Family Health Behaviors

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Abstract

This paper studies how health behaviors and investments are shaped through family spillovers. Leveraging administrative healthcare data, we identify the effects of health shocks to individuals on their family members' consumption of preventive care and health-related behaviors. Our identification strategy utilizes the timing of shocks to construct counterfactuals for 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. Notably, we show that these spillover effects are far-reaching and cascade to siblings, stepchildren, sons and daughters in-law, and even "close" coworkers. While some responses are consistent with learning new information about one's own health, evidence from cases where shocks are likely uninformative points to salience as a major operative explanation. Our results underscore the importance of one's family and social network for models of health behaviors and have potential implications 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 (McGinnis and Foege 1993; Mokdad et al. 2004; Cutler et al. 2009). 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. The importance of identifying what determines health-related behaviors, which are notorious for being hard to change, has led to an active literature on a range of potential factors, with some particular focus on financial incentives and health education.[1] Still, we lack a clear understanding of the channels through which health behaviors and habits evolve over the life cycle.

A long tradition of economic research has underscored the importance of family interactions in determining individual behavior, particularly in the context of consumption and labor supply choices (Becker 1991; Browning et al. 2014). Similarly, there could be a role for the family in forming health behaviors, via the flow of information, awareness, and the creation of habits and norms.[2] 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.[3] Moreover, it requires large data sets on health and consumption of healthcare with linkages across family members.

In this paper, we study how family spillovers, both within and across generations, shape health behaviors over the natural course of the life cycle. Specifically, we estimate the causal effects of health shocks to individuals on their family members' consumption of preventive care and health-related behaviors, focusing on spouses and adult children. Our estimation strategy relies on the timing of shocks, constructing counterfactuals for affected households using households that experience the same shock but a few years in the future. We leverage a long panel of administrative data, which covers the entire adult Danish population from the years 1980-2011 and includes medication prescriptions, contacts with medical-care professionals, inpatient and outpatient hospitalizations, and death records. An important advantage of our setting is the ability to link across different family and social connections, which enables us to investigate the scope of 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 economically significant and exhibit a high degree of persistence: a single family shock can explain 28% and 15% of the growth in consumption of preventive care over a nine-year period around the event by younger adult children and prime-age spouses, respectively. We further find that these spillovers in healthcare consumption are far-reaching across one's network with a meaningful implied

[1] Cutler (2004) and Cawley and Ruhm (2011) offer reviews for developed economies, and Kremer and Glennerster (2011) review evidence from randomized evaluations in developing countries.

[2] A series of papers documents correlations among peers and family members, mostly across spouses, in a variety of health-related behaviors (as reviewed in, e.g., Meyler et al. 2007 and Cawley and Ruhm 2011).

[3] Some interesting recent studies aim to address this challenge. Cutler and Glaeser (2010) exploit smoking bans at the workplace to study spousal smoking behavior; Fletcher and Marksteiner (2017) analyze spouses' behavior in clinical interventions to reduce individuals' smoking and alcohol consumption; and Cawley et al. (2017) study effects across siblings in weight and obesity by instrumenting for a sibling's obesity using genetic risk scores.

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. In contrast, 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.

Studying the multiple network layers and the heterogeneity in their responses allows us to also probe into mechanisms. Guided by the literature and the empirical patterns, we conceptualize two major classes of mechanisms by which network shocks can trigger changes in health behaviors. The first channel is potential learning and revelation of new information about one's *own* risk. Adult children may be induced to learn of their genetic risk, for example, whereas spouses may be exposed to information on joint risks attributed to similar lifestyles and habits. The second channel is the shock's salience itself, where one's attention may be drawn to the health domain even when shocks are unlikely to reveal new information.

With these channels in mind, we investigate responses by the different network circles and exploit the richness of the data to proxy 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. While different mechanisms seem to be at play, including learning information about one's own health, there is consistent evidence in support of salience as a key operative explanation. The findings suggest that agents' attention is specifically drawn to the local nature of the shock so they take actions particular to that risk's domain.

To study how health behaviors are shaped through family spillovers, our core analysis focuses on the effects of non-fatal heart attacks and strokes. Heart attacks and strokes are commonly studied as sudden and severe events whose particular timing is likely unpredictable, and are therefore particularly well-suited for our empirical strategy (see, e.g., Chandra and Staiger 2007; Doyle 2011). These cardiovascular shocks also naturally fit our research question as they are directly tied to disease-specific risk-reducing preventive care, i.e., the cholesterol-lowering medication statins, the consumption of which we study as our main outcome. Moreover, 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.[4]

We begin with intra-generational analysis of spouses, where spillovers cannot be attributed to biological channels. We show that prime-age individuals immediately increase their consumption of statins by 15% in response to their spouse's cardiovascular shock, and that this increased take-up persists for the duration of our analysis horizon. The effects on statin use are accompanied by a prompt increase of 30% in blood tests for cholesterol levels that determine cardiovascular risk. Furthermore, we show that spouses with higher predicted risk are much more likely to increase their statin consumption. However, we find no such risk gradient in spouses' information-seeking behavior through cholesterol testing. This implies that spouses across predictable risk types are similarly prompted to take actions

[4]Cardiovascular shocks are among the leading causes of morbidity and mortality in the developed world (WHO 2014). They account for 1 in every 3 deaths among adults in the U.S. (with similar rates across the developed world), and every year more than 1.5 million Americans experience either a heart attack or a stroke. Statins are among the most widely prescribed and best-selling medications, with the global market for statins estimated to be $20 billion annually in the last decade (Redberg and Katz 2016).

related to cardiovascular risk (through data gathering), but those with higher predicted risk end up having larger statin responses in practice, consistent with learning induced by the family shock. We additionally show that spouses in couples with more similar risk profiles, who are presumably more likely to learn from the shock about their own health risks, exhibit stronger statin consumption responses.

We next refer to the medical literature, which asserts that the relevant information for receiving cholesterol-reducing treatment is observable risk factors and cholesterol levels. Hence, spouses (and other non-blood relatives) who had been tested for cholesterol levels in the pre-shock period, have already gained access to the risk-related information set pertinent to statin consumption. Still, even such spouses exhibit similar-magnitude responses; so while the evidence points to a likely role of family shocks in inducing learning of own health risks, it simultaneously suggests the salience of the health condition as a likely channel. Alternative explanations, such as supply-side responses of family physicians who aggregate information across household members or greater valuation of spouse's health (who could become the main earner or caregiver), do not appear to be operative.

We then turn to study how parental shocks spill over to health behaviors in the next generation. For both younger and older adult children we find large and persistent statin consumption increases in response to parental cardiovascular shocks. We show that their responses are immediate and grow stronger over time, so that by the end of the analysis period of four years they amount to 36% and 16% for younger and older children, respectively. We find comparable patterns and response magnitudes by siblings, who are likely subject to similar impact mechanisms.

To take a step toward isolating potential mechanisms in the spillovers among the next generation, we study network circles for whom different channels may be at play. First, to abstract from the biological-risk channel, we analyze spillovers to stepchildren whose responses in statin consumption average to an 11% increase, which is half as large as that by biological children. To additionally abstract from the spillover channel of a family environment shared in childhood, we study the potential spillovers to sons and daughters in-law. We show that they exhibit an average increase in preventive-care consumption that amounts to about a quarter of that by their spouses, i.e., the biological children. Consistent with exposure intensity, we find that the 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. We show that "close" coworkers, as defined 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.

