcare system is the provision of near-complete and universal healthcare coverage. Importantly, this enables us to identify effects that are not confounded by the availability of health insurance, similar to, e.g., analyzing the Medicare population in the U.S..

4.1 Data Sources

Studying adult family members' health-related behavioral changes in response to severe health events requires information on healthcare utilization outcomes, as well as on health shocks, for the different members of a household. We therefore combine several administrative Danish registers that include individual-level records with family linkages from 1980 to 2011, which allow us to identify all families of married and cohabiting couples and their adult children's households, as well as other circles of one's family and social network.

For utilization outcomes indicative of health behaviors, our data consist of three databases that encompass both primary and secondary healthcare utilization records with exact dates and codes. These include: (1) the *Prescription Drug Database*, covering all prescribed drugs that were purchased from 1995-2011 (where 90% of all medications are subject to prescriptions in Denmark), with detailed information on doses prescribed and medication classification (using the Anatomical Therapeutic Chemical [ATC] classification system); (2) the *Health Insurance Registry*, covering all individual contacts with primary-care physicians and medical-care specialists outside of hospitals from 1985-2011; and (3) the *National Patient Registry*, covering all inpatient hospitalizations (from 1980-2011) and outpatient hospitalizations (from 1994-2011), in both private and public hospitals, with detailed diagnoses (using the International Statistical Classification of Diseases and Related Health Problems [ICD] system). The specific outcomes that we study are described within the empirical analysis in Section 5.

For identifying fatal and non-fatal severe health shocks, we use the latter dataset (the *National Patient Registry*), which allows us to identify severe health shocks and their exact timing, accompanied by the *Cause of Death Registry*, which includes exact death dates and specific causes from 1980 onward. Lastly, we extract demographic variables such as gender, age, and level of education from the *Integrated Database for Labor Market Research*. This dataset also includes register-based matches across employers and employees, from which we construct coworker linkages to proxy for peers.

All monetary values of healthcare expenditure are reported in nominal Danish Kroner (DKK) deflated to 2000 prices using the consumer price index. In that year the exchange rate was approximately DKK 8 per US $1.

4.2 Analysis Sample

To construct our sample, we start from the universe of households in which an individual experienced one of the shocks that we consider between the years 1985 and 2011, where all of our matches across household members are based on the pre-shock period $t = -1$. Our primary sample of non-fatal health shocks is comprised of all households in which one individual experienced a heart attack or a

stroke (for the first time) and survived for the four-year analysis horizon. The main family circles that we study are spouses and adult biological children. Our sample of spouses is based on all married and cohabiting couples among families in which one spouse experienced a shock. The registers provide such spousal matches across all individuals born between 1910 and 1970, who are the cohorts covered by our data. For children, the registers provide matches to biological parents for individuals born after 1960. Our sample of adult biological children is based on these matches. Naturally, the age composition of spouses and adult children substantially differ, so that the age splits in our analysis (which show effects for younger and older individuals within each sub-sample) will be guided by the data and will differ accordingly across these groups. In a supplementary analysis we also investigate the effects of shocks on siblings. This sample is based on parental linkages, such that siblings are defined as individuals who share biological parents.

For the more distant circles of family members and peers, we increase the statistical power by reducing the data requirement to include individuals who survived for at least three years after the cardiovascular shock (instead of four). This strengthens the ability to look at dynamics, not only at averages, in cases where samples become naturally much thinner (as in the case of stepchildren), or when the potential effects, if at all present, are expected to be smaller (as in the case of sons and daughters in-law). Doing so implies that we can only investigate the three-year dynamic causal effects but enables us to significantly increase sample sizes due to fatality rates of cardiovascular shocks. For example, the sample increases by 20 percent in the stepchildren application.[14]

We construct these additional samples as follows. Stepchildren are defined as any child with a non-biological link to the individual that experienced the shock. We establish these links by combining the spousal linkages and the biological parent linkages. Specifically, we define as a "stepchild" any person for whom neither biological parent is the individual that experienced the shock but for whom one biological parent is the spouse of that individual. Sons and daughters in-law (to whom we collectively refer as "children in-law") are simply the spouses of the biological children. Finally, we proxy for peers using coworkers based on matched employer-employee register data, where we define workplaces using physical establishment units. To approximate peers with whom individuals are more likely to interact, we focus on "close" coworkers in the following way.[15] From our sample of individuals who experience a health shock we identify those who, during the pre-shock periods from $t = -4$ to $t = -1$, have worked in smaller workplaces (with number of employees equal or lower than the sample's 25th percentile of approximately 20), in order to reduce the measurement error in proxying for peers using coworkers. We then study the effects on coworkers who have been employed in a similar occupation class,[16] and who are close to these individuals in terms of age (with an age gap of 5 years or less).[17] It is important

[14] More specifically, our choice of the degree to which power would increase, determined by the number of required years of survival, was governed by balancing between the decrease in the analysis horizon and the rate of change in standard errors as sample sizes increase. As benchmarks, the shrinkage in standard errors was compared to the impact magnitudes of spouses and children. For example, conditioning on three years of survival enables identification of mean effects on stepchildren that are at least 48% of that on children.

[15] This is in the spirit of definitions used in De Giorgi, Frederiksen, and Pistaferri (2016).

[16] For occupation classes we follow the official classification method of Statistics Denmark that is constructed based on the International Standard Classification of Occupations (ISCO). This method classifies employees into managers and non-managers and, among non-managers, it further classifies employees into occupations by their required skill level (low/medium/high).

[17] Estimations that perturb the thresholds of workplace size and age gap, which we chose to balance between sample size and

to note that we exclude from this sample any coworker who is also a family member.

Lastly, our secondary sample of fatal shocks includes all families in which one member died between 1985 and 2011. For these shocks we study spouses and biological children, whose respective samples are constructed in the same way as before. Appendix Table 1 summarizes the various analysis samples for each shock and for each network circle that we analyze and reports summary statistics.

4.3 Institutional Details

Overall, health insurance in Denmark is a universal scheme in which almost all costs are covered by the government. The few exceptions that entail a limited degree of out-of-pocket expenses include medical services provided by dentists, physiotherapists, psychologists, and chiropractors, as well as prescription drug co-insurance payments for prescriptions outside of hospitals as we describe below. The provision of public health insurance in Denmark is decentralized to the local government, specifically regions, that engage in common agreements with primary-care professionals and with non-hospital medical specialists and also fund public hospitals in the secondary healthcare sector. We describe the primary-care and the secondary-care sectors successively.

Primary Care. The main providers in the primary-care sector are the general practitioners (GPs), who act as gatekeepers to the healthcare system, e.g., in terms of referring patients to hospitals and specialists. GPs are organized in private self-employed businesses and are reimbursed according to a fee-for-service schedule. The union of general practitioners (*Praktiserende lægers organization*) and the regional administration (*Regionernes Lønnings- og Takstnævn*) negotiate the annual fees for specific services, which are funded by regional and state taxes.

Each patient is assigned one GP, whose main responsibilities include medical consultations, non-specialized treatments, and provision of preventive care. For doing so, GPs are eligible to prescribe drugs for both treatment and prevention purposes. Patients pay no out-of-pocket costs for standard services provided by the GP, but there is some degree of co-insurance payments for medication prescribed by GPs. Specifically, until March 2000 patients paid 50% of pharmacy sale prices, with a reduced rate of 25% for drugs that treat life-threatening or chronic conditions. In March 2000 the payment scheme introduced a deductible with decreasing marginal co-insurance rates beyond the deductible amount. For annual expenses on prescription drugs up to DKK 865 (in 2012 rates as an example) patients would pay the full amount; and for additional expenses patients would pay 50% in the range of DKK 865-1,410, 25% in the range of DKK 1,410-3,045, and 15% for expenses over DKK 3,045.[18]

Secondary Care. The main entities in secondary care are public hospitals, to which patients are referred either by their GP or following visits to emergency rooms. Public hospitals operate as independent units with own budgets funded via taxes by the regional government. Until the late 1990s hospitals were entirely funded by block grants and fee-for-service reimbursement schedules. From 1999, however, inspired by the American healthcare system, the funding gradually switched toward a scheme based on Diagnosis-Related Groups (DRGs). Within this scheme each patient's case is categorized into a DRG, and each DRG has prospectively set payment rates based on the average resources used to

"closeness" of peers, are provided in Appendix Table 3 for robustness.

[18] Additionally, there are annual caps for the chronically ill (so that, e.g., in 2012 patients were fully reimbursed for expenses above DKK 3,555), and retirees can apply for means-tested reimbursements from the municipality.

treat patients in that DRG. Initially, 10% of hospitals' budgets were funded through the DRG system. This share increased to 20% in 2004 and is today between 50% and 70% depending on the region.

The main challenge within the hospital sector in recent decades has been long waiting lists for specialized treatments, which through the 1990s was addressed by increasing the degree of flexibility in individuals' hospital choice. Specifically, in the early 1990s patients were offered flexible hospital choice within regions, which was later extended to flexible choice nationally, and was finally expanded to the eligibility to choose private hospitals in case there were no availabilities in public hospitals. However, private hospitals account for only 2.5% of all hospital beds in the secondary sector and provide only very specialized services. Note that visits to private hospitals that are not due to public hospitals' unavailability are paid out-of-pocket on a fee-for-service basis. Still, patients rarely pay the full amount of these expenses, as many are covered by supplementary private insurance through their employers (who have tax incentives to provide these policies).

5 Spillovers in Health Behaviors

We now turn to our analysis of how health behaviors are shaped through family spillovers, which has two complementary parts. In the first part, we study the effects of severe non-fatal health shocks, specifically, heart attacks and strokes, on family members' consumption of preventive care that is specific to these cardiovascular shocks. The analysis is conducted at two main levels for which different potential mechanisms can be at play: intra-generational analysis of the effects on spouses and inter-generational analysis of the effects on the next generation of adult children. To further study the breadth of shock spillovers, we analyze whether and to what degree the effects cascade to siblings, to the children's households (by studying sons and daughters in-law), to stepchildren, and even to coworkers. In the second part of the analysis, we study the effects of general fatal shocks on family members. In this part, we begin by analyzing overall changes in consumption of healthcare indicative of behaviors and awareness, and then proceed to studying utilization of condition-specific preventive-care by exploiting variation in causes of death. Preceding each of the two parts, we describe the motivation for the analysis that follows, and we then provide the corresponding spillover results. We focus on reporting the estimated effects in relative terms compared to the counterfactual in order to account for the baseline prevalence in the population of each healthcare utilization component that we analyze.

Recall that the goal of our empirical analysis is to identify the dynamic causal impacts of severe health shocks on the consumption of preventive care and health-related behaviors by family members (and peers). In addition, to offer an investigation of the nature and potential sources of these main effects, we examine different dimensions of heterogeneity in responses as well as a variety of outcomes throughout both parts of the empirical analysis. To reiterate the framework we have in mind, we conceptualize two main classes of routes for the spillover effects: learning and salience. For first-degree biological relatives, there could be direct learning regarding one's own health since the shock carries new genetics-related information. For non-biological family members and peers, there is a potential indirect learning route. Individuals can learn new general information regarding the medical condition their family member or peer has experienced, e.g., through interactions with him or her or with the medical system when accompanying the sick. They can also learn new specific information regarding

their own risk if the shock triggers them to engage in data gathering through, e.g., medical testing. The second channel we have in mind is responses to shocks due to the salience of the particular health shock to which individuals' attention can be drawn, even when shocks are unlikely to be directly informative of one's own health risk or to lead to revelation of new information through learning.

5.1 Part I: Effects of Severe Non-Fatal Health Shocks

To study how health shocks spill over to family members' health behaviors and consumption of preventive care, we focus on heart attacks and strokes. These shocks are directly tied to a disease-specific risk-reducing medication, so we focus on its consumption as a main behavioral outcome. This class of medication, called statins, is composed of prescription drugs taken to lower cholesterol that are pervasively used to prevent cardiovascular disease. We therefore proceed with analyzing how individuals' cardiovascular shocks affect the consumption of statins by members of their network.[19]

5.1.1 Effect on Spouses

We begin with an intra-generational analysis of spouses, to identify family spillovers that are not attributable to biological channels. To visualize the estimation strategy, we provide in this subsection figures that plot the raw data, and we then proceed to regression estimations which quantify the effects. Panel A of Figure 1 plots the average responses in statin consumption by prime-age spouses, of ages 25-55, in our sample of heart attacks and strokes. The structure of this and subsequent figures is as follows. The x-axis denotes time with respect to the shock, normalized to period 0. For the treatment group, period 0 is when the actual shock occurs; for the control group period 0 is when a "placebo" shock occurs (while their actual shock occurs in period 5). The dashed gray line plots the behavior of the control group. To ease the comparison of trends, from which the treatment effect is identified, we normalize the level of the control group's outcome to the pre-shock level of the treatment group's outcome (in period $t = -1$). This normalized counterfactual is displayed by the blue line and squares. The red line and circles plot the behavior of the treatment group.

Panel A of Figure 1 first provides a visual verification of parallel trends across the treatment and control groups prior to period 0, as required by the design. Then, analyzing the effect of the shock, the figure reveals that prime-age individuals immediately increase their consumption of statins in response to their spouse's cardiovascular shock, and that this increased take-up persists for at least four years after the shock. Note that, once taken, this medication should be consumed indefinitely for the purpose of reducing the risk of experiencing a heart attack or stroke, so the displayed degree of adherence is a key element of the response pattern. Column 1 of Table 1 estimates the corresponding regression using equation (2) and shows that the treatment effect grows over time, so that by the fourth year after the shock the increase in spouses' statin consumption amounts to 15%—an increase of 1.17 percentage points (pp) on a base of 7.86 pp.

Older spouses have much more frequent interactions with the medical system, including routine checkups, which are more common as individuals age (see Appendix Figure 1). As the main risk factors

[19] It is important to note that the effects on family members' consumption of statins are prevention related, and hence take the form of health investments, as these responses are not driven by managing own conditions that might have been induced by the shock. Specifically, there is no increase in the incidence of cardiovascular disease (as well as in hospital contacts of any type) among spouses or children following the non-fatal cardiovascular shocks that we analyze.

of cardiovascular disease (beyond age and gender) are commonly screened for by individuals' primary-care providers—including hypertension, cholesterol levels, and diabetes—older spouses are presumably already more informed of their personal risk. Still, even for spouses between the ages of 55 and 85 we find that similar-magnitude spillovers are present, albeit on a larger baseline as statin consumption rates are higher for this group due to their older age. See column 2 of Table 1 (and Appendix Figure 2) for these results.

For a subset of our sample, specifically those who reside in Greater Copenhagen, the data also consist of indicators for blood tests of cholesterol levels. As these tests are used to determine cardiovascular risk and precede statin consumption, their patterns can shed additional light on the dynamics of spousal responses. Panel B of Figure 1 and column 3 of Table 1 report the effects of cardiovascular shocks on spousal cholesterol testing. Promptly at the year of the shock, spouses respond with a large increase in their rate of cholesterol testing which amounts to about 30%. This response is consistent with the corresponding increase in their statin take-up within the immediate years following the shock. Compared to the counterfactual, cholesterol testing remains somewhat higher also in the following years. This is likely due to continuous monitoring tests that are common in medication maintenance for those who have started consuming statins, and it can also mirror the growing post-shock share of spouses who start consuming statins, as manifested by the gradual growth in the estimated statin consumption effect. Note that the pattern of response in cholesterol testing additionally suggests we should focus on the year just after the shock (period 1) as the initial response period when analyzing statin effects due to potential transitional delays: this is the first data period in which all individuals are fully exposed to the shock and have been already able to engage in the required medical testing that precedes medication consumption.

To understand the nature of these spousal spillover effects, we proceed by investigating response heterogeneity using different sources of variation. We first ask the following natural question: does the event-driven consumption response interact with underlying risk? That is, while all treated families are faced with a cardiovascular shock, we want to study whether they trigger more action among spouses whose baseline risk of own cardiovascular events is higher. To do that, we first calculate an annual measure of cardiovascular risk using population data. Our prediction relies on risk factors used by medical practitioners (De Backer et al. 2003; Pencina et al. 2014), constrained to those observable in our data. These include age and gender, as well as the presence of hypertension and diabetes, for which we proxy by condition-specific prescribed medications. Since smoking is also used for risk predictions by medical professionals, we include education as the best predictor included in our data that has been shown to strongly correlate with this behavior (see, e.g., Cutler and Lleras-Muney 2010).[20] Then, we match to each spouse a measure of his or her underlying risk based on the predicted probability of a cardiovascular shock in the baseline period $t = -1$, and split the sample into high and low risk using the population median.