To increase our understanding of spillovers, we next analyze the effects of fatal family shocks, focusing on spouses. We first show that fatal shocks increase family members' general awareness regarding their own health. We find increased rates of hospitalizations for suspected conditions that are ruled out upon examination and of urgent-care doctor visits. Likewise, we show significant behavioral improvements in the form of decreases in key harmful habits, which we find even when the shock does not bear new information on the risk involved in these behaviors.[5] Specifically, we show that

[5]Existing related work studies quality of care and health behaviors following spousal bereavement, mainly to test whether they can account for corresponding declines in health (see a review in Stahl and Schulz 2014). These papers use designs different from ours and generally compare outcomes before and after bereavement or around the event, or compare widowed to non-widowed individuals. In contrast to our analysis, their comparisons mostly find no changes or some declines in quality of care or in healthy

family members immediately engage in consumption of medications prescribed to assist in cessation of smoking or excessive drinking. We also find reductions in consumption of addictive harmful medication by studying prescription opioids, which account for the greatest share of deaths linked to prescription drug abuse.

Spousal death can have significant direct health effects.[6] To take a step toward isolating behavioral responses in this context, we study heterogeneity among treated spouses only, with the idea that they all face the main effects of the shock. We exploit variation in exact causes of spousal death to study consumption of different condition-specific preventive care, both within and across conditions, in the contexts of cardiovascular disease and cancer. We show that individuals whose spouse died of cardiovascular disease persistently consume statins at higher rates compared to those whose spouse died of other causes. In the same vein, individuals whose spouse died of cancer as compared to other causes, significantly increase their expenditure on diagnostic radiologists, who specialize in screening for major types of cancer. Importantly, we find similar responses in the case of husbands whose wives died of female cancers, where the spouse's cancer type is not likely informative of own cancer risk (which we verify in the data), consistent with a salience channel.

These findings highlight that family members' behavioral responses target the domain of the particular experienced risk. To stress this point, we further show that the behavioral changes are local to the vicinity of the experienced risk's domain, by studying cross-condition responses. We find no differential expenditures on diagnostic radiology by individuals whose spouse died of cardiovascular disease. Moreover, 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 crowd out. Namely, it is not only that individuals increase their take-up of preventive care specific to the family shock, 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.

In addition to the literature already mentioned, our findings also contribute to the theoretical work on health-related habits and choices (e.g., Grossman 1972; Becker and Murphy 1988; Orphanides and Zervos 1995; Laibson 2001; Bernheim and Rangel 2004). The results point to a role of one's network in health-related habit formation and underscore the importance of incorporating inter-personal interactions across different family generations and peers in analyzing health behaviors, investments, and the consumption of healthcare. The response patterns also highlight learning and, specifically, salience and attention as key modeling components (as in, e.g., DellaVigna 2009, Bordalo et al. 2012, 2013, and Gabaix 2017),[7] and point to event-driven responses, in line with frameworks such as Bernheim

behaviors, where an exception is Jin and Christakis (2009). Relatedly, Khwaja et al. (2006) study associations between smoking and spousal health in the Health and Retirement Study.

[6] Previous literature (see Stroebe et al. 2007 for a review) and our own investigation based on hospitalization data find meaningful effects on spouses' physical health. This is in contrast to non-fatal cardiovascular shocks, where we find no evidence for such effects on family members. We provide this analysis later in the paper.

[7] 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. Our work is also related to the growing evidence of how limited attention and salience affect economic behavior in a variety of settings; see DellaVigna (2009) and Gabaix (2017) for reviews.

and Rangel's (2004) cue-triggered decision making model. More generally, the results can additionally advance our understanding of the nature and scope of broader network effects in consumption, here through the lens of healthcare utilization.[8]

Lastly, the findings could also have implications for policies that aim to promote population health. They emphasize that health behaviors are not immutable and suggest the leveraging of family events as a window of opportunity for intervention, involving the required intrinsic motives for persistent behavioral changes. One could exploit these events to provide family members with individual-specific information on risks; or to introduce policies that leverage salience of health to actively offer preventive care. However, the evidence also suggests more broadly that salience should be used with caution as a policy tool. Agents' attention can be drawn to specific risks even in the absence of relevant information, which could end in excessive healthcare consumption; and, at the same time, their attention may be diverted away from conditions of which they might be at higher risk.

The remainder of the paper is organized as follows. In Section 2 we describe our empirical strategy to estimate the effects of adverse health shocks on family and network members' health behaviors. Section 3 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 4. Section 5 discusses the implications of our findings and concludes.

2 Research Design

The goal of our analysis is to identify the dynamic causal effects of severe health shocks on family members' consumption of preventive care and health-related behaviors. 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 to analyze heterogeneity in treatment effects to shed light on the nature of spillovers and underlying mechanisms.

2.1 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 a long panel of administrative data on the universe of Danish households allows us to utilize a quasi-experimental research design to mimic 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

[8] 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.

was realized. Specifically, we construct counterfactuals for 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 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 ten years apart ($\triangle = 10$) will allow us to estimate the effect of the shock for up to nine 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 (actual or assigned) 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 $\triangle$.[9]

The remainder of this subsection formalizes the research design and describes our estimator and econometric models.

Formal Description of the Design and Estimator. Similar to common practice (for example, in the use of matching estimators; see 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

[9] The empirical strategy allows for behavioral adjustments in expectation of a shock among treated households, as well as for baseline time trends in outcomes that could be attributed to assortative matching across spouses or to genetic correlations across parents and children. By construction, the strategy aims to provide a control group with non-differential expectations and time trajectories in the pre-shock years, as manifested by the parallel pre-trends across the two experimental groups.

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 the control group at time r, purged of the difference in their outcomes at the baseline period p. 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 main 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] Validation of the parallel trends assumption requires that

[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 identical to those from (1).

$\delta_r = 0$ for all $r < 0$. Unless otherwise indicated, we include in $X_{i,t}$ age fixed effects, calendar year fixed effects, gender, and education, and we 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 effect of shocks on family members' outcomes.

2.2 Response Heterogeneity

There are two related strategies that we employ when analyzing the nature 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 in the analysis of 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). Lastly, the corresponding equation that estimates how responses vary on average across treated households with different levels of z_i takes the form:

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

[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 $post_{i,t}$.

where δ is the parameter of interest.

3 Data and Institutional Background

In this section we describe our data sources and analysis sample, and provide relevant institutional background. The Danish setting that we study is a well-suited environment for identifying family spillovers in health behaviors in the context of developed economies. First, it provides us with the required long panel of detailed administrative healthcare records for linked family members (and other network circles). Moreover, 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.

3.1 Data Sources

Studying family members' health-related behavioral changes in response to severe health events requires information on healthcare utilization outcomes and 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 layers of their family and social network.

For utilization outcomes that measure health behaviors, we use 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 4.

For identifying family health shocks and their timing, we use the latter hospitalization dataset (the *National Patient Registry*) as well as 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.

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.

3.2 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 (that corresponds to $\triangle = 5$). 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 in 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.[13]

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.[14] 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 where the number of employees was equal to or lower than the sample's 25th

[13] Our choice of the degree to which power should increase, as determined by the number of required years of survival, was governed by balancing the decrease in the analysis horizon with 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 biological children.

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

percentile (of approximately 20). We then study the effects on coworkers who have been employed in a similar occupation class,[15] and who are close to these individuals in terms of age (with an age gap of 5 years or less).[16] We exclude from this sample any coworker who is also a family member.

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.

3.3 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 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.[17]

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 indepen-

[15] 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).

[16] Estimations that perturb the thresholds of workplace size and age gap, which we chose to balance sample size and "closeness" of peers, are provided in Appendix Table 5 for robustness.

[17] 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.

dent units with their 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 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. 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).

4 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 core part of our analysis, we study the effects of non-fatal heart attacks and strokes on family members' consumption of preventive care that is specific to these cardiovascular conditions. 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 to coworkers. In the second part of the analysis, we study the effects of general fatal shocks on family members' behaviors. In this part, we begin by analyzing overall changes in consumption of healthcare that is indicative of health behaviors and awareness, and then proceed to studying utilization of condition-specific preventive care by exploiting variation in causes of death. We focus on reporting the estimated effects in relative terms compared to the counterfactual, in order to account for the baseline prevalence of the healthcare utilization outcomes we analyze within each sub-population.

Whereas our goal is to identify the main effects of family shocks, we additionally investigate their nature and potential sources. To reiterate, we conceptualize two main classes of spillover channels: learning about one's own health risks and the salience of shocks. Our focus on these channels is guided by the literature and the empirical patterns; but, of course, alternative mechanisms could be at play, some of which we show can be likely ruled out.

4.1 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.[18] We therefore proceed with analyzing how individuals' cardiovascular shocks affect the consumption of statins by members of their network.

4.1.1 Effects on Spouses

We begin with an intra-generational analysis of spouses, to identify family spillovers that cannot be attributed 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. 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 (see Appendix Figure 1), which include routine checkups that are more common as individuals age. As the main risk factors for 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 more informed of their personal risk. Still, even for spouses between the ages of 55 and 85 we find

[18] We further verify that family members' statin consumption responses are prevention related, and hence take the form of health investments, as they are not driven by managing own conditions that might have been induced by the family shock. There is no increase in the incidence of cardiovascular disease (and of hospital contacts of any type) among spouses or children following the non-fatal cardiovascular shocks that we analyze. See Appendix Table 7 for details.

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 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 take up statins, as manifested by the gradual growth in the estimated statin consumption effect. 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 the 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 lagged indicators for 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). 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 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 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 an additional 1.08 pp. Nonetheless, we find no such risk gradient in the spouses' information-seeking behavior through cholesterol testing. This suggests that the shock uniformly drives spouses across predictable risk types to take actions related to cardiovascular risk (in the form of

gathering data), whereas those with higher predicted risk end up having larger statin responses in practice, consistent with learning of own health risks induced by the family shock (see columns 1 and 2 of Appendix Table 2).[19] 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 more 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 data on cholesterol testing to probe into mechanisms. A large body of medical research and the corresponding clinical guidelines indicate that the information on risk pertinent to receiving cholesterol-reducing treatment is a combination of one's LDL ("bad") cholesterol levels and the predicted cardiovascular risk we discussed in previous paragraphs, 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 information regarding their own cardiovascular risk by which statin eligibility is determined. 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 such spouses respond to the shock in their statin consumption in any way, and we find that they do (see column 2 of Table 2). Consistent with a salience channel, spouses increase their consumption of preventive care in response to the family shock even when the relevant information set had been available to them prior to the event.[20]

The analysis so far suggests both a likely role of family shocks in inducing learning about own health and that salience and attention are likely operative channels. We additionally show that several leading alternative explanations do not appear to be operative in the responses we estimate. Specifically, the evidence does not support the hypothesis that the spousal health investments are driven by greater valuation of the spouse's health due to realized household income risk, child rearing considerations, or caregiving needs. First, due to generous social insurance, households are well insured and experience very small income losses to begin with (taking into account all income sources and transfers). Moreover, while households in which the sick person was the primary earner experience larger income losses, spousal health investments do not vary by this dimension. Second, spouses in households with younger children do not exhibit larger investments in health. And, third, while individuals who experience more severe shocks, as measured by hospitalization days, are more likely to drop out of the labor force and potentially require more caregiving, spouses' investments do not vary by this dimension either (see Appendix Table 3). In addition, the evidence does not support the hypothesis that the spillover can be explained by supply-side responses of family physicians who aggregate information across the different members of the household. Analyzing households in which husbands and wives do not share the same doctor (defined in several ways), we find similar-magnitude responses (see Appendix Table 4).[21]

[19] 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.

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

[21] In this table, we also reach similar conclusions from an analogous exercise for adult children, whom we study in the next subsection.

4.1.2 Inter-generational Effects

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 36%. Analogously, panel B of Figure 2 plots the spillovers to 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 statin consumption that amounts to 16% by the fourth year after the shock (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 about 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 learning channel, we find a strong negative partial correlation between children's statin consumption responses and their parent's age at the time the shock occurs.

The causal spillovers to health investments by biological children involve several channels. These channels could include revelation of information about biological risk as we just discussed, learning information on own risk that could pertain to shared environmental risk growing up, and salience and increased awareness. Subject to a similar set of channels, we also find comparable 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).

With the aim to shed light on the nature of the estimated spillovers to the next generation, we focus in the rest of this subsection on family connections that can enable us to isolate response channels. 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 to non-biological

children and amounts to an 11% 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 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 responses that are half as large as those by biological children. 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 precision. Column 1 of Table 4 estimates 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 responses come into effect in the year just after the shock (when all individuals are already exposed to it for a full period) and grow 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. After abstracting from the biological-risk channel, we seek to additionally abstract from the spillover channel by which children may respond to learning information on joint risks attributed to sharing the same environment with their parents when growing up. This includes several dimensions such as the immediate physical surroundings, 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 using equation (3). It shows that, following the cardiovascular shock to their spouse's parent, children in-law exhibit an average increase that 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.

To further investigate 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 in the baseline period $t = -1$.[22] We divide the sample into children in-law 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.[23] 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

[22] Specifically, our analysis is based on post-2007 definitions, which divide Denmark into 98 municipalities. Distance is measured 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).

[23] 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.

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 precisely estimated using the dynamic regression of specification (2) (see column 2 of Table 4). They exhibit immediate responses following the shock with a 15% increase by the end of the analysis' period (similar to spouses).[24]

4.1.3 Effects on Coworkers

In the final empirical exercise of this section, we study how far the spillovers of health shocks to 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 3.2).[25] Column 1 of Table 5 displays the average treatment effect of health shocks on close coworkers' consumption of preventive care. The results indicate an 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 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 which are purely social, and is on the same order of magnitude as the estimated spillovers to prime-age spouses.

Our setting also provides us with the opportunity to illustrate the limit of spillovers. While 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 (column 2 of Table 5). 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 gaps or on those with similar ages within the same physical establishment but with different occupation classes, who may therefore represent peers with weaker social ties (see columns 3-4 of Table 5).[26] Put together, the patterns of spillover impacts on healthcare consumption among close coworkers, and the lack thereof among distant coworkers, are consistent with the strength of the social tie (and the likely corresponding degree of shock exposure and prominence) as operative channels.

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

[25] Namely, we look at coworkers within the same occupation class and age range in smaller workplaces. Perturbations to cutoffs used in these definitions are reported in Appendix Table 5 as we mentioned before.