Column 1 of Table 2 estimates equation (4), where z_i denotes an indicator for whether the spouse's

[20] For completeness, note that medical professionals estimate an individual's ten-year cardiovascular risk using age, gender, blood pressure, cholesterol levels (total and HDL/"good" cholesterol), the presence of diabetes, and smoking habits. Correspondingly, the variables included in our annual predictions (based on a probit regression) are age fixed effects, gender, education (in months), and lagged indicators for whether an individual consumes medication for high blood pressure or diabetes.

predicted risk is above the median, and the post-shock periods for which $post_{i,t}$ assumes the value 1 are periods 3 and 4 when the response levels out (as seen if Figure 1). We find that even low risk spouses are induced to increase their take-up of statins as preventive care in response to the family shock (by 0.43 pp). However, at the same time, high risk spouses are much more likely to increase their statin consumption—by additional 1.08 pp—in response to the shock. This behavior is consistent with the economic intuition by which greater investments, here in health through the consumption of preventive treatments, are made by agents whose expected returns from doing so are higher. Interestingly, we find that there is no such risk gradient in spouses' "information-seeking" behavior itself through cholesterol testing. This implies that spouses across risk types are uniformly induced to take actions related to cardiovascular risk (in the form of gathering data), whereas those with actual higher predicted risk end up having larger statin responses in practice, consistent with a learning channel of the spillover effect (see columns 1 and 2 of Appendix Table 2).[21] Lastly, we find additional support to the learning hypothesis by providing evidence consistent with the idea that individuals who are more likely to learn from spousal shocks, due to similar risk profiles across them, exhibit stronger responses. Columns 3 and 4 of Appendix Table 2 show that spouses with closer predicted underlying risk tend to increase their consumption of statins to a larger extent following the family shock.

Next, we further exploit the information on cholesterol testing to probe into the likely mechanisms that underlie spousal responses. A large body of medical research and the corresponding clinical guidelines indicate that the information on risk relevant for receiving cholesterol-reducing treatment is a combination of one's LDL ("bad") cholesterol levels and the predicted cardiovascular risk we discussed in the previous paragraph, which is based on observables (see, e.g., De Backer et al. 2003 and Pencina et al. 2014). Accordingly, spouses whose cholesterol levels had been tested for in the periods prior to the shock, already have access to the entire information set regarding their own cardiovascular risk by which statin eligibility is determined. Note that this is not the case for family circles with first-degree biological links (such as children or siblings), for whom eligibility should also incorporate family history. We are therefore interested in studying whether this set of spouses respond in any way to the shock. Notably, we find that they do: even spouses who had access to the set of relevant information on their own risk prior to the event increase their consumption of statins in response to the family shock (see column 2 of Table 2).[22]

Combined, the heterogeneity analysis so far highlights both the likely role of family shocks in inducing learning about one's own health (when not all the preexisting relevant information is known prior to the shock), and that salience and attention are likely operative channels, as spouses respond even when the relevant information set had been available to them before the family shock occurred.[23]

[21] We increase power to get informative standard errors on interaction terms in this small sub-sample of residents of Greater Copenhagen by including spouses of all individuals who survived for at least three years following their cardiovascular shock.

[22] We find similar (slightly noisier) results when studying spouses with even more updated information (those who have been tested in periods -2 or -1). See column 5 of Appendix Table 2.

[23] In addition to the analysis in this subsection 5.1.1, we were interested in studying whether the effects on spouses can be explained by supply-side responses in the form of family physicians who aggregate information across the different members of the household. To this end, Appendix Table 4 analyzes households in which husbands and wives do not share the same doctor, defined in several ways as explained in the table's notes. We find similar-magnitude effects among these households so that the spillover is unlikely attributed to aggregation of information by the family doctor. In the same appendix table, we also conduct this exercise for adult children, whom we study in the next subsection, and we reach similar conclusions.

5.1.2 Inter-generational Effects

Having studied intra-generational effects on spouses, we next turn to analyze households in which adult children are present at the time of the shock, to study how parental shocks spill over to health behaviors in the next generation. We begin by analyzing the statin consumption responses of biological children to parental heart attacks or strokes at different stages of the life cycle.

Biological Children. Panel A of Figure 2 plots the average response in statin consumption by the younger adult children in our sample, who were between ages 25 and 40 at the time of their parent's health shock. As before, the figure provides a visual verification of parallel pre-trends across our treatment and control groups, in validation of the estimation strategy. Studying the effect of the shock, the plot reveals an immediate response by children which grows stronger over time. Estimating equation (2), column 4 of Table 1 shows that by the fourth year after their parent experiences a heart attack or stroke, adult children increase their take-up of statins by 0.43 pp. From a low baseline of 1.18 pp due to this group's younger age, the treatment effect amounts to a large increase of more than 36%. Analogously, panel B of Figure 2 plots the average causal effect for the older children in our sample, who were between ages 40 and 65 at the time of the parental shock. This group reveals a similar pattern of an immediate increase in stain consumption that amounts to more than 16% by the fourth year after the shock—an increase of 0.80 pp on a base of 4.93 (see column 5 of Table 1).

Per the convention of the medical profession, premature parental cardiovascular shocks are viewed as revealing more information on a child's biological risk (and are therefore incorporated into cardiovascular risk predictions; see De Backer et al. 2003 and Pencina et al. 2014). Accordingly, we study whether children whose parents were younger at the time they experienced the cardiovascular shock, who are hence more likely to learn new information of their own risk, are also more prone to increase their consumption of preventive care. Column 3 of Table 2 estimates equation (4), where we interact the treatment effect with both the child's own age and the parent's age at the time of the parental shock. Consistent with a direct ("exogenous") learning channel, by which shocks to older parents reveal less information regarding a child's biological risk, we find a strong negative partial correlation between children's statin consumption responses and their parent's age at the time the shock occurs.

These causal spillovers to health investments by biological children involve several channels. These channels could include direct revelation of information about biological risk as we just discussed, indirect revelation of information (by induced learning) on own risk that could pertain to shared environmental risk growing up, and salience and increased awareness. Subject to the same set of channels, we find similar response patterns by siblings in Figure 3. In fact, this turns out to be an important route through which family spillovers operate. Among the different family circles we study, siblings seem to display the largest spillovers, consistent with a stronger signal regarding one's own risk from a shock to a first-degree family member of the same generation. Specifically, their increases in consumption of preventive care amount to 41% and 24% for younger (25-40) and older (40-65) siblings, respectively (see columns 6 and 7 of Table 1 for the magnitudes). We report these findings here for completeness, and focus our analysis in the rest of this section on family and social connections that can enable us to isolate response channels, with the aim to shed light on the spillovers' nature and sources. Accordingly, we first abstract from the biological-risk channel by proceeding to analyze

potential spillovers to stepchildren.

Stepchildren. Column 1 of Table 3 estimates the mean treatment effect of cardiovascular shocks to non-biological parents on stepchildren's consumption of statins, using the differences-in-differences specification of equation (3). We find that the spillover in health behaviors cascades also to non-biological children and amounts to an 11% (or 0.25 pp) increase in their consumption of statins. To compare magnitudes across non-biological and biological children, we estimate the mean-effect equation (3) for the sample of comparable biological children, so that we include individuals of all ages (25-65) whose parent survived for at least three years. Column 2 of Table 3 estimates an average effect of 0.41 pp for this sample of biological children that represents a 19% increase from a baseline similar to that of stepchildren. Hence, the results imply that stepchildren exhibit causal responses that are half as large as those of biological children—a spillover effect that cannot be attributed to revelation of new information related to genetic risk. While the standard errors are more than twice as large in the analysis of stepchildren, the dynamic regression is nonetheless able to provide estimates with sufficient accuracy. Column 1 of Table 4 displays the estimation of specification (2) and shows that the qualitative pattern of the dynamic effects of parental shocks on non-biological children bears a close resemblance to that on biological children. The impact begins in the year just after the shock (when all individuals are already exposed to the shock for a full period) and grows over time, so that by the end of our analysis' horizon adult stepchildren increase their consumption of statins by 17%.

Sons and Daughters In-Law. Next, after abstracting from the biological-risk channel, we seek to abstract from the spillover channel by which children may respond to shocks as a result of learning information on joint risks attributed to sharing the same environment with their parents when growing up. This includes a variety of dimensions such as the immediate physical surroundings, as well as similar lifestyles and nutrition habits across generations within the household. To take a step in this direction, we constrain our sample to married children, and study the potential spillovers to their spouses—i.e., to sons and daughters in-law. Column 3 of Table 3 estimates the average treatment effect specification of equation (3) and finds that, following the cardiovascular shock to their spouse's parent, children in-law exhibit an average increase of 0.11 pp in their consumption of statins. As a benchmark, note that this effect amounts to about a quarter of that of their spouses, i.e., the biological children (for whom the estimate is reported in column 4 of Table 3). While this effect is small in magnitude, its importance lies in revealing the breadth of the spillovers that we identify. That is, family health shocks not only affect the behavior of the next generation of biological and non-biological children, but they also spill over to the next generation's households. Notably, this effect cannot be directly explained by the revelation of information related to own risk either due to genetics or due to the familial environment in childhood or adolescence.

To further investigate the nature of the spillover to sons and daughters in-law, we test whether "distance" across households matters. As the simplest measure for distance that may capture the degree of exposure to the shock and its prominence, we look at variation in geographical distance based on municipality of residence at the baseline period $t = -1$.[24] We divide the sample into children in-law

[24]Specifically, we use individuals' residence at $t = -1$ to assign households to municipalities based on the municipality division

who live closer to or further from their parents in-law using the median distance. Column 5 of Table 3 first replicates the overall average effect on in-laws from column 3 but for the sample of families for whom we have non-missing data on residence.[25] Then, in column 6 we provide the average effect for each sub-sample split by distance, calculated using a regression of specification (4). The results reveal that the effect on in-laws is fully attributed to the next generation households who live closer to their parents. Cutting the sample further, we can see (in column 7) that these are actually children in-law whose distance from their spouses' parents are shortest, less than the 25th percentile, who drive the results. In fact, because of that, the time pattern of their responses in statin consumption can be accurately estimated using the dynamic regression of specification (2) (see column 2 of Table 4). They exhibit immediate responses following the shock with an effect of a 15% increase by the end of the analysis' period (similar to spouses). Overall, these results of a distance gradient are consistent with exposure intensity and salience of family shocks as operative routes for the estimated spillover.[26]

5.1.3 Effects on Coworkers

In the final empirical exercise of this section, we study how far the spillovers of health shocks into improved health behaviors can reach, by analyzing the effects on coworkers. To approximate peers with whom individuals are more likely to interact, we focus the analysis on "close" coworkers (as defined in Section 4.2).[27] Column 1 of Table 5 displays the average treatment effect of health shocks on close coworkers' consumption of preventive care. Interestingly, the results indicate a meaningful increase of 1.39 pp, which amounts to an average effect of 13%.

As this effect is economically considerable, it also clearly shows up in the dynamic regression as displayed in column 3 of Table 4. With no differential trends in the pre-period, coworkers' take-up of statins exhibits a prompt increase in the years following a peer's cardiovascular shock which persists for the full analysis period. This evidence highlights a meaningful spillover that is transmitted through ties that are purely social which is, notably, on the same order of magnitude as the estimated spillovers to prime-age spouses.

Our setting also provides us with the opportunity to identify the limit of spillovers, by revealing that close workers are likely the point at which they stop. That is, while notable spillovers are present among close coworkers, they are absent when we study "distant" coworkers within the same workplace, for whom we find no causal effects with precisely estimated zeros. In particular, we first show there are no spillovers to coworkers in larger workplaces, in which the average frequency of interactions between any two coworkers is likely lower. More interestingly, we find no effects on coworkers within smaller workplaces (so that they are still likely exposed to the shock) when we focus on those with greater age

post 2007 which divides Denmark into 98 municipalities. Distance is defined as the length of the straight line between municipality centroids. The median distance in our sample between parents and children in-law is 22 kilometers (14 miles).

[25] Note that we additionally constrain the sample to include only parents and children in-law who do not share the same doctors to verify a distance gradient would not be attributable to that.

[26] Another potential explanation for a distance gradient could be environmental health risks (such as air pollution). In such a case, one would expect a similar distance gradient in the responses of the children married to these sons and daughters in-law, for which we find no evidence in the data.

[27] Namely, we look at coworkers within the same occupation class and age range in smaller workplaces (where perturbations to cutoffs used in these definitions are reported in Appendix Table 3 as we mentioned before).

gaps or those within the same physical establishment but with different occupation classes, who may therefore represent peers with weaker social ties (see columns 2-4 of Table 5).[28] Therefore, put together, the patterns of spillover impacts on the consumption of healthcare by close coworkers, and the lack thereof on distant coworkers, are consistent with the strength of the social tie and the corresponding degree of shock exposure and prominence as operative channels.

5.1.4 Economic Magnitudes and Benchmarks

Comparing our estimated spillovers to baseline counterfactual levels as simple benchmarks has pointed to economically significant impacts on multiple network circles. To further gauge economic magnitudes, we provide two additional exercises.

First, we quantify how much of the time pattern in the consumption of preventive care by the different circles of family members and peers can be explained by the spillover of the one shock alone. Specifically, we calculate the overall increase in consumption within the full analysis window (from period -4 to $+4$), which takes into account time and life-cycle effects and the impact of the spillover, and we then assess the share of the spillover effect out of this overall increase. In order to evaluate this quantity across different network layers of comparable ages, we avoid including the very old, for whom we have observations only in the spouses sample.

With this metric, we find that the spillover effect to adult biological children in our younger sample accounts for 28% of the growth in their statin use within the entire 9-year analysis period. For older children, this number amounts to 15%, so that on average in the overall sample 22% of children's growth in consumption of preventive care is attributed to the one parental shock. Similarly, among siblings the spillover effect represents 31% and 21.5% of the 9-year growth in statin consumption for younger and older individuals, respectively.

For prime-age spouses, our estimated spillover explains about 15% of the growth in statin consumption over time. Notably, we estimate similar magnitudes for individuals in the further circles that we study. Specifically, the share of change in health behaviors that is explained by the spillover is 15.5% for stepchildren, 15.4% for sons and daughters in-law that live close to their in-laws, and 12% for peers as proxied by close coworkers. It is worth noting that these assessments capture the effect of a single shock, of one network member, and of a particular type of health risk; so that, over the natural course of the life cycle, spillovers from shocks to different circles of family and peers can add up to play an important role in determining individuals' health behaviors.

To provide a second exercise to put the effect magnitudes in perspective, it may be useful to compare them to benchmarks that pertain to under-utilization of statins as preventive care and to the recommended population shares that should be consuming them. The most straightforward benchmark that we know of, which provides these exact moments albeit for a different population, is the study by Pencina et al. (2014) in the U.S. context based on the National Health and Nutrition Examination Survey (NHANES). Pencina et al. (2014) report assessments using different sets of medical guidelines

[28] Note that these workers within small workplaces, who are close in age but are in a different occupation class, still share the same geographical location and hence also environmental risks, so the absence of a spillover to them suggests this type of risk is not likely to drive the effects on close coworkers. Also, the finding of no effect on coworkers in similar location and occupation but with a larger age difference suggests that job-related risk (occupational risk, stress, etc) is not a likely channel as well.

for the treatment of cholesterol, and we use their estimates that are based on the "2004-updated ATP-III" criteria as they are the ones that are relevant for our time period and most closely mirror the Danish guidelines of that time.[29] Their findings suggest that the ratio of those eligible for statin therapy, who do not receive it, to those eligible, who do receive it, is 71.4%. Hence, for adults of ages 40-75 who are covered by the survey, an effect of this magnitude would close the gap of under-utilization. To get the most comparable numbers from our application, we calculate as an illustration the medium-run (year-4) effect for spouses, adult children, and siblings, who are in this age range at the end of the analysis period.[30] For these subgroups (where a similar exercise can be conducted for the other circles), we find that statin take-up increases by 10% for spouses, by 21.5% for adult children, and by 23.7% for siblings. *If* one is willing to assume that baseline utilization for these family members requires a 71.4% increase to reach recommended levels, the spillovers close 14% (=10/71.4) of the gap for spouses, about 30% (=21.5/71.4) of the gap for adult children, and they close 33% (=23.7/71.4) of the potential under-utilization gap among siblings.