[26] Coworkers 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. Likewise, the finding of no effect on coworkers in similar location and occupations but with a larger age difference suggests that job-related risk (occupational risk, stress, etc) is not a likely channel as well.

4.1.4 Economic Magnitudes and Benchmarks

Benchmarking our estimated spillovers against baseline counterfactual levels 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 of preventive care within the full analysis window (from period -4 onward), which takes into account time, life-cycle effects, and the impact of the spillover, and we then assess the share of the spillover effect out of the 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 nine-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 nine-year growth in statin consumption for younger and older adults, 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. 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. 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 health behaviors more generally.

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 consume 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 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.[27] 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 included in their study, 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-

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

run (year-4) effect for spouses, adult children, and siblings, who are in this age range at the end of the analysis period.[28] For these sub-groups (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.

4.2 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 shocks. The advantages of this analysis in advancing our understanding of family spillovers are the opportunity to study a variety of behavioral margins (supplemental to those related to cardiovascular risk), as well as the ability to explore additional mechanism-related tests. We focus on spouses to identify spillovers that cannot be attributed to biological channels. Additionally, spouses in our sample are older and hence have higher baseline levels of healthcare utilization compared to 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.[29]

4.2.1 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 may 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 specification (2) along with their 95-percent confidence interval. 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.[30]

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

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

[29] See Appendix Table 6. 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.

[30] 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 2.1, 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.

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 that are initiated outside of regular working hours with local doctors or nurses who are on call.[31] 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.

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 (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.

We first explore the consumption of medication that treats nicotine or alcohol dependence. This class of medication is prescribed to individuals engaged in *chronic* smoking or excessive drinking for lengthy periods, who wish to cease their unhealthy behavior or to switch to a less damaging substitute (Siu 2015; Swift and Aston 2015).[32] As such, evidence of prompt increases in their consumption would suggest they are likely 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 estimates of equation (2), where the outcome variable is an indicator for the purchase of a prescription drug within this class. The estimates show an immediate increase in individuals' consumption of these medications following spousal death, which amounts to 41% compared to the counterfactual.[33]

To shed light on potential response channels, we further 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 fatal spousal shocks 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). 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 (see column 1 of Appendix Table 6).

Next, we move on to study prescription medication abuse, where concerns pertain to the dangers of developing dependence or addiction. We analyze 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).[34] In this case, health-promoting

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

[32] 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 part of cessation regimens.

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

[34] These medications have been a recent focus of the medical literature and public debate due to the common practice of abuse

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. 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.[35] Panel D of Figure 4 reveals a pattern that is consistent with improved behaviors. From a baseline counterfactual level of 9.74 prescription days (in period 0), spousal death induces a decline in the amount of prescription opioids consumed, which 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. 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.

4.2.2 Condition-Specific Preventive Care

We proceed with the final portion of our analysis, in which we study the consumption of condition-specific preventive care by exploiting variation in causes of death among treated households. Recall that this first allows us to take a step toward isolating behavioral responses in the context of spousal death, where the shock can lead to meaningful declines in health (see Appendix Table 7). The underlying idea is that all spouses among treated households are exposed to the main effects of the shock, but their experience may differ by the cause of their spouse's death.[36] Moreover, this analysis enables us to investigate the directionality of spillover responses and the degree to which they may be local to the particular nature of the experienced shock.

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 z_i 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 the 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. In support of the empirical design, there are no differential pre-trends across the two groups of households, so that

and the high risk of addiction or dependence associated with them.

[35] 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.

[36] Among different alternative explanations, this analysis reduces the plausibility that potential complementarities between treatment and preventive care explain the findings of the effects of fatal shocks on spousal health behaviors.

$\delta_r = 0$ for $r < 0$. Observing the post-shock coefficients, a clear pattern emerges: following fatal shocks, individuals whose spouse died of cardiovascular disease promptly and persistently consume statins at a higher rate compared to those whose spouse died of any other cause.[37]

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 the cardiovascular deaths application, we compare individuals whose spouse died of cancer to those whose spouse died of any other cause. However, unlike the case of statins whose consumption is for continuous health risk management, the current utilization outcome of visits to radiologists is for diagnosis purposes only, so that behavioral responses should translate to transitory effects. We estimate the corresponding regression in column 3 of Table 6, which shows there are no differential pre-trends across the two groups of spouses in support of the design. 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 line with effects of family shocks on condition-specific *diagnostic* preventive care.[38] The pattern is also in accordance with the analysis of outpatient hospital contacts for the reason of having a family member with a history of cancer, which exhibit increased incidence just after spousal death (column 4 of Table 6).

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 particular cancer types. We find 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 (column 1 of Table 7). In contrast, there are no such differential responses when we compare spouses of individuals who died of non-lung cancer to spouses of individuals who died of any non-cancer cause (column 2 of Table 7).

The second class of cancer types that we study is female cancers, which in our sample include ovarian, cervical, and breast cancer.[39] 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-cancer cause. The advantage of this context is the limited potential for information revelation regarding husbands' own risk of cancer, as we verify in the data.[40] Still, we find responses that are very similar

[37] For this application there is also sufficient power to characterize the dynamics for children, for whom we find a similar pattern (see column 2 of Table 6).

[38] Guided by this dynamic specification, our following estimations that study expenditure on diagnostic radiology using equation (6) assign the value 1 to $post_{i,t}$ in periods 1 and 2.

[39] 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).

[40] 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

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 on spouses' own risk and consistent with responses to the health condition's salience, 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).[41]

Cross-Condition Preventive Care. Whereas the within-condition analysis suggests that spillover responses target the experienced risk, cross-condition analysis can further indicate how local they are to the vicinity of the shock. This analysis can also provide placebo tests for the hypothesis of salience of specific risks as an operative explanation. It addresses alternative hypotheses such as the conjecture that individuals whose spouse dies of a cardiovascular shock or cancer may be generally more responsive than others in any preventive care margin, not only in margins related to the experienced condition.

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'.[42] Column 5 of Table 7 finds that expenditures on diagnostic radiology by individuals whose spouse died of cardiovascular disease are not different from those by individuals whose spouse died of other causes; suggesting that their increased responses in preventive care (in this case, statins) were local to the particular experienced shock. Similarly, further strengthening this result, we find no increases in expenditure on diagnostic radiology in the context of Section 4.1.1, namely, in response to a spouse's non-fatal cardiovascular shock. Moreover, 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. This points to the possibility of crowd out: it is not only the case that individuals increase their take-up of preventive care specific to the family shock, even when shocks are likely uninformative of their own risks, but they may also reduce the take-up of preventive care that pertains to competing, non-salient 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.

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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). Ex-ante, before investigating the data for verification, one could not have precluded some degree of learning about own cancer risk, since risk factors that lead to a wife's female cancer could potentially lead to a husband's cancer.

[41] 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 that resulted in standard errors three times as large as those in the analysis of female cancers, which produced wide and uninformative confidence intervals.

[42] 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).

5 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 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 coworkers. This reveals network spillovers in consumption that are significant in magnitude, breadth, and persistence, within 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 generally 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 major operative channels, within a variety of cases where shocks are likely uninformative of one's own risk. While this salience channel 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 specific to the realm of that risk.

Consequently, our findings also have implications for models of health-related behaviors. The results highlight the importance of inter-personal interactions, suggesting we should 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 results also inform models with respect to the ingredients they should suitably include. They point to event-driven decision processes and suggest that within-network learning and, in particular, salience and attention should be key modeling components.

Finally, our findings could have implications for policies that aim to improve population health. 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 family-centered approach to healthcare delivery, in which medical professionals actively involve family members in the treatment process. As family health events induce responses with a high degree of adherence, they seem to involve the intrinsic motives necessary for persistent behavioral changes. Building on the likely channels, one could devise policies that provide individual-specific information on 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 treatments.[43] However, our findings also point more generally to potential pitfalls in using salience as a policy tool

[43] 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), which recommend family screenings of high-risk individuals to identify additional family members who would benefit from assessment and treatment.

(e.g., through information provision or through surveying as in Zwane et al. 2011). We have found that 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. What is more, 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 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.

References

Becker, G. S. (1991). *A Treatise on the Family*. Harvard University Press.

Becker, G. S. and K. M. Murphy (1988). A theory of rational addiction. *Journal of Political Economy 96*(4), 675–700.

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

Bordalo, P., N. Gennaioli, and A. Shleifer (2012). Salience theory of choice under risk. *The Quarterly Journal of Economics 127*(3), 1243–1285.

Bordalo, P., N. Gennaioli, and A. Shleifer (2013). Salience and consumer choice. *Journal of Political Economy 121*(5), 803–843.

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

Cawley, J., E. Han, J. J. Kim, and E. C. Norton (2017). Testing for peer effects using genetic data. Working Paper 23719, 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.

Cutler, D. M. (2004). Behavioral health interventions: What works and why. *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*, 643–674.

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., E. L. Glaeser, and A. B. Rosen (2009). Is the us population behaving healthier? In *Social Security Policy in a Changing Environment*, pp. 423–442. 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. *European Heart Journal 24*(17), 1601–1610.

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

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.

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

Gabaix, X. (2017). Behavioral inattention. Working Paper 24096, National Bureau of Economic Research.

Grossman, M. (1972). On the concept of health capital and the demand for health. *Journal of Political Economy 80*(2), 223–255.

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.

Kremer, M. and R. Glennerster (2011). Improving health in developing countries. *Handbook of Health Economics 2*, 201–315.

Laibson, D. (2001). A cue-theory of consumption. *The Quarterly Journal of Economics 116*(1), 81–119.

McGinnis, J. and W. Foege (1993). Actual causes of death in the united states. *JAMA 270*(18), 2207–2212.

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.

Mokdad, A. H., J. S. Marks, D. F. Stroup, and J. L. Gerberding (2004). Actual causes of death in the united states, 2000. *JAMA 291*(10), 1238–1245.

National Institutes of Health (2016). Understanding autoimmune diseases.

Orphanides, A. and D. Zervos (1995). Rational addiction with learning and regret. *Journal of Political Economy 103*(4), 739–758.

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), 1979–1981.

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.

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.

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 32*(1), 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.

Stroebe, M., H. Schut, and W. Stroebe (2007). Health outcomes of bereavement. *The Lancet 370*(9603), 1960–1973.

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

Volkow, N. D. (2014). Prescription opioid and heroin abuse. *National Institute on Drug Abuse*.

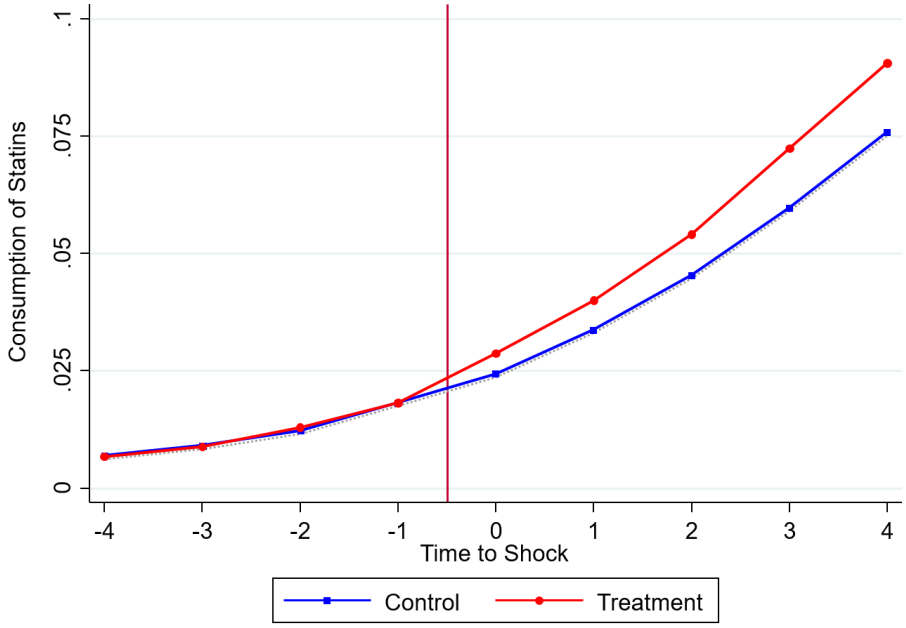
World Health Organization (2014). The top 10 causes of death.

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.

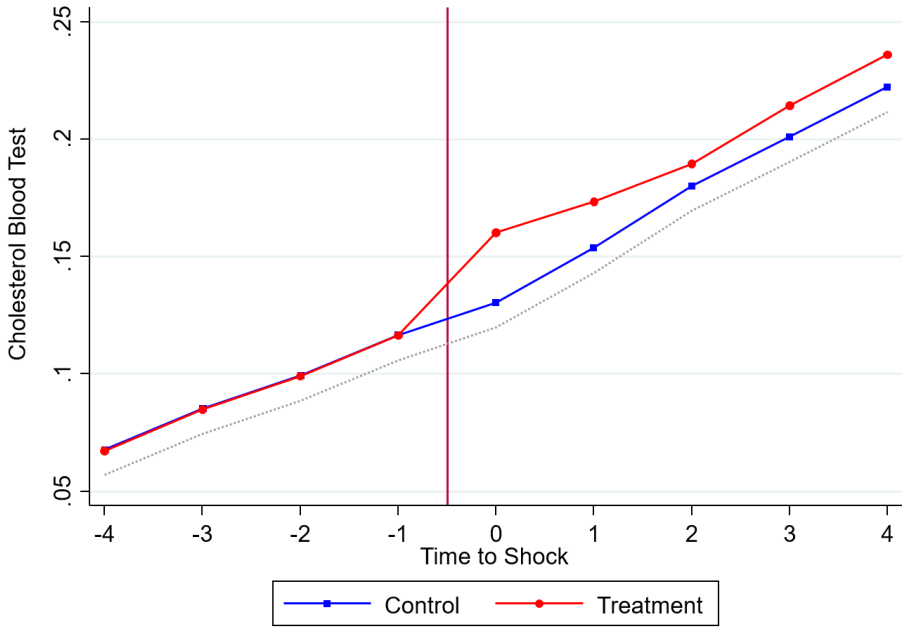
Zwane, A. P., J. Zinman, E. Van Dusen, W. Pariente, C. Null, E. Miguel, M. Kremer, D. S. Karlan, R. Hornbeck, X. Giné, et al. (2011). Being surveyed can change later behavior and related parameter estimates. *Proceedings of the National Academy of Sciences 108*(5), 1821–1826.