To summarize our results so far, we find immediate and sustainable effects of family health shocks on spouses' and adult children's consumption of preventive care. We show that these spillovers in health-care consumption are far-reaching as they cascade to stepchildren, to the next generation's households, and even to peers as approximated by coworkers. The effects that we estimate are economically significant and the displayed degree of adherence highlights a role of the family (and peers) in habit formation within the context of health. The analysis of different circles of one's family and social network, for whom the set of potential channels of impact vary, and the investigation of heterogeneity along the dimensions of potential exposure and of information on own health risks, point to likely mechanisms that govern these effects. Specifically, while we find evidence consistent with the more traditional channel of learning new information, the analysis also demonstrates that attention and salience are likely key operative channels. We provide further evidence in support of these conclusions in our next set of results.

5.2 Part II: Effects of Fatal Health Shocks

We now turn to the second complementary part of our analysis and investigate family spillovers in health behaviors in the context of the extreme events of fatal health shocks. The goal of this analysis is to further explore the nature of family spillovers that shape health behaviors and their likely underlying explanations. The advantages of the current context are the opportunity to investigate a variety of utilization margins indicative of changes in health behaviors and investments (where before we have focused on one margin particular to cardiovascular risk), as well as the ability to test different mechanism-related hypotheses by leveraging detailed codes of causes of death. The analysis proceeds in two steps. In the first step, we study the effects of general fatal shocks on family members' awareness of health and common health-related behaviors. In the second step, we exploit variation in specific

[29] As we alluded to before, these guidelines use a combination of an assessment of one's ten-year cardiovascular risk and the low-density lipoprotein (LDL/"bad") cholesterol level to recommend treatment. See more details in the appendix of Pencina et al. (2014).

[30] Of course, this exercise for the constrained age range yields different conditional age distributions for the different samples due to their different unconditional age distributions. See Appendix Table 1 for unconditional age means.

causes of death to study consumption of different condition-specific preventive care, both within and across conditions. This enables us to explore the directionality of responses and the degree to which they may be local to the particular nature of the experienced shock. In what follows, the analysis focuses on spouses since, as before, it identifies family spillovers that are not attributed to biological channels. Additionally, spouses within our sample are older and hence have higher baseline levels of healthcare utilization compared to their children, which allows clear estimation of spillover dynamics for any given level of relative response. Still, we replicate the main findings for the average spillover impact on the next generation of adult children in the appendix.[31]

5.2.1 Health Awareness and Common Health Behaviors

Increased Awareness of Health. We begin by analyzing the effects of fatal shocks on family members' general awareness of health using two proxies for the degree to which individuals pay attention to health issues. The first outcome that we study is an indicator for whether spouses are hospitalized for visits that end up being classified as encounters for medical observation of suspected conditions that are ruled out ex-post. These hospital contacts can be indicative of greater vigilance to symptoms that are retrospectively realized as "false alarm". For visual clarity of the dynamic patterns that we find in this subsection, we report our findings by plotting the δ_r coefficients from estimating specification (2) along with their 95-percent confidence intervals. We also indicate on the figures the counterfactual outcome levels for periods $t = 0$ and $t = 4$, the beginning and end of the analysis period, to gauge response magnitudes relative to underlying levels.[32]

Panel A of Figure 4 displays spousal responses using our first proxy for health alertness. The figure shows that, in the immediate years just after their spouse's death, individuals' propensity to visit hospitals on account of suspected health conditions clearly and meaningfully increases. This effect seems to dissipate over time, although the increased propensity is still present four years out.

The second outcome that can be indicative of a sense of urgency regarding one's own health is (non-hospital) "urgent care" contacts with the medical system—i.e., contacts with local doctors or nurses who are on call that are initiated outside of regular working hours.[33] Panel B of Figure 4 reveals a similar (and more pronounced) response pattern in this outcome: there are significant on-impact increases in the propensity of urgent care contacts that then fade out, though do not fully disappear, in the course of four years after the shock. Together, the two outcomes suggest that individuals experience a period of several years in which they exhibit heightened awareness regarding their own health status, that translates to increased consumption of healthcare, following their spouse's death.

Declines in Harmful Behaviors and Medication. A large share of the literature on adverse health behaviors has focused on smoking, excessive alcohol consumption, and drug and medication abuse

[31] See Appendix Table 5. Our data use agreement excludes some information on drug prescriptions for the sample of adult children (as opposed to spouses). Drug dosage is part of the excluded data, so that responses in prescription opioid doses are the one outcome for which we cannot provide the corresponding estimation for adult children.

[32] In this subsection we analyze a variety of utilization codes that cover different calendar years (due to institutional changes in data reporting). Therefore, as we mentioned in Section 3.1, we rely here on specification (2) which can further balance the treatment and control groups in a disciplined way. That said, the dynamics of spousal responses are visually clear in raw data figures which are available on request.

[33] Each geographical location is assigned a primary-care provider that is on call outside of regular working hours which are 8 am to 4 pm.

(see Cawley and Ruhm 2011). Guided by this literature, we exploit the prescription drug data to explore potential changes in such existing harmful behaviors in response to family shocks as additional measures for improvements in health behaviors. Specifically, we study whether fatal family shocks lead individuals to seek treatments to reduce their smoking or drinking, and whether these shocks induce individuals to decrease their utilization of addictive medication.

To this end, we first explore the consumption of medication that treats nicotine or alcohol dependence. This class of medication is prescribed to individuals who have been engaged in *chronic* smoking or excessive drinking for lengthy periods of time, and who wish to cease their unhealthy behavior or to switch to a less damaging substitute (Siu 2015; Swift and Aston 2015).[34] It is worth noting that, as such, evidence of prompt increases in their consumption would suggest they are taken for treating preexisting conditions (rather than for treating newly-acquired conditions that may have been caused by spousal death). Panel C of Figure 4 displays the estimates of equation (2) where the outcome variable is an indicator for the purchase of a prescription drug within this class. Notably, the estimates show an immediate increase in individuals' consumption of these medications following spousal death, which amounts to an increase of 41% compared to the counterfactual.[35]

To shed light on the potential response channels, we further wish to test whether these effects can be fully attributed to learning new information regarding the risk involved in smoking or drinking, which might have been revealed to unaware agents by their spouse's death. We therefore study the mean effects of spousal death for a small class of causes of death that the medical research has not been able to link to any risky behavior and, in particular, to smoking or drinking: autoimmune diseases (NIH 2016). Interestingly, we find that even in these cases survivors engage in treatments to reduce smoking or drinking, although the fatal event itself is not directly related to or informative of the risk associated with these behaviors (column 1 of Appendix Table 5).

Next, we move on to prescription medication abuse, where concerns pertain to the dangers of developing dependence or addiction. In this regard, we study a main class of addictive harmful medication—prescription opioids for pain relief—which account for the greatest proportion of mortality cases linked to prescription drug abuse (Volkow 2014; Rudd et al. 2016).[36] In this case, health-promoting behavioral responses would translate into reductions in consumption. Since opioid dose reductions should be gradual due to withdrawal symptoms (Miller and Kipnis 2006; Volkow 2014), we study a continuous measure of dosage. Specifically, we use the standard "defined daily dose" (DDD) measure of drug consumption (defined by the World Health Organization), which standardizes the amount of the prescribed medication in day equivalents.[37] Panel D of Figure 4 reveals a pattern that is closely consistent with improved behavior in the current context. From a baseline counterfactual level of 9.74 prescription days (in period 0), spousal death induces a decline in the amount of prescription opioids

[34] This class of prescription drugs is labeled under ATC codes N07BA and N07BB. For smoking cessation these prescription drugs include medications and nicotine replacement therapies (such as nicotine chewing gum and patches), which are widely recommended to all adults as a part of cessation regimens.

[35] We find similar results when we run separate regressions for sub-classes of this group of prescription medication (that treat either nicotine dependence or alcohol dependence).

[36] These medications have been a recent focus of the medical literature and public debate due to the common practice of abuse and the high risk of addiction or dependence associated with them.

[37] This measure is based on the assumed average maintenance dose per day of a drug for adults and is used to compare drug usage across different drugs or healthcare environments.

consumed that is already detectable in the year the shock occurs, and gradually reaches a decrease of 0.93 days by the fourth year after the shock (on a counterfactual level of 14.85). Panel E shows that the same results hold when we exclude prescription opioid poisoning as a cause of death, so that the event itself is not directly linked to the studied behavioral response.

Put together, the results of this subsection imply that fatal family shocks increase individuals' awareness of their own health and induce them to engage in efforts to cease unhealthy behaviors in the context of key harmful habits. The findings suggest that this is true even when the shock is not tied to the spousal behavioral response or when it is unlikely informative of the health risks involved in certain behaviors, consistent with a salience channel by which individuals' attention is generally drawn to their own health following family shocks.

5.2.2 Condition-Specific Preventive Care

We now proceed with the second part of the analysis of fatal shocks, which constitutes our final set of results. In this part, we study the consumption of condition-specific preventive care by exploiting variation in specific causes of death. As we show below, the analysis provides us with evidence regarding the directionality of family spillovers, in terms of the domain of affected behaviors, as well as with supplementary tests for underlying channels.

Within-Condition Preventive Care. Our core question for the current analysis is the following: do individuals increase their utilization of types of preventive care that are directly-linked to the particular health condition of the family shock? We answer this question in the context of two main classes of preventive-care practices that are tied to the two leading causes of death in the developed world: cardiovascular disease and cancer. For cardiovascular deaths, we study the consumption of statins as we did before. For cancer deaths, we study expenditure associated with visits to diagnostic radiologists, who specialize in disease diagnosis and are responsible for screening patients for major types of cancer. The empirical strategy follows equation (5) estimated on treated households only, where we let z_i divide the sample by cause of death, so that it is assigned the value 1 if the family member died of disease x and it is assigned the value 0 otherwise. By letting the outcome variable be a measure of preventive care that is particular to disease x, we analyze the utilization of this preventive care by individuals whose spouse died of disease x compared to those whose spouse died of any other cause.

Starting with the application to cardiovascular disease as cause of death, the results bolster our findings from the analysis of non-fatal cardiovascular shocks. Column 1 of Table 6 estimates equation (5) where the outcome is statin consumption and the main right-hand side variable (z_i) indicates whether the spouse died of a heart attack or a stroke or of any other cause. It is worth noting that, in support of the empirical design, there are no differential pre-trends across the two groups of households, so that $\delta_r = 0$ for $r < 0$. Observing the post-shock coefficients, a clear pattern emerges: following fatal shocks, individuals whose spouse died of cardiovascular disease persistently consume statins at a higher rate compared to those whose spouse died of any other cause. This differential utilization already begins in the year of the shock and then gradually grows over time until it reaches an increase of 0.71 pp by the fourth year after the shock (on a baseline of 7.7 pp when the shock occurs).[38]

[38] For this application, we also have sufficient power to characterize the dynamics for children, for whom we find the same pattern (see column 2 of Table 6). Note that, as in the case of non-fatal cardiovascular shocks, family members of individuals who died

Next, we study our second class of preventive care and analyze healthcare costs associated with visits to diagnostic radiologists in the context of fatal cancer. Similar to cardiovascular deaths, we compare individuals whose spouse died of cancer to those whose spouse died of any other cause. However, unlike the case of statins (where improved health behaviors require persistent changes in consumption), the current utilization outcome of visits to radiologists is for diagnosis purposes only (rather than continuous health risk management), so that behavioral responses should translate to transitory effects.

Column 3 of Table 6 estimates equation (5), where the outcome is expenditure on visits to diagnostic radiologists, and z_i is an indicator variable that assumes the value 1 if the deceased died of cancer and the value 0 otherwise. Again, there are no differential pre-trends across the two experimental groups in support of the design. Investigating the outcome following the shock, we find that in the year just after spousal death of cancer, individuals' healthcare expenditure on diagnostic radiology significantly increases compared to spousal death of any other cause. This differential response amounts to an increase of 12.2% and vanishes by the third year after the shock. In the current context, this pattern of behavioral responses is tightly in line with effects of family shocks on condition-specific *diagnostic* preventive care. The pattern is also in accordance with results from the estimation of equation (5) where the outcome variable is outpatient hospital contact for the reason of having a family member with history of cancer, which exhibits increased incidence just after spousal death (column 4 of Table 6).[39]

The detailed information on causes of death allows us to further investigate how directed the responses are toward particular risks, by looking into specific types of cancer. The first high-incidence type of cancer we investigate is lung cancer and, in this context, we study the consumption of medication for smoking cessation. If improved health behaviors are more targeted toward the particular risk of which the family member had died, we would expect stronger responses by those whose spouse died of lung cancer than by those whose spouse died of non-lung cancer. For this analysis, as we investigate thinner sub-samples, we estimate the average differences-in-differences type specification of equation (6), where now z_i further splits the sample by the specific type of cancer. Doing so, we find in column 1 of Table 7 that individuals whose spouse died of lung cancer are 24% more likely to engage in smoking cessation following the shock compared to those whose spouse died of any non-cancer cause. In contrast, column 2 of Table 7 displays no such differential response when we compare spouses of individuals who died of non-lung cancer to those of individuals who died of any non-cancer cause. This further points to the directionality of behavioral responses toward actions that are more relevant to the particular experienced risk.

The second class of cancer types that we study is female cancers, which in our sample include ovarian, cervical, and breast cancer.[40] For this class of cancers, we study husbands' diagnostic radiology expenditure, comparing those whose wife died of female cancers to those whose wife died of any non-

of heart attacks or strokes as compared to any other cause do not exhibit higher incidence of cardiovascular disease following the shock, so that the increased use of statins is for preventive purposes.

[39] The specific code is Z80 in the ICD-10 classification: "family history of malignant neoplasm."

[40] Note that, while very rarely, men can also die of breast cancer. In our sample, we detect 138 such cases (0.04% of all male deaths), as compared to 14,541 female deaths of breast cancer (7.6% of all female deaths).

cancer cause. The advantage of this context is the limited potential for information revelation regarding husbands' own risk of cancer. Indeed, we find that a wife's death of female cancer does not predict a higher incidence of cancer for the husband himself.[41] Still, we find responses that are very similar in magnitude to what we have found so far, so that husbands whose wife has died of female cancer meaningfully increase their expenditure on diagnostic radiology in the years just after the shock relative to those whose wife has died of any non-cancer cause (column 3 of Table 7). In line with no revelation of information that pertains to spouses' own risk, we find no evidence of a differential incidence of cancer diagnoses across these two groups of husbands despite their differential expenditure on diagnostic tests (column 4 of Table 7). As such, the latter set of results highlights attention towards particular risks and their salience as likely operative channels in explaining spouses' behavioral responses.[42]

Cross-Condition Preventive Care. Lastly, we take advantage of the current setting to study cross-condition responses. This provides us with placebo tests that can also indicate how local responses are to the vicinity of the experienced shock. It addresses potential hypotheses such as one by which individuals whose spouse dies of cardiovascular risk or cancer may be generally more responsive than others in any preventive care margin, not only in the margins that are specific to that condition.