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

A. Statin Consumption by Prime-Age Spouses



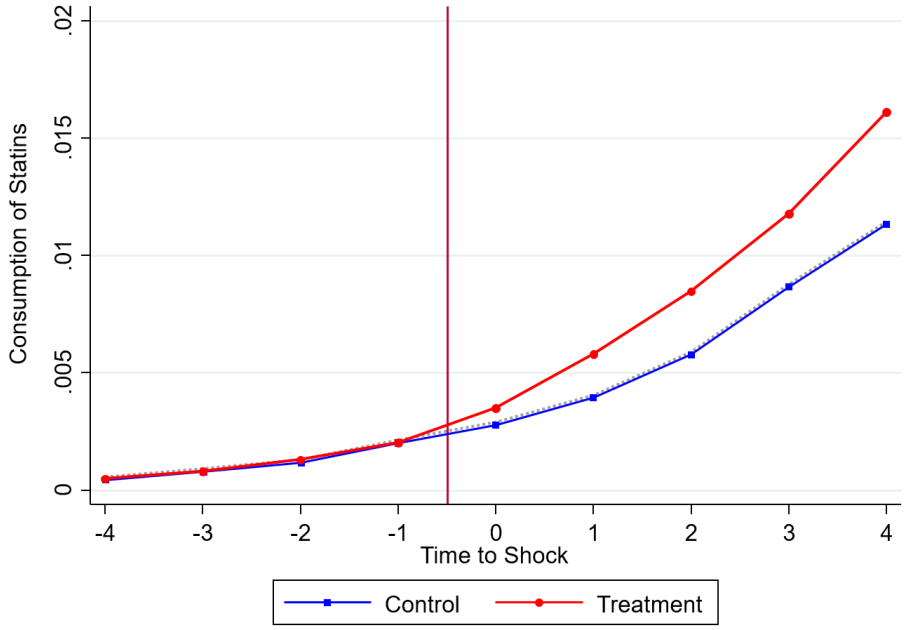
B. Cholesterol Blood Tests



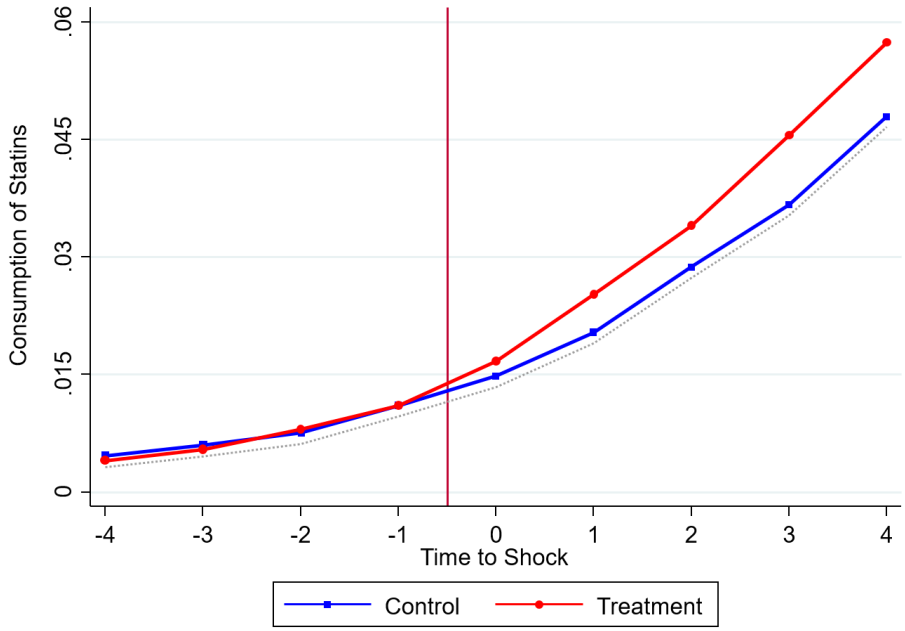
Notes: These figures plot changes in consumption of preventive care by spouses in response to family cardiovascular shocks. The x-axis denotes time with respect to the shock, normalized to period 0. For the treatment group, period 0 is when the actual shock occurs; for the control group, period 0 is 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 Adult Children (Ages 25-40)



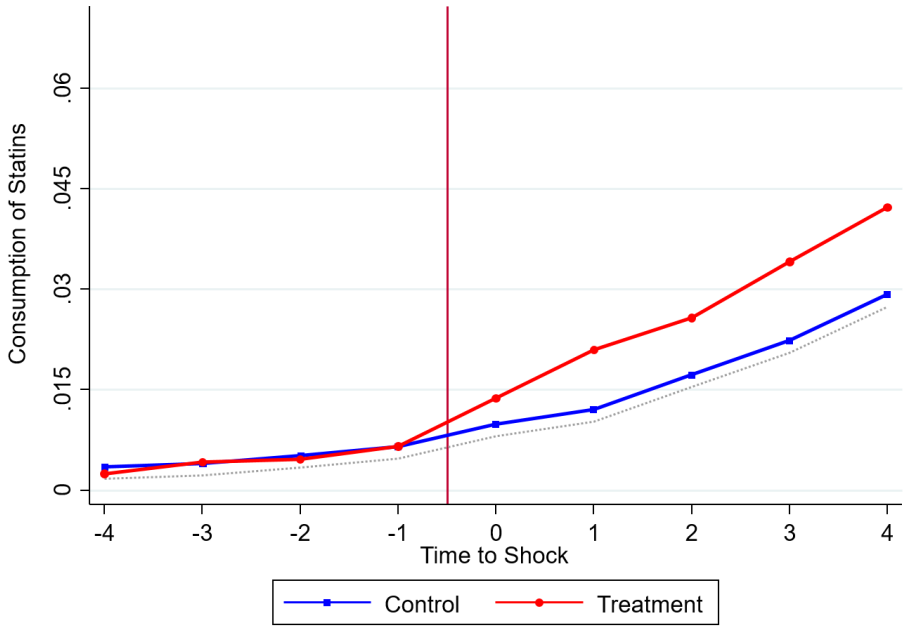
B. Statin Consumption by Older Children (Ages 40-65)



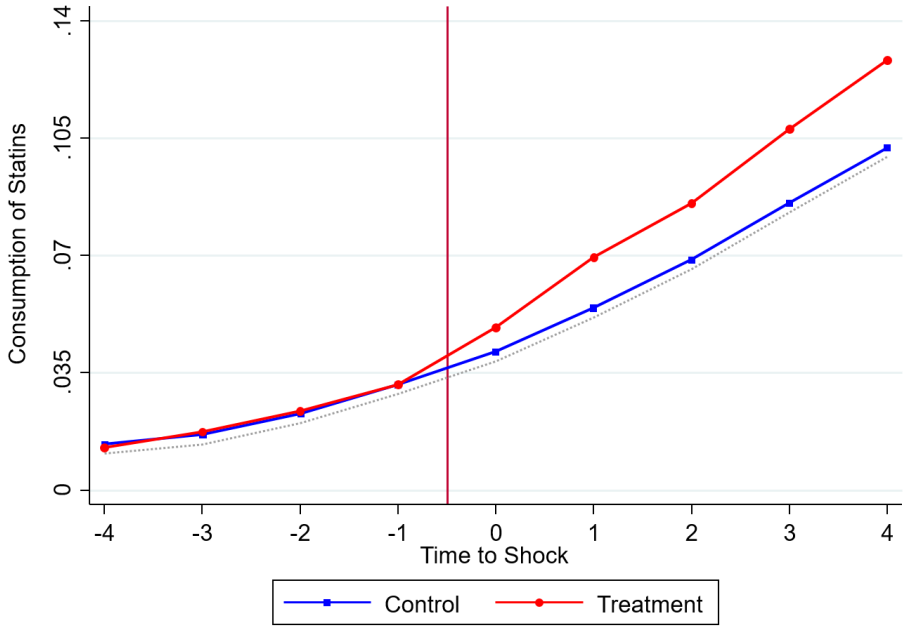
Notes: These figures plot changes in consumption of preventive care by adult children in response to family cardiovascular shocks. The figures are constructed as described in the notes of Figure 1.