To do so, we run similar specifications that analyze the behavior of individuals whose spouse died of disease x compared to those whose spouse died of any other cause, but where the outcome variable is a measure of preventive care that is particular to a different disease x'.[43]

Column 5 of Table 7 finds that expenditures on diagnostic radiology of individuals whose spouse died of cardiovascular disease are not different compared to those whose spouse died of other causes, suggesting that their increased responses in condition-specific preventive care (in this case, statins) were limited to the particular shock they experienced. Note that similarly, further strengthening this result, we find no increases in expenditure on diagnostic radiology in the context of Section 5.1.1, namely, in response to a spouse's non-fatal cardiovascular shock. Next, for cross-condition responses in the context of fatal cancer, we even find declines (see column 6 of Table 7). That is, individuals whose spouse died of cancer exhibit decreased propensity to consume preventive care against cardiovascular disease following the shock. Interestingly, this points to the possibility of crowd out: it is not only the case that individuals increase take-up of condition-specific care, even when shocks are likely uninformative of own risks, but they may also reduce the take-up of preventive care that pertains to competing, non-salient

[41] Specifically, we study in the cross section of households with deceased wives whether a wife's (future or experienced) death of female cancers can be predictive of husbands' contacts with inpatient or outpatient hospital departments for any cancer-related reason (beyond potential diagnostic tests). Regressions that include our set of controls find quite precise zeros, whether we use the post-shock period (-0.0003, s.e. 0.0015) or the pre-shock period (-0.0014, s.e. 0.0013); regressions of raw correlations with no controls actually produce negative estimates (-0.0045 with s.e. of 0.0012 for post-shock periods; -0.0096 with s.e. of 0.0012 for pre-shock periods). Note that ex-ante, before investigating the data, one could not have completely precluded some degree of learning on own cancer risk, since risk factors that have led to the wife's female cancer could potentially lead to a husband's cancer.

[42] We were unable to conduct any meaningful analysis with male-related cancers (such as prostate cancer). We ended up with a very small sample size which resulted in standard errors that were three times as large as those in the analysis of female cancers, which produced wide and uninformative confidence intervals.

[43] Since both cardiovascular disease and cancer compose a large share of deaths, we have excluded them from the baseline groups to avoid mechanical correlations. For example, if we neglect to do it and find that individuals whose spouse died of cardiovascular shock are less likely to visit diagnostic radiologists, it could be driven by the fact the those whose spouse died of cancer are more likely to do so and also constitute a large share of the baseline group (of individuals whose spouse died of any non-cardiovascular cause).

health risks. This is consistent with limited attention and, while suggestive, can have implications for pitfalls in leveraging salience as a policy tool, which we discuss in the next section.

In summary, the findings of this subsection on fatal health events bolster the results from the first part of the analysis. We show that family shocks increase individuals' awareness of their own health and induce them to engage in efforts to reduce unhealthy habits in the context of major harmful behaviors. Furthermore, the results show that behavioral responses in health investments are directed to the vicinity of the particular experienced risk, even when no information on own risk can be likely revealed. This strengthens the conjecture of increased attention to aspects that have become salient to the family due to the shock as an operative explanation for spouses' behavioral changes and health investment choices.

6 Discussion and Conclusion

Tying together our set of results, this paper has identified intra- and inter-generational family spillovers as a causal channel through which health behaviors are shaped over the natural course of the life cycle. We have seen that spouses and adult children immediately increase their health investments and improve their health behaviors in response to family shocks, and that these effects are both economically significant and long-lasting. We have found that the impacts of shocks can be far-reaching, as they also spill over to the consumption of healthcare by siblings, stepchildren, sons and daughters in-law, and even peers. But we have also illustrated when they approach their limit, as individuals with likely weaker social ties are not affected. Thus, overall, we have found network spillovers in consumption that are significant in magnitude, scope, and persistence over time, in the context of healthcare which constitutes a large share of households' expenditure. As such, our findings can also be informative for consumption network effects and multipliers more broadly and for our understanding of their nature and scope. Using different strategies, we have additionally highlighted likely mechanisms that may underlie the estimated spillovers. The evidence supports the hypothesis that shocks within the family or social network act as a vehicle through which individuals learn information about their own health, and points to salience and attention as key operative channels, within a variety of cases where shocks are likely uninformative of one's own risk (either directly or through learning). While this salience route seems to lead to overall increased awareness of health, the findings suggest that agents' attention is particularly drawn to the local nature of the shock so that they take actions that are specific to the realm of that risk.

Consequently, our findings have implications for models of health behaviors and demand for healthcare. First, the results underscore the importance of incorporating inter-personal interactions, similar to the common practice in applications such as the sharing of resources for the consumption of goods and labor supply choices. That is, the evidence suggests that we should model and analyze health behaviors, the demand for health investments, and the consumption of healthcare at the family level; and even more broadly, in the context of one's social network. Our findings could also inform models of health behaviors with respect to the ingredients they should suitably include. For example, they point to event-driven responses, which are in the spirit of Bernheim and Rangel's (2004) cue-triggered decision processes. Moreover, the evidence suggests that within-network learning and, in particular,

salience and attention should be key components in modeling health behavior and investment choices.

Finally, our findings have potential implications for policies that aim to improve population health. Notably, while inducing individuals to change their health habits is challenging, the results provide a proof of concept that health behaviors are not immutable. More concretely, the findings offer the leveraging of family events as a window of opportunity for targeted interventions. This can become increasingly implementable with the growing attention to the family-centered approach of delivering healthcare to adults, in which medical professionals actively involve family members in the treatment process. As we have shown, family health events induce responses with a high degree of adherence, so that they seem to involve the intrinsic motives necessary for behavioral changes. Building on our findings of likely channels, one could devise policies that provide individual-specific information of risks in the course of these family events, or even strategies that exploit salience of health to actively offer preventive care, e.g., by introducing "defaults" that automatically opt family members into optional checkups, screenings, or basic risk-reducing treatment regimens.[44] However, our findings also point to potential pitfalls of salience if leveraged as a policy tool (e.g., through information provision or through surveying as in Zwane et al. 2011). We have found that, on the one hand, individuals' attention can be drawn to particular risks even in the absence of relevant information, which can lead to excessive preventive care that may be both harmful and expensive. But on the other hand, consistent with limited attention, we have seen that increased salience of one risk may come at the expense of another, which can simultaneously divert individuals' attention away from non-salient conditions of which they might actually be at higher risk. Hence, salience-based interventions may be designed more effectively by taking a broad view of their potential consequences and by using more fine-grained personal data so that they could be tightly tailored to households' particular circumstances. Such interventions may induce greater gains by drawing agents' attention to a pertinent aspect specific to them (e.g., their most likely health risk), and, at the same time, may reduce the potential loss involved in unintended crowd out of non-salient dimensions, as these would be made less relevant by design.

[44] This fits the spirit of formal guidelines to reduce cardiovascular risk by the American College of Cardiology (ACC) and the American Heart Association (AHA) (Stone et al. 2013). Specifically, these guidelines recommend family screenings of high-risk individuals to identify additional family members who would benefit from assessment and treatment.

References

Argys, L. M. and D. I. Rees (2008). Searching for peer group effects: A test of the contagion hypothesis. *The Review of Economics and Statistics 90*(3), 442–458.

Babcock, P. S. and J. L. Hartman (2010). Networks and workouts: Treatment size and status specific peer effects in a randomized field experiment. Technical report, National Bureau of Economic Research.

Banks, J., E. Kelly, and J. P. Smith (2013). Spousal health effects: The role of selection. In *Discoveries in the Economics of Aging*, pp. 255–279. University of Chicago Press.

Bernheim, B. D. and A. Rangel (2004). Addiction and cue-triggered decision processes. *The American Economic Review 94*(5), 1558–1590.

Browning, M., P.-A. Chiappori, and Y. Weiss (2014). *Economics of the Family*. Cambridge University Press.

Carrell, S. E., M. Hoekstra, and J. E. West (2011). Is poor fitness contagious? evidence from randomly assigned friends. *Journal of Public Economics 95*, 657–663.

Cawley, J., E. Han, J. J. Kim, and E. C. Norton (2017). Testing for peer effects using genetic data. Technical report, National Bureau of Economic Research.

Cawley, J. and C. J. Ruhm (2011). The economics of risky health behaviors. *Handbook of Health Economics 2*, 95–199.

Chandra, A. and D. O. Staiger (2007). Productivity spillovers in health care: Evidence from the treatment of heart attacks. *Journal of Political Economy 115*(1), 103–140.

Christakis, N. A. and J. H. Fowler (2007). The spread of obesity in a large social network over 32 years. *The New England Journal of Medicine*.

Christopoulou, R., A. Jaber, and D. R. Lillard (2013). The inter-generational and social transmission of cultural traits: theory and evidence from smoking behavior. Technical report, National Bureau of Economic Research.

Clark, A. E. and F. Etilé (2006). Don't give up on me baby: Spousal correlation in smoking behaviour. *Journal of health economics 25*(5), 958–978.

Clark, A. E. and Y. Lohéac (2007). " it wasn't me, it as them!" a study of social influence in risky behaviour by adolescents. *Journal of Health Economics 26*(4), 763–784.

Cobb, L. K., M. A. McAdams-DeMarco, R. R. Huxley, M. Woodward, S. Koton, J. Coresh, and C. A. Anderson (2014). Editor's choice: The association of spousal smoking status with the ability to quit smoking: The atherosclerosis risk in communities study. *American Journal of Epidemiology 179*(10), 1182.

Cohen-Cole, E. and J. Fletcher (2008). Is obesity contagious? social networks vs. environmental factors in the obesity epidemic. *Journal of Health Economics 27*(5), 1382–1387.

Cutler, D. M. and E. L. Glaeser (2010). Social interactions and smoking. In *Research Findings in the Economics of Aging*, pp. 123–141. University of Chicago Press.

Cutler, D. M. and A. Lleras-Muney (2010). Understanding differences in health behaviors by education. *Journal of Health Economics 1*(29), 1–28.

De Backer, G., E. Ambrosioni, K. Borch-Johnsen, C. Brotons, R. Cifkova, J. Dallongeville, S. Ebrahim, O. Faergeman, I. Graham, G. Mancia, et al. (2003). European guidelines on cardiovascular disease prevention in clinical practice: third joint task force of european and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of eight societies and by invited experts). *European Heart Journal 24*(17), 1601–1610.

DellaVigna, S. (2009). Psychology and economics: Evidence from the field. *Journal of Economic Literature 47*(2), 315–372.

Doyle Jr, J. J. (2011). Returns to local-area health care spending: evidence from health shocks to patients far from home. *American Economic Journal: Applied Economics 3*(3), 221–243.

Eisenberg, D., E. Golberstein, and J. L. Whitlock (2014). Peer effects on risky behaviors: New evidence from college roommate assignments. *Journal of Health Economics 33*, 126–138.

Falba, T. A. and J. L. Sindelar (2008). Spousal concordance in health behavior change. *Health services research 43*(1p1), 96–116.

Fletcher, J. and R. Marksteiner (forthcoming). Causal spousal health spillover effects and implications for program evaluation. *American Economic Journal: Economic Policy*.

Franks, M. M., A. M. Pienta, and L. A. Wray (2002). It takes two: marriage and smoking cessation in the middle years. *Journal of Aging and Health 14*(3), 336–354.

Giorgi, G. D., A. Frederiksen, and L. Pistaferri (2016, June). Consumption network effects. Working Paper 22357, National Bureau of Economic Research.

Imbens, G. W. and J. M. Wooldridge (2009). Recent developments in the econometrics of program evaluation. *Journal of Economic Literature 47*(1), 5–86.

Jin, L. and N. A. Chrisatakis (2009). Investigating the mechanism of marital mortality reduction: the transition to widowhood and quality of health care. *Demography 46*(3), 605–625.

Khwaja, A., F. Sloan, and S. Chung (2006). The effects of spousal health on the decision to smoke: Evidence on consumption externalities, altruism and learning within the household. *Journal of Risk and Uncertainty 32*(1), 17–35.

Kling, J. R., J. B. Liebman, and L. F. Katz (2007). Experimental analysis of neighborhood effects. *Econometrica 75*(1), 83–119.

Kremer, M. and R. Glennerster (2011). Chapter four - improving health in developing countries: Evidence from randomized evaluations11we are grateful to jacobus de hoop, ludovica gazze, martin rotemberg, mahvish shaukat, and anna yalouris for excellent research assistance. this chapter draws on ahuja et al. (2010); holla and kremer (2009); glennerster et al. (2009); and bates et al. (2011). In M. V. Pauly, T. G. Mcguire, and P. P. Barros (Eds.), *Handbook of Health Economics*, Volume 2 of *Handbook of Health Economics*, pp. 201 – 315. Elsevier.

Leonard, K. E. and P. Mudar (2004). Husbands' influence on wives' drinking: testing a relationship motivation model in the early years of marriage. *Psychology of Addictive Behaviors 18*(4), 340.

Ludwig, J., L. Sanbonmatsu, L. Gennetian, E. Adam, G. J. Duncan, L. F. Katz, R. C. Kessler, J. R. Kling, S. T. Lindau, R. C. Whitaker, et al. (2011). Neighborhoods, obesity, and diabetes: a randomized social experiment. *New England Journal of Medicine 365*(16), 1509–1519.

Lundborg, P. (2006). Having the wrong friends? peer effects in adolescent substance use. *Journal of Health Economics 25*(2),

214–233.

McGeary, K. A. (2015). Spousal effects in smoking cessation: Matching, learning, or bargaining? *Eastern Economic Journal 41*(1), 40–50.

Meyler, D., J. P. Stimpson, and M. K. Peek (2007). Health concordance within couples: A systematic review. *Social Science and Medicine 64*(11), 2297–2310.

Miller, N. S. and S. S. Kipnis (2006). *Detoxification and substance abuse treatment*. US Department of Health and Human Services, Public Health Service, Substance Abuse and Mental Health Services Administration, Center for Substance Abuse Treatment.

National Institutes of Health (2016). Understanding autoimmune diseases. (Publication Number 16-7582).

Pencina, M. J., A. M. Navar-Boggan, R. B. D'Agostino Sr, K. Williams, B. Neely, A. D. Sniderman, and E. D. Peterson (2014). Application of new cholesterol guidelines to a population-based sample. *New England Journal of Medicine 370*(15), 1422–1431.

Redberg, R. and M. Katz (2016). Statins for primary prevention: The debate is intense, but the data are weak. *JAMA 316*(19), 1979.

Renna, F., I. B. Grafova, and N. Thakur (2008). The effect of friends on adolescent body weight. *Economics & Human Biology 6*(3), 377–387.

Rosenbloom, C. A. and F. J. Whittington (1993). The effects of bereavement on eating behaviors and nutrient intakes in elderly widowed persons. *Journal of Gerontology 48*(4), S223–S229.

Rosenquist, J. N., J. Murabito, J. H. Fowler, and N. A. Christakis (2010). The spread of alcohol consumption behavior in a large social network. *Annals of Internal Medicine 152*(7), 426–433.

Rudd, R., N. Aleshire, J. Zibbell, and R. Gladden (2016). Increases in drug and opioid overdose deaths-united states, 2000-2014. *CDC–Morbidity and Mortality Weekly Report 64*(50), 1378–1382.

Sacerdote, B. (2014). Experimental and quasi-experimental analysis of peer effects: Two steps forward? *Annual Review of Economics 6*(1), 253–272.

Shah, S., I. Carey, T. Harris, S. Dewilde, C. Victor, and D. Cook (2013). Impact of partner bereavement on quality of cardiovascular disease management. *Circulation 128*(25), 2745–2753.

Simeonova, E. (2013). Marriage, bereavement and mortality: The role of health care utilization. *Journal of Health Economics 1*(32), 33–50.

Siu, A. (2015). Behavioral and pharmacotherapy interventions for tobacco smoking cessation in adults, including pregnant women: Us preventive services task force recommendation statement. *Annals of Internal Medicine 163*(8), 622–634.

Stahl, S. T. and R. Schulz (2014). Changes in routine health behaviors following late-life bereavement: A systematic review. *Journal of Behavioral Medicine 4*(37), 736–755.

Stone, N. J., J. Robinson, A. H. Lichtenstein, C. N. B. Merz, C. B. Blum, R. H. Eckel, A. C. Goldberg, D. Gordon, D. Levy, D. M. Lloyd-Jones, et al. (2013). 2013 acc-aha guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults. *Circulation*, 01–cir.

Swift, R. M. and E. R. Aston (2015). Pharmacotherapy for alcohol use disorder: current and emerging therapies. *Harvard Review of Psychiatry 23*(2), 122.

Trogdon, J. G., J. Nonnemaker, and J. Pais (2008). Peer effects in adolescent overweight. *Journal of Health Economics 27*(5), 1388–1399.

Volkow, N. D. (2014). Prescription opioid and heroin abuse. *National Institute on Drug Abuse. The Science of Drug Abuse and Addiction. Bethesda, MD: National Institutes of Health*.

World Health Organization (2014). The top 10 causes of death.

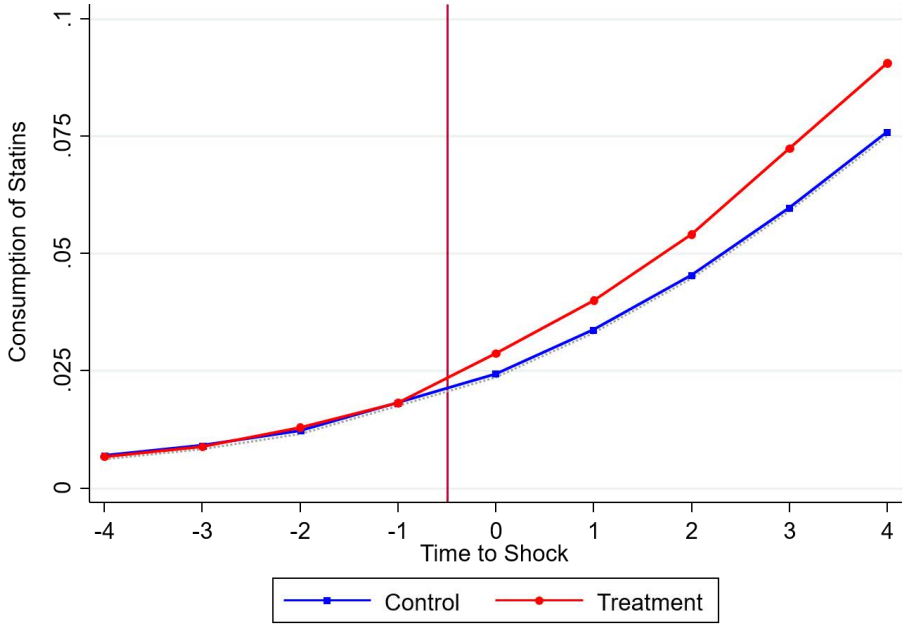
Yakusheva, O., K. A. Kapinos, and D. Eisenberg (2014). Estimating heterogeneous and hierarchical peer effects on body weight using roommate assignments as a natural experiment. *Journal of Human Resources 49*(1), 234–261.

Zisook, S., S. R. Shuchter, and M. Mulvihill (1990). Alcohol, cigarette, and medication use during the first year of widowhood. *Psychiatric Annals 20*(6), 318–326.

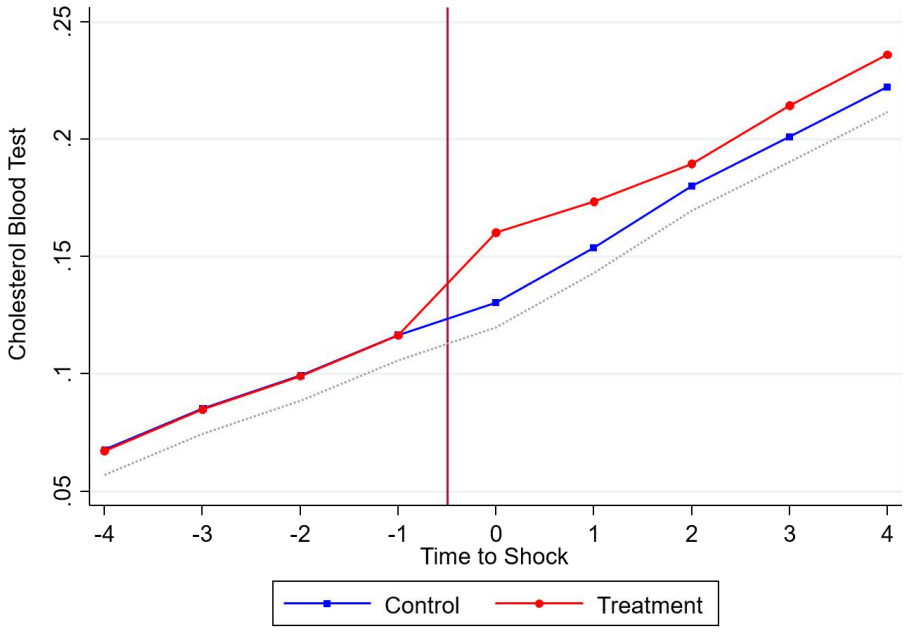
Zwane, A. P., J. Zinman, E. Van Dusen, W. Pariente, C. Null, E. Miguel, M. Kremer, D. S. Karlan, R. Hornbeck, X. Giné, et al. (2011). Being surveyed can change later behavior and related parameter estimates. *Proceedings of the National Academy of Sciences 108*(5), 1821–1826.

Figure 1: Effects of Cardiovascular Shocks on Spousal Consumption of Preventive Care

A. Statin Consumption by Prime-Age Spouses



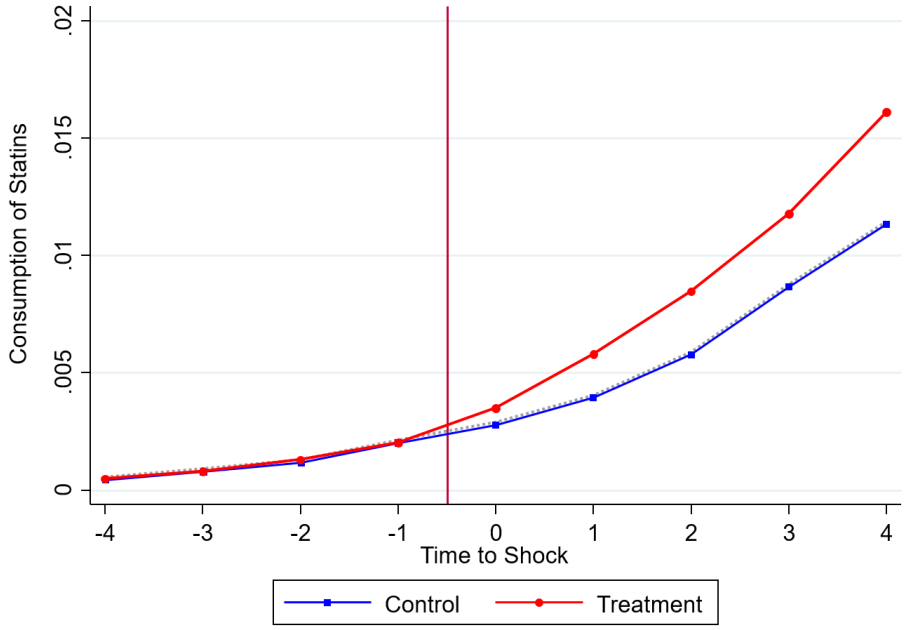
B. Cholesterol Blood Tests



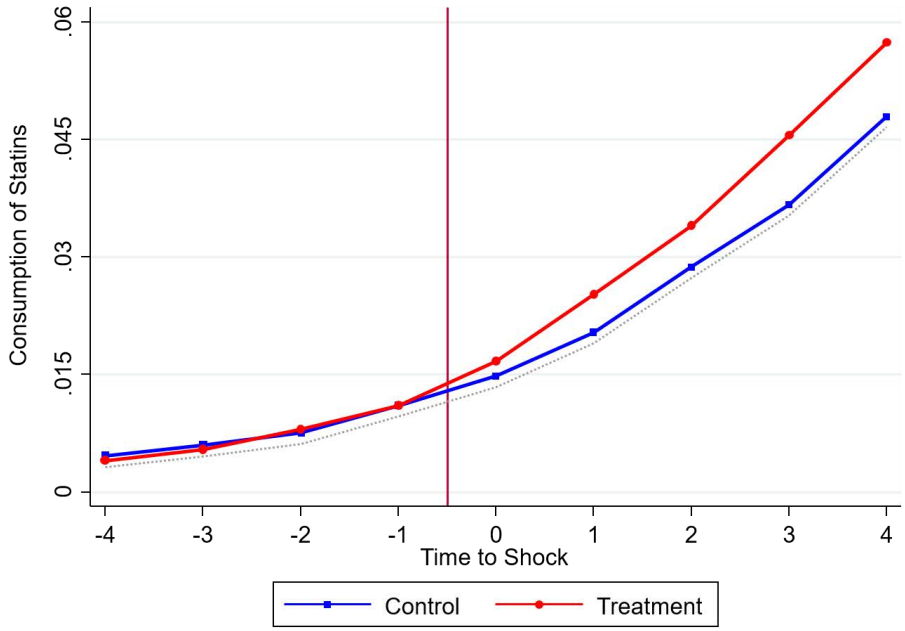
Notes: These figures plot changes in consumption of preventive care by spouses in response to family cardiovascular shocks. The structure of this and subsequent figures is as follows. The x-axis denotes time with respect to the shock, normalized to period 0. For the treatment group, period 0 is when the actual shock occurs; for the control group period 0 is when a "placebo" shock occurs (while their actual shock occurs in period 5). The dashed gray line plots the behavior of the control group. To ease the comparison of trends, from which the treatment effect is identified, we normalize the level of the control group's outcome to the pre-shock level of the treatment group's outcome (in period $t = -1$). This normalized counterfactual is displayed by the blue line and squares. The red line and circles plot the behavior of the treatment group.

Figure 2: Effects of Cardiovascular Shocks on Adult Children's Consumption of Preventive Care

A. Statin Consumption by Younger Children (Ages 25-40)



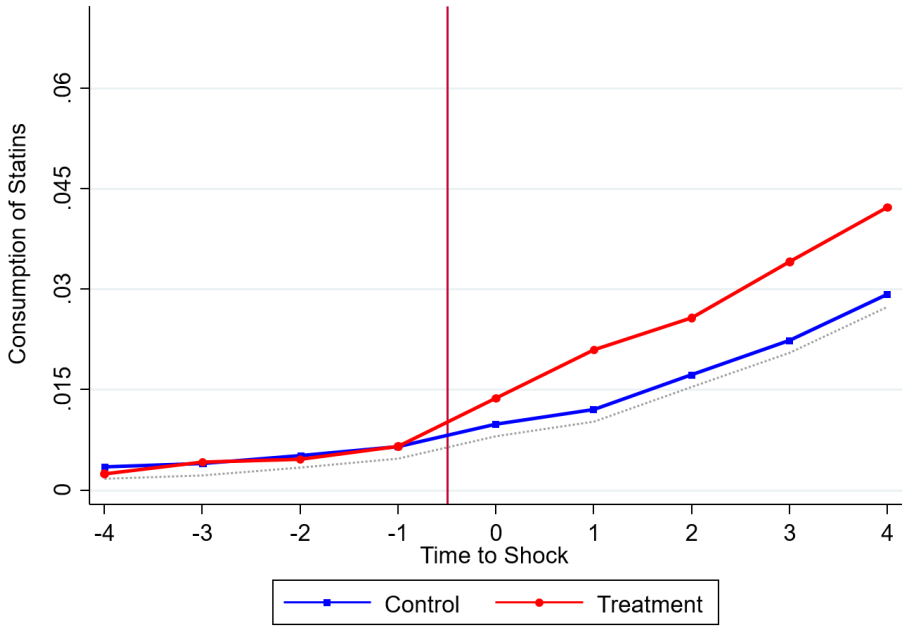
B. Statin Consumption by Older Children (Ages 40-65)



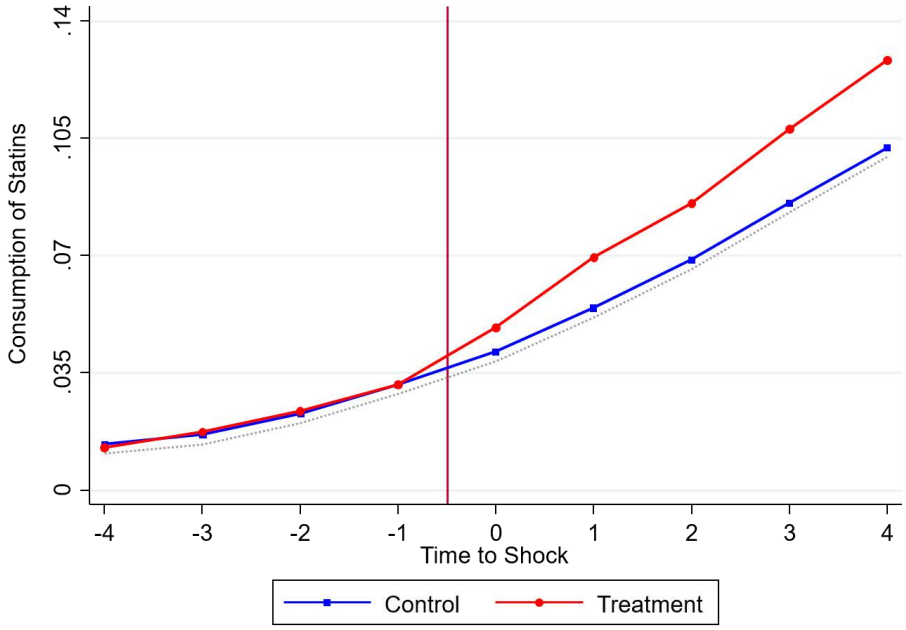
Notes: These figures plot changes in consumption of preventive care by adult children in response to family cardiovascular shocks. The figures are constructed as described in the notes of Figure 1.

Figure 3: Effects of Cardiovascular Shocks on Siblings' Consumption of Preventive Care

A. Statin Consumption by Younger Siblings (Ages 25-40)



B. Statin Consumption by Older Siblings (Ages 40-65)

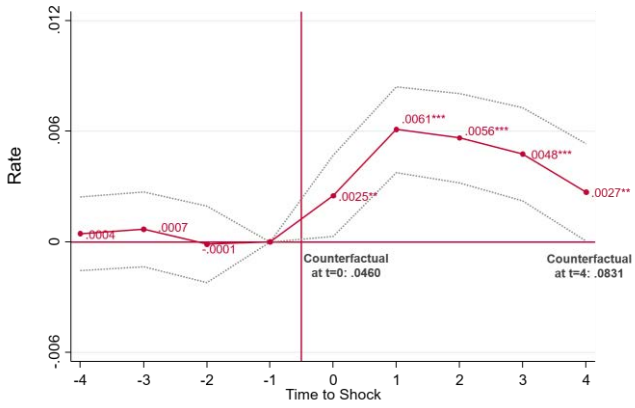


Notes: These figures plot changes in consumption of preventive care by adult siblings in response to family cardiovascular shocks. The figures are constructed as described in the notes of Figure 1.