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

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



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

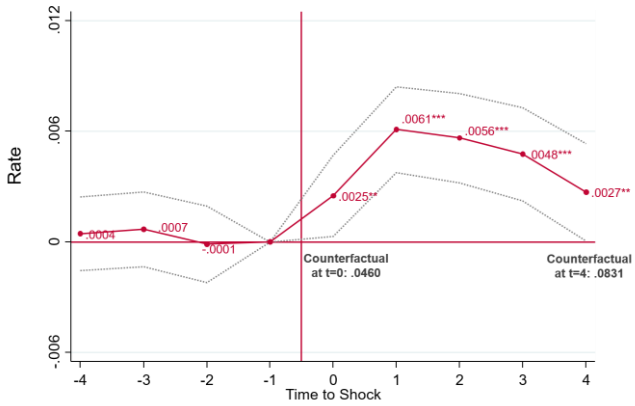


Notes: These figures plot changes in consumption of preventive care by adult siblings in response to family cardiovascular shocks. The figures are constructed as described in the notes of Figure 1.

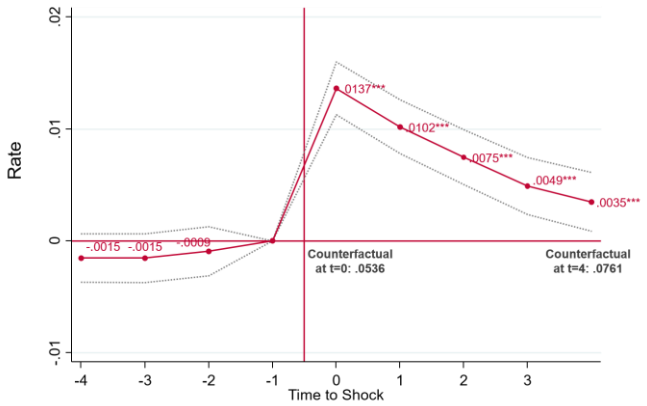
Figure 4: Effects of Fatal Shocks on Spousal Health Behaviors

Increased Awareness of Health

A. Hospital Medical Observation for Conditions that Are Ruled Out

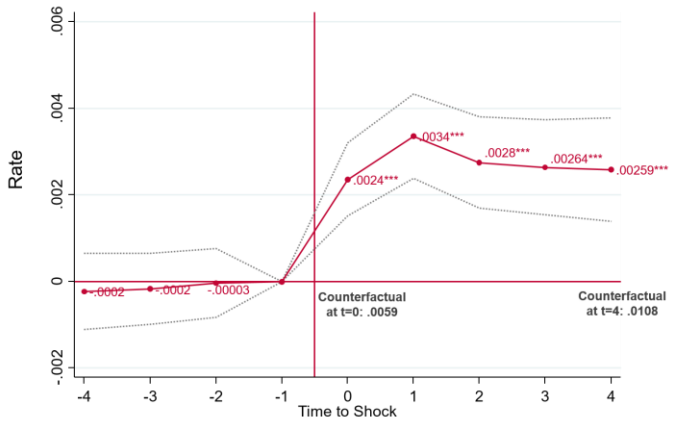


B. Non-Hospital Urgent Care Contacts

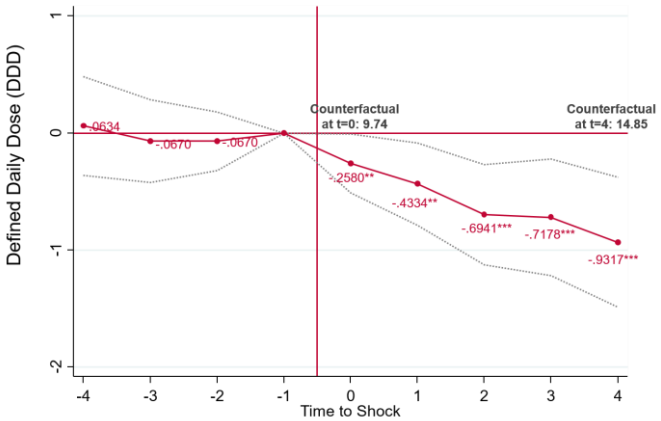


Declines in Harmful Behaviors and Medication

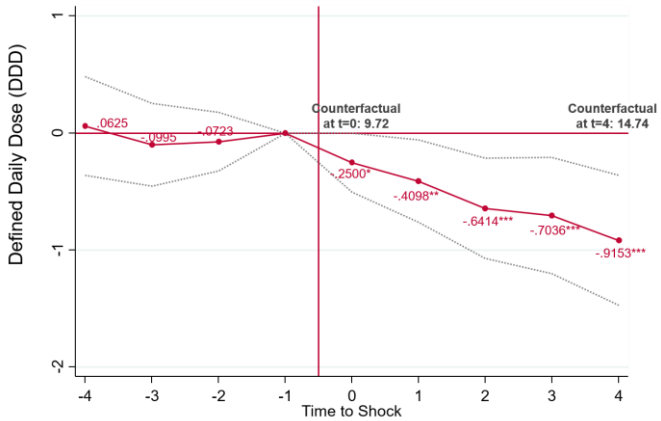
C. Medication to Treat Chronic Dependence (Smoking/Alcohol)



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). The figures plot the estimates for δ_r along with their 95-percent confidence interval. 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' Statin Consumption		Spouses' Cholesterol Testing	Adult Children's Statin Consumption		Adult Siblings' Statin Consumption	
	Prime Age (Ages 25-55)	Older (Ages 55-85)		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. 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**	.0178**	Treat x Post x	-.00023***
	(.0020)	(.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 Statin Consumption by More 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 of Distance
	(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)
Counterfactual	.0226	.0219	.0297	.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. The comparability of biological children included in column 2 to stepchildren included in column 1 is in terms of ages and data construction considerations, so that we include biological children of all ages (25-65) whose parent survived for at least three years. 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 we report the average effect for each subsample 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 to 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 Statin Consumption by More 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=3	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. 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	.0139***	-.0002	.0041	.0054
	(.0050)	(.0029)	(.0030)	(.0047)
Counterfactual	.1055	.1034	.0742	.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's Statin Use when Cause of Death is Cardiovascular	Spouses' Diagnostic Radiology when Cause of Death is Cancer	Spouses' Hospital Contact with Family Cancer 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