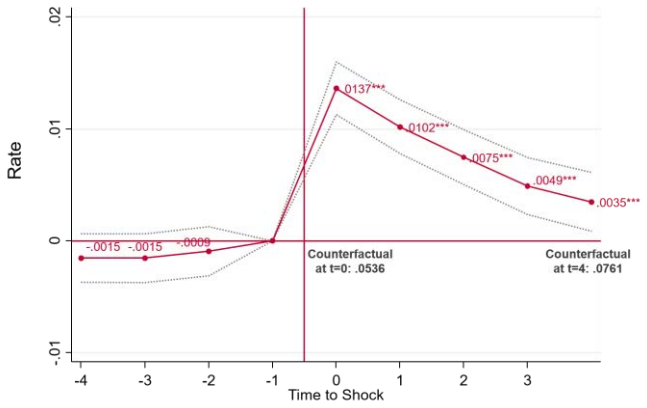
Figure 4: Effects of Fatal Shocks on Spousal Health Behaviors

Increased Awareness of Health

A. Hospital Medical Observation for Conditions that Are Ruled Out

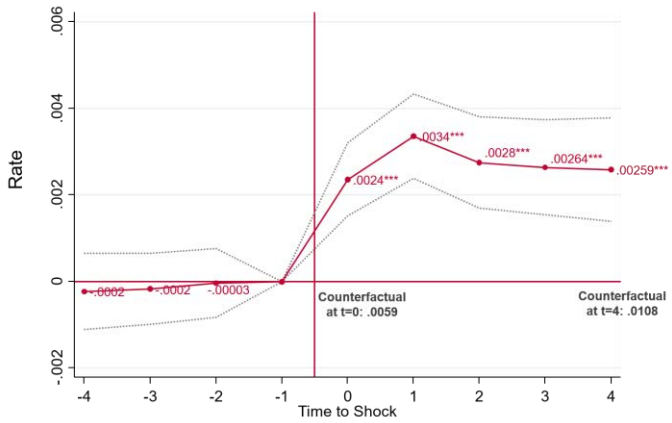


B. Non-Hospital Urgent Care Contacts

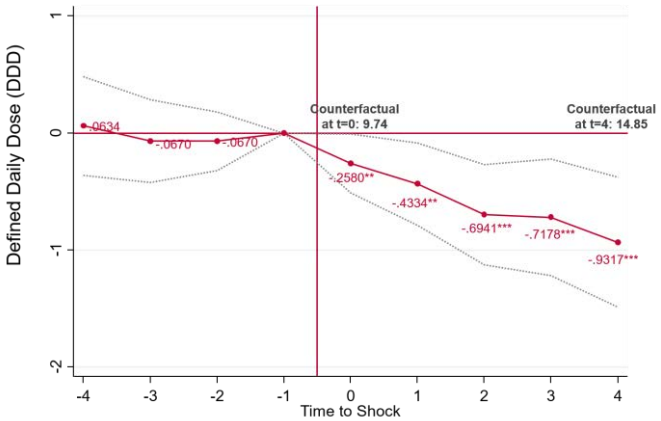


Declines in Harmful Behaviors and Medication

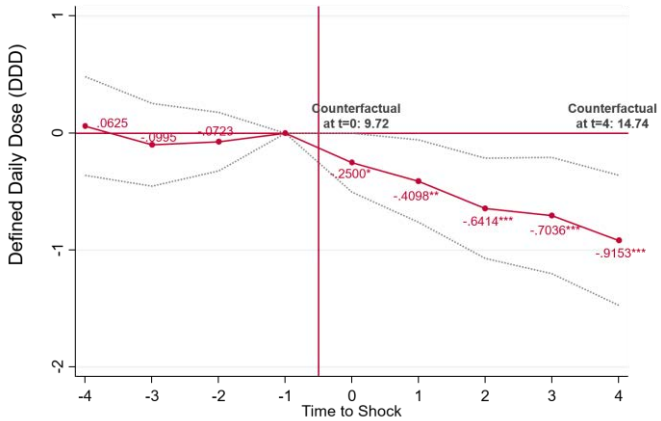
C. Medication to Treat Chronic Dependence (Smoking/Alcohol)



D. Opioid Dosage



E. Opioid Dosage – Excluding Events with Prescription Opioid Poisoning as Cause of Death



Notes: These figures display changes in health behaviors in response to fatal spousal shocks by plotting the dynamic differences-in-differences estimator of equation (2). Specifically, the figures plot the δ_r coefficient estimates along with their 95-percent confidence intervals. In each panel, we also indicate the counterfactual outcome levels for period $t = 0$ (the beginning of the analysis period) and period $t = 4$ (the end of the analysis period) based on specification (2), to gauge response magnitudes relative to underlying levels. *** p<0.01, ** p<0.05, * p<0.1.

Table 1: Dynamic Family Effects of Cardiovascular Shocks

	Spouses			Adult Children		Siblings	
	Prime-Age (Ages 25-55)	Older (Ages 55-85)	Cholesterol Testing	Younger (Ages 25-40)	Older (Ages 40-65)	Younger (Ages 25-40)	Older (Ages 40-65)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Time to Shock:							
-4	.0005	-.0007	-.0014	.0001	-.0000	-.0011	.0001
	(.0010)	(.0016)	(.0044)	(.0002)	(.0007)	(.0011)	(.00220
-3	.0003	-.0006	-.0011	.0001	-.0001	.0001	.0014
	(.0010)	(.0014)	(.0046)	(.0002)	(.0006)	(.0010)	(.0020)
-2	.0010	-.0007	-.0006	.0002	.0007	-.0006	.0009
	(.0008)	(.0011)	(.0045)	(.0002)	(.0005)	(.0009)	(.0016)
-1	0	0	0	0	0	0	0
	0	0	0	0	0	0	0
0	.0039***	.0023*	.0303***	.0007***	.0015**	.0037***	.0067***
	(.0010)	(.0012)	(.0050)	(.0002)	(.0006)	(.0013)	(.0021)
1	.0051***	.0093***	.0205***	.0017***	.0043***	.0087***	.0145***
	(.0013)	(.0018)	(.0051)	(.0003)	(.0009)	(.0018)	(.0030)
2	.0070***	.0109***	.0110**	.0024***	.0043***	.0079***	.0165***
	(.0017)	(.0022)	(.0054)	(.0004)	(.0011)	(.0021)	(.0036)
3	.0101***	.0104***	.0157***	.0028***	.0077***	.0113***	.0213***
	(.0020)	(.0025)	(.0057)	(.0005)	(.0014)	(.0025)	(.0041)
4	.0117***	.0123***	.0172***	.0043***	.0080***	.0123***	.0249***
	(.0023)	(.0028)	(.0059)	(.0006)	(.0016)	(.0028)	(.0045)
Counterfactual at t=4	.0786	.2284		.0118	.0493	.0302	.1035
Percent Change	14.90%	5.40%		36.44%	16.23%	40.73%	24.06%
Counterfactual at t=0			.1031				
Percent Change			29.4%				
Number of Obs.	441,720	667,980	214,793	1,179,387	647,667	166,689	157,491
Number of Clusters	49,080	74,220	23,866	75,759	45,380	14,001	13,009

Notes: This table reports the dynamic differences-in-differences estimates for the evolution of household responses using specification (2). It displays estimates for the δ_r parameter vector of the interaction between the treatment indicator and the indicators for time with respect to the shock from -4 to +4, where the baseline period is -1. In all columns the outcome variable is an indicator for statin consumption, besides column 3 where the outcome is an indicator for cholesterol blood testing. We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report robust standard errors clustered at the household by experimental-group level. *** p<0.01, ** p<0.05, * p<0.1.

Table 2: Heterogeneity in Family Effects of Cardiovascular Shocks

	Spouses' Statin Consumption	Spouses' Statin Consumption by Previously Tested		Adult Children's Statin Consumption
	(1)	(2)		(3)
Treat x Post	.0043**	0.0178**	Treat x Post x	-.00023***
	(.0020)	(0.0080)	Parent's Age	(.00008)
Treat x Post x	.0108***		Treat x Post x	.00059***
High Risk	(.0030)		Own Age	(.0001)
Number of Obs.	715,692	45,787		1,548,616
Number of Clusters	119,282	6,541		97,265

Notes: This table studies the heterogeneity in family responses to cardiovascular shocks along different dimensions. Column 1 estimates equation (4) and analyzes how spouses' responses in statin consumption vary by whether the spouse's own predicted cardiovascular risk is above or below the median. Column 2 estimates equation (3) and analyzes statin consumption responses by spouses whose cholesterol levels had been already tested for in the pre-shock periods. In column 3 we study whether children whose parents were younger at the time they experienced the cardiovascular shock are also more prone to increase their consumption of preventive care. Specifically, we estimate equation (4), where we interact the treatment effect with both the child's own age and the parent's age at the time of the parental shock. We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report robust standard errors clustered at the household by experimental-group level. *** p<0.01, ** p<0.05, * p<0.1.

Table 3: Mean Effects of Cardiovascular Shocks on Distant Family Circles

	Stepchildren	Biological Children Comparable to Column 1	Children In-Law	Biological Children Married to Sample of Column 2	Children In-Law by Distance		
					Subsample Mean	By Median Distance	By 25th Percentile
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Treat x Post	.0025**	.0041***	.0011**	.0045***	.0012**		
	(.0010)	(.0004)	(.0005)	(.0005)	(.0006)		
Further from						.0006	.0009
Parents In-Law						(.0007)	(.0012)
Closer to						.0021**	.0025**
Parents In-law						(.0009)	(.0011)
Baseline	.0226	.0219	.0297	0.0241			
Percent change	11%	19%	4%	19%			
Number of Obs.	280,196	1,822,954	1,206,065	1,206,065	1,041,215	1,041,215	508,893
Number of Clusters	40,028	260,422	172,295	172,295	148,745	148,745	72,699

Notes: This table reports mean differences-in-differences estimates for family members' responses to cardiovascular shocks. Columns 1 to 4 estimate equation (3) for different family circles. Columns 5 to 7 study how the spillover to sons and daughters in-law varies by distance. Column 5 replicates the overall average effect on in-laws from column 3 but for the sample of families for whom we have non-missing data on residence. In column 6 we divide the sample into children in-law who live closer to or further from their parents in-law using the median distance, and report the average effect for each sub-sample split calculated using a regression of specification (4). Cutting the sample further, column 7 includes only those whose distance from their in-laws is shorter than the sample median, and splits the remaining sample according the 25th percentile of the distance distribution. We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report robust standard errors clustered at the household by experimental-group level. *** p<0.01, ** p<0.05, * p<0.1.

Table 4: Dynamic Effects of Cardiovascular Shocks on Distant Circles

	Stepchildren	Nearby In-Laws	Close Coworkers
	(1)	(2)	(3)
Time to Shock:			
-4	.0004	.0008	-.0033
	(.0007)	(.0008)	(.0038)
-3	.0002	.0008	-.0029
	(.0006)	(.0007)	(.0033)
-2	.0002	.0006	-.0022
	(.0005)	(.0006)	(.0027)
-1	0	0	0
	0	0	0
0	.0007	.0017**	.0054*
	(.0006)	(.0007)	(.0028)
1	.0018**	.0027***	.0121***
	(.0009)	(.0010)	(.0042)
2	.0028***	.0025**	.0108**
	(.0011)	(.0012)	(.0053)
3	.0036***	.0040***	.0129**
	(.0013)	(.0015)	(.0063)
Counterfactual at t=4	0.0216	0.0263	0.1064
Percent Change	17%	15%	12%
Number of Obs.	320,224	283,176	59,632
Number of Clusters	40,028	35,397	4,238

Notes: This table reports the dynamic differences-in-differences estimates for the evolution of responses to cardiovascular shocks by different circles of one's family and social network. Using specification (2), the table displays estimates for the δ_r parameter vector of the interaction between the treatment indicator and the indicators for time with respect to the shock, where the baseline period is -1. In all columns the outcome variable is an indicator for statin consumption. We include as controls age fixed effects, calendar year fixed effects, gender, and education. We report robust standard errors clustered at the household by experimental-group level in columns 1 and 2 and at the workplace level in column 3. *** p<0.01, ** p<0.05, * p<0.1.

Table 5: Mean Effects of Cardiovascular Shocks on Coworkers

	Close Coworkers	Distant Coworkers		
		Larger Workplaces	Large Age Gap	Different Occupation
	(1)	(2)	(3)	(4)
Treat x Post	0.0139***	-0.0002	0.0041	0.0054
	(0.0050)	(0.0029)	(0.0030)	(0.0047)
Baseline	0.1055	0.1034	0.0742	0.1038
Percent change	13%			
Number of Obs.	52,178	137,179	93,925	56,756
Number of Clusters	4,238	4,860	5,770	4,920

Notes: This table reports mean differences-in-differences estimates for coworkers' responses to cardiovascular shocks using specification (3). We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report robust standard errors clustered at the workplace level. *** p<0.01, ** p<0.05, * p<0.1.

Table 6: Condition-Specific Preventive Care following Fatal Shocks

	Spousal Statin Use when Cause of Death is Cardiovascular	Children Statin Use when Cause of Death is Cardiovascular	Spouse's Diagnostic Radiology when Cause of Death is Cancer	Spouse's Hospital Contact with Family Code when C.o.d is Cancer
	(1)	(2)	(3)	(4)
Time to Shock:				
-4	.0021	-.0007	-.5069	-.0000
	(.0020)	(.0006)	(.5222)	(.0001)
-3	.0001	-.0006	.1728	-.0001
	(.0018)	(.0005)	(.5213)	(.0001)
-2	.0013	-.0002	-.2273	-.0000
	(.0014)	(.0004)	(.5141)	(.0001)
-1	0	0	0	0
	0	0	0	0
0	.0036**	.0020***	.6333	-.0000
	(.0015)	(.0005)	(.5316)	(.0001)
1	.0047**	.0020***	1.5464***	.0002**
	(.0020)	(.0007)	(.5472)	(.0001)
2	.0050**	.0033***	1.2307**	.0000
	(.0023)	(.0009)	(.5568)	(.0001)
3	.0062**	.0049***	.9273	-.0000
	(.0025)	(.0010)	(.5676)	(.0001)
4	.0071***	.0066***	.5947	.0001
	(.0027)	(.0012)	(.5851)	(.0001)
Baseline Levels	.0769	.0495	12.72	.00017
Number of Obs.	889,837	2,922,141	2,382,999	1,524,096
Number of Clusters	126,816	167,586	303,192	213,925
Households with Condition	13,589	38,076	107,565	76,101

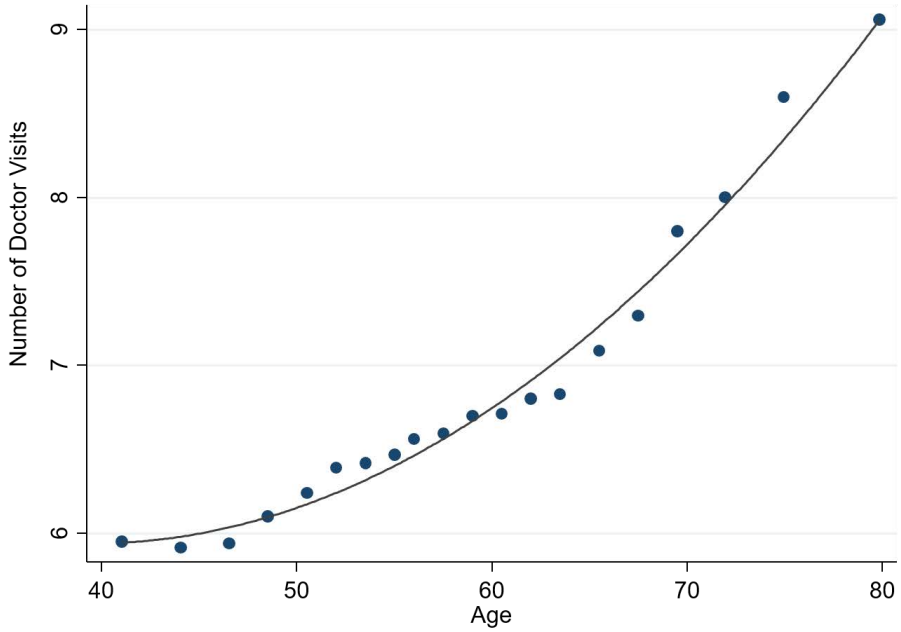
Notes: This table reports the dynamic differences-in-differences estimates for the evolution of household responses around fatal shocks using specification (5). The regressions are estimated on treated households only, where we divide the sample by cause of death. The table displays estimates for the δ_r parameter vector of the interaction between cause of death indicators and the indicators for time with respect to the shock from -4 to +4, where the baseline period is -1. As such, for each preventive care outcome that we study, the estimates display how the utilization of this preventive care by individuals whose family member died of some disease *x* compares to the utilization by those whose family member died of any other cause. Column 1 studies statin consumption of individuals whose spouse died of cardiovascular disease compared to those whose spouse died of any other cause. Column 2 provides a similar analysis of statin consumption by adult children around parental death. Column 3 studies healthcare costs associated with visits to diagnostic radiologists, comparing individuals whose spouse died of cancer to those whose spouse died of any other cause. Column 4 provides a similar analysis but where the outcome variable is an indicator for an outpatient hospital contact for the reason of having a family member with history of cancer. We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report robust standard errors clustered at the household by experimental-group level. *** p<0.01, ** p<0.05, * p<0.1.

Table 7: Spousal Health Behaviors following Fatal Shocks

	Smoking Cessation by Lung Cancer	Smoking Cessation by (Non-Lung) Cancer	Diagnostic Radiology by Female Cancer	Cancer-Related Hospital Contact by Female Cancer	Cross-Condition Responses	
					Diagnostic Radiology by Cardiovascular	Statins by Cancer
	(1)	(2)	(3)	(4)	(5)	(6)
C.o.d x Post	.0006**	.0002	1.5139**	0.0003	0.1065	-0.0045***
	(.0003)	(.0002)	(0.7546)	(0.0015)	(0.6693)	(0.0015)
Counterfactual	.0025	.0021	7.8633	0.0252	12.0123	0.1312
Number of Obs.	583,585	768,480	311,810	297,927	454,129	746,573
Number of Clusters	84,301	109,912	60,056	50,314	84,893	105,677
Households with Condition	12,599	34,705	12,889	9,309		

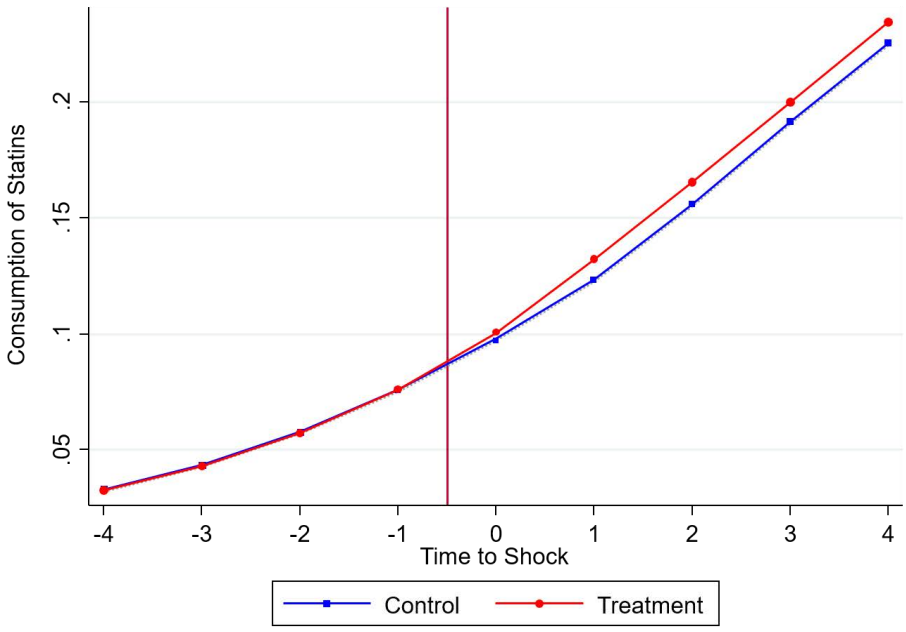
Notes: This table reports mean differences-in-differences estimates for spousal responses to fatal shocks using different specifications of equation (6). Column 1 studies the consumption of medication for smoking cessation by individuals whose spouse died of lung cancer compared to those whose spouse died of any non-cancer cause. Column 2 studies the consumption of this medication by individuals whose spouse died of non-lung cancer compared to those whose spouse died of any non-cancer cause. Column 3 studies husbands' expenditure on diagnostic radiology, comparing those whose wife died of female cancers (ovarian, cervical, or breast cancer) to those whose wife died of any non-cancer cause. Column 4 provides a similar analysis but where the outcome variable is husbands' incidence of cancer diagnoses, measured as an indicator for husbands' contacts with inpatient or outpatient hospital departments for any cancer-related reason (beyond potential diagnostic tests). Columns 5 and 6 study cross-condition responses. Column 5 studies expenditures on diagnostic radiology by individuals whose spouse died of cardiovascular disease compared to those whose spouse died of other causes (excluding cancer). Column 6 studies statin consumption by individuals whose spouse died of cancer compared to those whose spouse died of other causes (excluding cardiovascular disease). We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report robust standard errors clustered at the household level. *** p<0.01, ** p<0.05, * p<0.1.

Appendix Figure 1: Interactions with the Medical System by Age



Notes: This figure plots averages for the number of doctor visits per individual within a year as a function of age. The blue dots represent raw means for each of the equal-sized age bin in the range of 40 to 80; the solid line represents the best quadratic fit (using the individual-level data).

Appendix Figure 2: Effects of Cardiovascular Shocks on Older Spouses' Consumption of Preventive Care



Notes: This figure plots changes in consumption of preventive care by older spouses (ages 55-85) in response to family cardiovascular shocks. The figure is constructed as described in the notes of Figure 1.

Appendix Table 1: Summary Statistics of Analysis Sample

		Year	Age	Education (Months)	Percent Female	Number of Individuals
Non-Fatal Severe Health Shocks						
Spouses						
Younger (25-55)	Treatment	2002	46.7	155.4	.72	20,381
	Control	2002	45.8	156.6	.704	28,699
Older (55-85)	Treatment	2002.2	65.7	136	.64	37,828
	Control	2002.1	64.6	139	.60	36,392
Biological Children						
Younger (25-40)	Treatment	2002	33.4	169	.492	63,323
	Control	2001.9	33.1	170	.492	68,437
Older (40-65)	Treatment	2002.4	44.6	166.3	.46	39,783
	Control	2002.3	44.14	167.4	.463	32,926
Siblings						
Younger (25-40)	Treatment	2002.7	34.76	159.8	.497	6,172
	Control	2002.5	34.50	159.62	.488	11,809
Older (40-65)	Treatment	2003.5	45.5	155.7	.472	7,356
	Control	2003.4	45	156.2	.4735	10,143
Non-Biological Children	Treatment	2002.8	35.7	162.4	.496	19,254
	Control	200.7	34.5	163.3	.492	20,774
Sons and Daughters In-Law	Treatment	2002.6	38.7	168.7	.495	86,874
	Control	2002.5	37.7	169.4	.489	85,421
Coworkers	Treatment	2002.2	48.2	161.5	.37	63,122
	Control	2002.1	48.1	161.6	.38	83,087
Fatal Shocks						
Spouses	Treatment	1996.5	63.2	118.3	.72	255,994
	Control	1996.4	62.4	119.9	.70	341,329
Biological Children	Treatment	2003.7	41.16	166.6	.47	324,594
	Control	2003.7	40.5	167.5	.473	395,861

Notes: This table presents means of key variables in our analysis sample based on data from period t = -1. For each event, the treatment group is comprised of individuals who experienced a shock in different years, to which we match as control groups individuals from the same cohorts that experienced the same shock but five years later (Δ=5). To construct our sample, we start from the universe of households in which an individual experienced one of the shocks that we consider between the years 1985 and 2011, where all of our matches across household members are based on the pre-shock period t = -1. Our primary sample of non-fatal health shocks is comprised of all households in which one individual experienced a heart attack or a stroke (for the first time) and survived for the four-year analysis horizon. The main family circles that we study are spouses and adult biological children. Our sample of spouses is based on all married and cohabiting couples among families in which one spouse experienced a shock. The registers provide such spousal matches across all individuals born between 1910 and 1970, who are the cohorts covered by our data. For children, the registers provide matches to biological parents for individuals born after 1960. Our sample of adult biological children is based on these matches. The sample of siblings is also based on parental linkages, such that siblings are defined as individuals who share biological parents. For the more distant circles of family members and peers, we increase the statistical power by reducing the data requirement to include individuals who survived for at least three after the cardiovascular shock (instead of four). Stepchildren are defined as any child with a non-biological link to the individual that experienced the shock. We establish these links by combining the spousal linkages and the biological parent linkages. Specifically, we define as a "stepchild" any person for whom neither biological parent is the individual that experienced the shock but for whom one biological parent is the spouse of that individual. Sons and daughters in-law are simply the spouses of the biological children. Finally, we proxy for peers using coworkers based on matched employer-employee register data, where we define workplaces using physical establishment units. To approximate peers with whom individuals are more likely to interact, we focus on "close" coworkers in the following way. From our sample of individuals who experience a health shock we identify those who, during the pre-shock periods from -4 to -1, have worked in smaller workplaces (with number of employees equal or lower than the sample's 25th percentile of approximately 20), in order to reduce the measurement error in proxying for peers using coworkers. We then focus on coworkers who have been employed in a similar occupation class, and who are close to the individual that experienced the shock in terms of age (with an age gap of 5 years or less). We exclude from this sample any coworker who is also a family member. Lastly, our secondary sample of fatal shocks includes all families in which one member died between 1985 and 2011. For these shocks we study spouses and biological children, whose respective samples are constructed in the same way as described above.

Appendix Table 2: Heterogeneity in Spousal Responses to Cardiovascular Shocks

	Spouse's Cholesterol Testing	Spouse's Statin Consumption for Subsample from Column 1	Spouse's Statin Consumption	Spouse's Statin Consumption	Spouse's Statin Consumption by Previously Tested
	(1)	(2)	(3)	(4)	(5)
Treat x Post	.0142***	.0019			.0196**
	(.0043)	(.0030)			(.0096)
Treat x Post x	.0032	.0097**		.0132***	
High Risk	(.0055)	(.0047)		(.0027)	
Treat x Post x			-1.4504***	-.9216**	
Risk Gap			(.4130)	(.4453)	
Number of Obs.	231,519	202,199	930,448	930,448	34,041
Number of Clusters	28,940	28,940	116,306	116,306	4,863

Notes: This table studies the heterogeneity in spousal responses to cardiovascular shocks along different dimensions. Column 1 estimates equation (4) and analyzes how spouses' responses in cholesterol testing vary by whether the spouse's own predicted cardiovascular risk is above or below the median. It includes residents of Greater Copenhagen for whom data on blood tests are available. Column 2 provides a similar analysis for this sub-sample but where the outcome variable is spouses' statin consumption. Columns 3 and 4 estimate equation (4) to study how spousal responses in statin consumption vary by the similarity of their predicted baseline cardiovascular risk to that of their partners who experience the shock. Column 3 interacts the treatment effect with this risk gap, and column 4 also adds an interaction with an indicator for whether the spouse's own predicted risk is above or below the median. Column 5 estimates equation (3) and analyzes statin consumption responses by spouses whose cholesterol levels had been already tested for in periods -2 or -1. We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report robust standard errors clustered at the household by experimental-group level. *** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 3: Robustness for Closeness of Peers

Age Gap					
Max. Years of Age Gap:	7	6	***5***	4	3
Treat x Post	.0115***	.0123***	.0139***	.0151***	.0115*
	(.0044)	(.0047)	(.0050)	(.0054)	(.0060)
Number of Obs.	68,250	60,326	52,178	43,365	34,685
Number of Clusters	4,898	4,588	4,238	3,808	3,264
Workplace Size					
Max. Number of Employees:	24	22	***20***	18	16
Treat x Post	.0094**	.0105**	.0139***	.0112**	.0153**
	(.0044)	(.0047)	(.0050)	(.0054)	(.0061)
Number of Obs.	67,648	59,850	52,178	44,072	36,575
Number of Clusters	5,059	4,663	4,238	3,764	3,294

Notes: This table reports mean differences-in-differences estimates for coworkers' responses to cardiovascular shocks using specification (3). The table provides as robustness checks estimations that perturb the thresholds of age gap (5 years) and workplace size (20 employees) in our definition of "close" coworkers, which we chose to balance between sample size and closeness of peers. The upper panel perturbs the age gap between coworkers and the person that experiences the shock around our choice of 5 years; and the lower panel perturbs the workplace size around our choice of 20 employees (the sample's 25th percentile). We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report robust standard errors clustered at the workplace level. *** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 4: Family Effects of Cardiovascular Shocks—Different Physicians

	Spouses					Adult Children				
	Different Matched GP	Number of Patients Overlapped		Share of Patients Overlapped		Different Matched GP	Number of Patients Overlapped		Share of Patients Overlapped	
		Less than 50	Less than 20	Less than 0.05	Less than 0.02		Less than 50	Less than 20	Less than 0.05	Less than 0.02
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Time to Shock:										
-4	-.0019	-.0013	-.0012	-.0022	-.0021	-.0000	-.0000	-.0000	-.0000	-.0000
	(.0019)	(.0020)	(.0022)	(.0020)	(.0021)	(.0003)	(.0003)	(.0003)	(.0003)	(.0003)
-3	-.0016	-.0012	-.0015	-.0017	-.0018	-.0001	-.0001	-.0001	-.0000	-.0001
	(.0018)	(.0019)	(.0020)	(.0018)	(.0019)	(.0003)	(.0003)	(.0003)	(.0003)	(.0003)
-2	-.0017	-.0011	-.0011	-.0012	-.0009	.0002	.0002	.0002	.0003	.0002
	(.0015)	(.0016)	(.0017)	(.0015)	(.0019)	(.0003)	(.0003)	(.0003)	(.0003)	(.0003)
-1	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0	0	0
0	.0039**	.0033*	.0037*	.0035*	.0034*	.0011***	.0011***	.0011***	.0010***	.0011***
	(.0017)	(.0018)	(.0019)	(.0018)	(.0019)	(.0003)	(.0003)	(.0003)	(.0003)	(.0003)
1	.0068***	.0068***	.0067**	.0063**	.0059**	.0029***	.0027***	.0028***	.0027***	.0028***
	(.0024)	(.0025)	(.0027)	(.0025)	(.0026)	(.0005)	(.0005)	(.0005)	(.0005)	(.0005)
2	.0104***	.0100***	.0103***	.0087***	.0083***	.0036***	.0034***	.0034***	.0035***	.0035***
	(.0029)	(.0031)	(.0033)	(.0030)	(.0032)	(.0006)	(.0006)	(.0006)	(.0006)	(.0006)
3	.0140***	.0143***	.0151***	.0123***	.0144***	.0049***	.0047***	.0048***	.0049***	.0049***
	(.0034)	(.0036)	(.0039)	(.0035)	(.0037)	(.0007)	(.0007)	(.0007)	(.0007)	(.0007)
4	.0107***	.0107***	.0122***	.0095**	.0097**	.0063***	.0061***	.0062***	.0063***	.0062***
	(.0038)	(.0040)	(.0043)	(.0040)	(.0042)	(.0008)	(.0008)	(.0008)	(.0008)	(.0008)
Number of Obs.	238,779	204,201	176,040	214,515	188,865	1,296,423	1,236,303	1,182,555	1,254,375	1,207,953
Number of Clusters	26,531	22,689	19,560	23,835	20,985	83,136	80,740	78,387	81,351	79,405

Notes: This table reports the dynamic differences-in-differences estimates for the evolution of household responses using specification (2). It displays estimates for the δ_r parameter vector of the interaction between the treatment indicator and the indicators for time with respect to the shock from -4 to +4, where the baseline period is -1. In all columns the outcome variable is an indicator for statin consumption. In this table, we analyze only households in which the family members whose behaviors we study do not share the same doctor with the person the experiences the cardiovascular shock. We do so to study whether the spillover effects can be explained by supply-side responses in the form of family physicians who aggregate information across the different members of the household. The data allow matching patients to their general practitioner (GP) since any service provided to a patient by a GP documents the GP's identifier and whether he or she is the patient's assigned GP. The analysis of family members with different matched GPs is reported in column 1 for spouses and in column 6 for children. As the different physicians may share clinics which could lead to information flows across doctors, we further guarantee the separation of providers by studying only physicians whose patient overlap is minimal. Specifically, we exclude observations for whom the GP of the person that experienced the shock treated a non-negligible portion of the patients of the GP that is assigned to the family member. Columns 2-3 and 7-8 include only observations where patient overlap falls below a threshold level (with an average of 1,279 patients per GP), and column 4-5 and 9-10 include only observations where patient overlap falls below a threshold share. Overall, we find similar-magnitude effects among these households so that the spillover is unlikely attributed to aggregation of information by the family doctor. The regressions include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report robust standard errors clustered at the household by experimental-group level. *** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 5: Family Members' Health Behaviors following Fatal Shocks

	Spouses'	Adult Children				
	Dependence Medication when C.o.d is Autoimmune Disease	Hospital Medical Observation for Conditions that Are Ruled Out	Non-Hospital Urgent Care Contacts	Medication to Treat Chronic Dependence (Smoking/Alcohol)	Statin Use when Cause of Death is Cardiovascular	Diagnostic Radiology when Cause of Death is Cancer
	(1)	(2)	(3)	(4)	(5)	(6)
Treat x Post	.0067**	.0019***	.0015**	.0006***		
	(.0031)	(.0004)	(.0006)	(.0002)		
C.o.d x Post					.0047***	.7401**
					(.0008)	(.2932)
Counterfactual	.0047	.0634	.0617	.0121		
Baseline					.0363	12.12
Number of Obs.	18,381	6,276,868	3,002,647	5,764,516	2,597,547	2,612,139
Number of Clusters	2,650	306,841	188,719	294,943	167,586	228,835

Notes: This table reports mean differences-in-differences estimates for family members' responses to fatal shocks. In column 1, using equation (3), we estimate the consumption of medication that treats nicotine or alcohol dependence by individuals whose spouse's cause of death was autoimmune disease. Columns 2 to 4 estimate equation (3) for different behavioral outcomes of adult children, which are indicated at the top of each column. Columns 5 and 6 estimate specifications of equation (6) for adult children. Column 5 compares statin consumption by individuals whose parent died of cardiovascular disease to the behavior of those whose parent died of any other cause; column 6 compares expenditure on diagnostic radiology by individuals whose parent died of cancer to those whose parent died of any other cause. We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report robust standard errors clustered at the household level. *** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 6: Studies on Health Behaviors' Formation and Determinants in Developed Countries

Context	Applications
Self-control, commitment mechanisms, and financial incentives	DellaVigna and Malmendier (2006), Finkelstein et al. (2007), Volpp et al. (2008), Charness and Gneezy (2009), Volpp et al. (2009), Gine et al. (2010), Gneezy et al. (2011), John et al. (2011), Cawley and Price (2013), Just and Price (2013), Milkman et al. (2013), Acland and Levy (2015), Babcock et al. (2015), Bhattacharya et al. (2015), Halpern et al. (2015), Royer et al. (2015), Patel et al. (2016), Loewenstein et al. (2016), Carrera et al. (2017), Mochon et al. (2017).
Health insurance coverage and features	Choudhry et al. (2011), Adams et al. (2013), Bitler and Carpenter (2016), Cabral and Cullen (2017), Cotti et al. (2017), Courtemanche et al. (2017), Maclean et al. (2017), Simon et al. (2017).
Medical information, disease outbreaks, advertisements, and the media	Cram and Cowen (2003), Sanchez et al. (2005), Adda (2007), Avery et al. (2007), Oster et al. (2013a,b), Oster (2015), Desai and Jena (2016), Oster (2016), Darden (2017)
Workplace policies and government regulations	Evans et al. (1999), Variyam and Cawley (2006), Downs et al. (2009), Wisdom et al. (2010), Bollinger et al. (2011), Downs et al. (2013), Dave et al. (2017), Kenkel et al. (2017)
Doctor practices	Koulayev et al. (2017)
Message framing and information provision	Detweiler et al. (1999), Farrell et al. (2001), Schneider et al. (2001), Rothman et al. (2006), Toll et al. (2007), Parkes et al. (2008), Hoffner and Ye (2009), May et al. (2010), Volandes et al. (2009), van der Linden et al. (2015)
Defaults, opt-ins, and opt-outs	Chapman et al. (2010), Halpern et al. (2013)
Nudging, convenience, and reminders	Milkman et al. (2011), Hanks et al. (2012), Beshears et al. (2016)
Sales and excise taxes	Adda and Cornaglia (2006), Chetty, Looney and Kroft (2009), Goldin and Homonoff (2013)

Appendix References

Acland, D. and M. R. Levy (2015). Naiveté, projection bias, and habit formation in gym attendance. *Management Science 61*(1), 146–160.

Adams, E. K., S. Markowitz, P. M. Dietz, and V. T. Tong (2013). Expansion of medicaid covered smoking cessation services: Maternal smoking and birth outcomes. *Medicare & Medicaid Research Review 3*(3).

Adda, J. (2007). Behavior towards health risks: An empirical study using the mad cow crisis as an experiment. *Journal of Risk and Uncertainty 35*(3), 285–305.

Adda, J. and F. Cornaglia (2006). Taxes, cigarette consumption, and smoking intensity. *The American Economic Review*, 1013–1028.

Avery, R., D. Kenkel, D. R. Lillard, and A. Mathios (2007). Private profits and public health: Does advertising of smoking cessation products encourage smokers to quit? *Journal of Political Economy 115*(3), 447–481.

Babcock, P., K. Bedard, G. Charness, J. Hartman, and H. Royer (2015). Letting down the team? social effects of team incentives. *Journal of the European Economic Association 13*(5), 841–870.

Beshears, J., J. J. Choi, D. I. Laibson, B. C. Madrian, and G. I. Reynolds (2016). Vaccination rates are associated with functional proximity but not base proximity of vaccination clinics. *Medical care 54*(6), 578.

Bhattacharya, J., A. M. Garber, and J. D. Goldhaber-Fiebert (2015). Nudges in exercise commitment contracts: a randomized trial. Technical report, National Bureau of Economic Research.

Bitler, M. P. and C. S. Carpenter (2016). Health insurance mandates, mammography, and breast cancer diagnoses. *American Economic Journal: Economic Policy 8*(3), 39–68.

Bollinger, B., P. Leslie, and A. Sorensen (2011). Calorie posting in chain restaurants. *American Economic Journal: Economic Policy 3*(1), 91–128.

Cabral, M. and M. R. Cullen (2017). The effect of insurance coverage on preventive care. *Economic Inquiry*.

Carrera, M., H. Royer, M. F. Stehr, and J. R. Sydnor (2017). The structure of health incentives: Evidence from a field experiment. Technical report, National Bureau of Economic Research.

Cawley, J. and J. A. Price (2013). A case study of a workplace wellness program that offers financial incentives for weight loss. *Journal of Health Economics 32*(5), 794–803.

Chapman, G. B., M. Li, H. Colby, and H. Yoon (2010). Opting in vs opting out of influenza vaccination. *JAMA 304*(1), 43–44.

Charness, G. and U. Gneezy (2009). Incentives to exercise. *Econometrica 77*(3), 909–931.

Chetty, R., A. Looney, and K. Kroft (2009). Salience and taxation: Theory and evidence. *The American Economic Review 99*(4), 1145.

Choudhry, N. K., J. Avorn, R. J. Glynn, E. M. Antman, S. Schneeweiss, M. Toscano, L. Reisman, J. Fernandes, C. Spettell, J. L. Lee, et al. (2011). Full coverage for preventive medications after myocardial infarction. *New England Journal of Medicine 365*(22), 2088–2097.

Cotti, C. D., E. Nesson, and N. Tefft (2017). Impacts of the aca medicaid expansion on health behaviors: Evidence from household panel data.

Courtemanche, C., J. Marton, B. Ukert, A. Yelowitz, and D. Zapata (2017). Early effects of the affordable care act on health care access, risky health behaviors, and self-assessed health. Technical report, National Bureau of Economic Research.

Cram, P., A. M. Fendrick, J. Inadomi, M. E. Cowen, D. Carpenter, and S. Vijan (2003). The impact of a celebrity promotional campaign on the use of colon cancer screening: the katie couric effect. *Archives of Internal Medicine 163*(13), 1601–1605.

Darden, M. (2017). Smoking, expectations, and health: A dynamic stochastic model of lifetime smoking behavior. *Journal of Political Economy 125*(5), 000–000.

Dave, D. M., A. M. Grecu, and H. Saffer (2017). Mandatory access prescription drug monitoring programs and prescription drug abuse. Technical report, National Bureau of Economic Research.

Desai, S. and A. B. Jena (2016). Christmas 2016: Famous figures: Do celebrity endorsements matter? observational study of brca gene testing and mastectomy rates after angelina jolie's new york times editorial. *The BMJ 355*.

Detweiler, J. B., B. T. Bedell, P. Salovey, E. Pronin, and A. J. Rothman (1999). Message framing and sunscreen use: gain-framed messages motivate beach-goers. *Health Psychology 18*(2), 189.

Downs, J. S., G. Loewenstein, and J. Wisdom (2009). Strategies for promoting healthier food choices. *The American Economic Review 99*(2), 159–164.

Downs, J. S., J. Wisdom, B. Wansink, and G. Loewenstein (2013). Supplementing menu labeling with calorie recommendations to test for facilitation effects. *American Journal of Public Health 103*(9), 1604–1609.

Evans, W. N., M. . Farrelly, and E. Montgomery (1999). Do workplace smoking bans reduce smoking? *The American Economic Review 89*(4), 728–747.

Farrell, K., E. Ferguson, V. James, and K. C. Lowe (2001). Confidence in the safety of blood for transfusion: the effect of message framing. *Transfusion 41*(11), 1335–1340.

Finkelstein, E. A., L. A. Linnan, D. F. Tate, and B. E. Birken (2007). A pilot study testing the effect of different levels of financial incentives on weight loss among overweight employees. *Journal of Occupational and Environmental Medicine 49*(9), 981–989.

Giné, X., D. Karlan, and J. Zinman (2010). Put your money where your butt is: a commitment contract for smoking cessation. *American Economic Journal: Applied Economics 2*(4), 213–235.

Gneezy, U., S. Meier, and P. Rey-Biel (2011). When and why incentives (don't) work to modify behavior. *The Journal of Economic Perspectives 25*(4), 191–209.

Goldin, J. and T. Homonoff (2013). Smoke gets in your eyes: cigarette tax salience and regressivity. *American Economic Journal: Economic Policy 5*(1), 302–336.

Halpern, S. D., B. French, D. S. Small, K. Saulsgiver, M. O. Harhay, J. Audrain-McGovern, G. Loewenstein, T. A. Brennan, D. A. Asch, and K. G. Volpp (2015). Randomized trial of four financial-incentive programs for smoking cessation. *New England Journal of Medicine 372*(22), 2108–2117.

Halpern, S. D., G. Loewenstein, K. G. Volpp, E. Cooney, K. Vranas, C. M. Quill, M. S. McKenzie, M. O. Harhay, N. B. Gabler, T. Silva, et al. (2013). Default options in advance directives influence how patients set goals for end-of-life care. *Health Affairs 32*(2), 408–417.

Hanks, A. S., D. R. Just, L. E. Smith, and B. Wansink (2012). Healthy convenience: nudging students toward healthier choices in the lunchroom. *Journal of Public Health 34*(3), 370–376.

Hoffner, C. and J. Ye (2009). Young adults' responses to news about sunscreen and skin cancer: The role of framing and social comparison. *Health Communication 24*(3), 189–198.

John, L. K., G. Loewenstein, A. B. Troxel, L. Norton, J. E. Fassbender, and K. G. Volpp (2011). Financial incentives for extended weight loss: A randomized, controlled trial. *Journal of General Internal Medicine 26*(6), 621.

Just, D. R. and J. Price (2013). Using incentives to encourage healthy eating in children. *Journal of Human Resources 48*(4), 855–872.

Kenkel, D. S., S. Peng, M. F. Pesko, and H. Wang (2017). Mostly harmless regulation? electronic cigarettes, public policy and consumer welfare. Technical report, National Bureau of Economic Research.

Koulayev, S., E. Simeonova, and N. Skipper (2017). Can physicians affect patient adherence with medication? *Health Economics 26*(6), 779–794.

Loewenstein, G., J. Price, and K. Volpp (2016). Habit formation in children: Evidence from incentives for healthy eating. *Journal of Health Economics 45*, 47–54.

Maclean, J. C., M. F. Pesko, and C. Steven (2017). The effect of insurance expansions on smoking cessation medication use: Evidence from recent medicaid expansions.". Technical report, National Bureau of Economic Research.

May, R., G. H. Tofler, R. Bartrop, P. Heinrich, J. Baird, E. Jozefiak, and S. de Burgh (2010). Smoking cessation through a novel behavior modification technique. *The American Journal of Cardiology 106*(1), 44–46.

Milkman, K. L., J. Beshears, J. J. Choi, D. Laibson, and B. C. Madrian (2011). Using implementation intentions prompts to enhance influenza vaccination rates. *Proceedings of the National Academy of Sciences 108*(26), 10415–10420.

Milkman, K. L., J. A. Minson, and K. G. Volpp (2013). Holding the hunger games hostage at the gym: An evaluation of temptation bundling. *Management Science 60*(2), 283–299.

Mochon, D., J. Schwartz, J. Maroba, D. Patel, and D. Ariely (2017). Gain without pain: The extended effects of a behavioral health intervention. *Management Science 63*(1), 58–72.

Oster, E. (2015). Diabetes and diet: Behavioral response and the value of health. Technical report, National Bureau of Economic Research.

Oster, E. (2016). Does disease cause vaccination? disease outbreaks and vaccination response. Technical report, National Bureau of Economic Research.

Oster, E., I. Shoulson, and E. Dorsey (2013a). Limited life expectancy, human capital and health investments. *The American Economic Review 103*(5), 1977–2002.

Oster, E., I. Shoulson, and E. Dorsey (2013b). Optimal expectations and limited medical testing: evidence from huntington disease. *The American Economic Review 103*(2), 804–830.

Parkes, G., T. Greenhalgh, M. Griffin, and R. Dent (2008). Effect on smoking quit rate of telling patients their lung age: the step2quit randomised controlled trial. *BMJ 336*(7644), 598–600.

Patel, M. S., D. A. Asch, R. Rosin, D. S. Small, S. L. Bellamy, J. Heuer, S. Sproat, C. Hyson, N. Haff, S. M. Lee, et al. (2016). Framing financial incentives to increase physical activity among overweight and obese adultsa randomized, controlled trialfinancial incentives for physical activity in overweight and obese adults. *Annals of Internal Medicine 164*(6), 385–394.

Rothman, A. J., R. D. Bartels, J. Wlaschin, and P. Salovey (2006). The strategic use of gain-and loss-framed messages to promote healthy behavior: How theory can inform practice. *Journal of Communication 56*(s1).

Royer, H., M. Stehr, and J. Sydnor (2015). Incentives, commitments, and habit formation in exercise: evidence from a field experiment with workers at a fortune-500 company. *American Economic Journal: Applied Economics 7*(3), 51–84.

Sánchez, C. D., L. K. Newby, D. K. McGuire, V. Hasselblad, M. N. Feinglos, and E. M. Ohman (2005). Diabetes-related knowledge, atherosclerotic risk factor control, and outcomes in acute coronary syndromes. *The American Journal of Cardiology 95*(11), 1290–1294.

Schneider, T. R., P. Salovey, A. M. Apanovitch, J. Pizarro, D. McCarthy, J. Zullo, and A. J. Rothman (2001). The effects of message framing and ethnic targeting on mammography use among low-income women. *Health Psychology 20*(4), 256.

Simon, K., A. Soni, and J. Cawley (2017). The impact of health insurance on preventive care and health behaviors: evidence from the first two years of the aca medicaid expansions. *Journal of Policy Analysis and Management 36*(2), 390–417.

Toll, B. A., S. S. O'malley, N. A. Katulak, R. Wu, J. A. Dubin, A. Latimer, B. Meandzija, T. P. George, P. Jatlow, J. L. Cooney, et al. (2007). Comparing gain-and loss-framed messages for smoking cessation with sustained-release bupropion: a randomized controlled trial. *Psychology of Addictive Behaviors 21*(4), 534.

van der Linden, S. L., C. E. Clarke, and E. W. Maibach (2015). Highlighting consensus among medical scientists increases public support for vaccines: evidence from a randomized experiment. *BMC public health 15*(1), 1207.

Variyam, J. N. and J. Cawley (2006). Nutrition labels and obesity. Technical report, National Bureau of Economic Research.

Vigna, S. D. and U. Malmendier (2006). Paying not to go to the gym. *The American Economic Review 96*(3), 694–719.

Volandes, A. E., M. K. Paasche-Orlow, M. J. Barry, M. R. Gillick, K. L. Minaker, Y. Chang, E. F. Cook, E. D. Abbo, A. El-Jawahri, and S. L. Mitchell (2009). Video decision support tool for advance care planning in dementia: randomised controlled trial. *BMJ 338*, b2159.

Volpp, K. G., L. K. John, A. B. Troxel, L. Norton, J. Fassbender, and G. Loewenstein (2008). Financial incentive–based approaches for weight loss: A randomized trial. *JAMA 300*(22), 2631–2637.

Volpp, K. G., A. B. Troxel, M. V. Pauly, H. A. Glick, A. Puig, D. A. Asch, R. Galvin, J. Zhu, F. Wan, J. DeGuzman, et al. (2009). A randomized, controlled trial of financial incentives for smoking cessation. *New England Journal of Medicine 360*(7), 699–709.

Wisdom, J., J. S. Downs, and G. Loewenstein (2010). Promoting healthy choices: Information versus convenience. *American Economic Journal: Applied Economics*, 164–178.