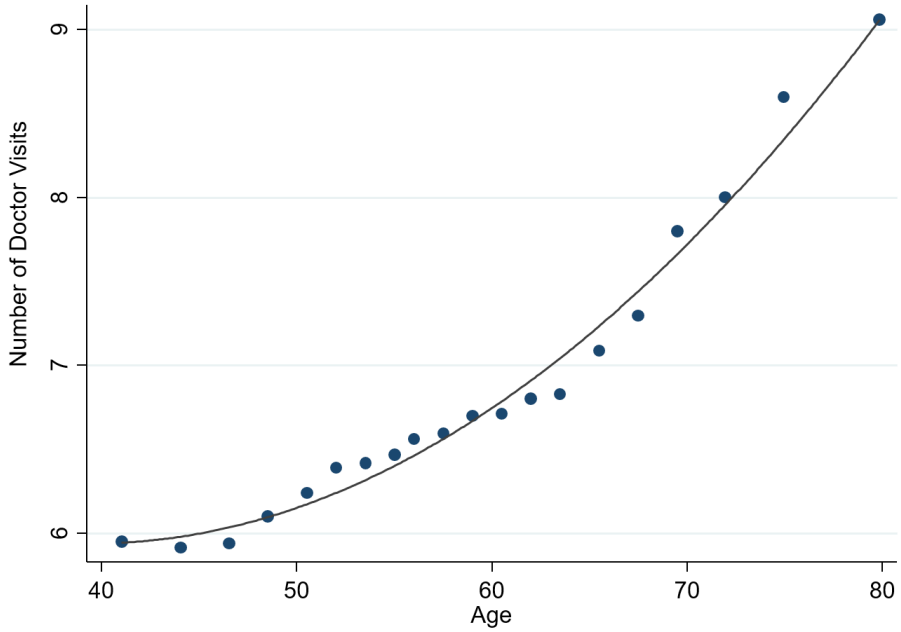
Notes: This table reports the dynamic differences-in-differences estimates for the evolution of household responses around fatal shocks using specification (5). The regressions are estimated on treated households only, where we divide the sample by cause of death. The table displays estimates for the 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 a history of cancer (code Z80 in ICD-10 classification). 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.

Table 7: Spousal Health Behaviors following Fatal Shocks

	Smoking Cessation by Lung Cancer	Smoking Cessation by Non-Lung Cancer	Husbands' Diagnostic Radiology by Female Cancer	Husbands' Cancer-Related Hospital Contact by Female Cancer	Cross-Condition Responses	
					Diagnostic Radiology by Cardiovascular	Statin Consumption 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		

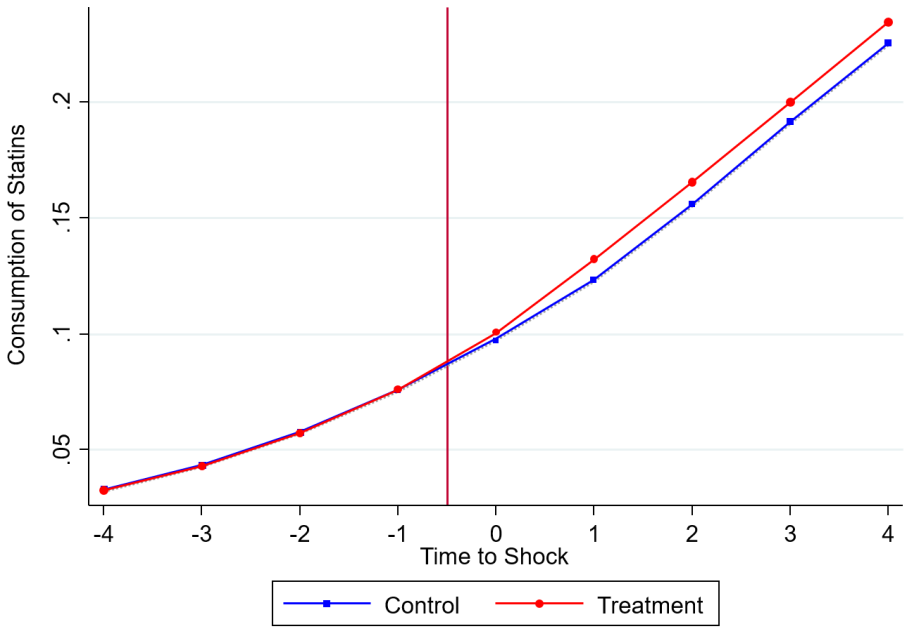
Notes: This table reports mean differences-in-differences estimates for spousal responses to fatal shocks using different specifications of equation (6). Column 1 studies the consumption of medication for smoking cessation by individuals whose spouse died of lung cancer compared to those whose spouse 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 bins in the range of 40 to 80; the solid line represents the best quadratic fit (based on 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 (of 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 Health Shocks						
Spouses						
Prime Age (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
Stepchildren	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 Health 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 whose family member (or peer) experienced a shock in some calendar year, to whom we match as a control group individuals from the same cohorts whose family member (or peer) 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 in 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. We proxy for peers using coworkers based on matched employer-employee register data, where we define workplaces using physical establishment units. From our sample of individuals who experience a health shock we identify those who have worked during the pre-shock periods from -4 to -1, and we include in the coworkers sample all employees from the corresponding workplaces. We exclude any coworker who is also a family member. 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

	Cholesterol Testing	Statin Consumption for Subsample from Column 1	Statin Consumption	Statin Consumption	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. In this regression, the post-shock years also include period 0, in which the dynamic analysis found a large effect, and the sample comprises residents of Greater Copenhagen for whom data on blood tests are available. Column 2 provides a similar analysis for this subsample 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 period -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: Testing Alternative Mechanisms for Spousal Responses

	Presence of Children		Severity of Shock		Income Loss			
	Statin Consumption	Statin Consumption	Statin Consumption	Sick Spouse's Labor Supply	Mean Changes		By Sick Spouse's Share of Income	
					Statin Consumption	Household Income	Statin Consumption	Household Income
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treat x Post	.0115***		.0180***	-.0331***	.0174***	-9,273***	.0064**	-8,255***
	(.0026)		(.0016)	(.0024)	(.0015)	(604)	(.0027)	(1,641)
Treat x Post x	-.0087***	-.0045						
Child Below 18	(.0032)	(.0036)						
Treat x Post x		.0006***						
Own Age		(.0002)						
Treat x Post x			-.00003	-.0011***				
Hospital Days			(.00004)	(.00009)				
Treat x Post x							-.0002	-4,405**
Sick Primary Earner							(.0036)	(2,242)
Counterfactual						443,765		
Percent Change						-2.09%		
Number of Obs.	392,640	392,640	955,722	955,722	955,174	955,174	332,891	332,891
Number of Clusters	49,080	49,080	119,506	119,506	119,502	119,502	41,659	41,659

Notes: This table studies the heterogeneity in spousal responses to cardiovascular shocks along different dimensions. Columns 1-2 estimate equation (4) and analyze whether spouses' responses in statin consumption vary by the presence of younger children. Column 1 interacts the treatment effect with an indicator for the presence of children younger than 18, and column 2 also adds an interaction with the spouse's own age. Similar results are found for other age thresholds (12 and 6). In these regressions we include prime-age spouses (ages 25-55) who are more likely to have younger children. Columns 3-4 estimate equation (4) to study whether spousal responses vary by the severity of the family shock, as defined by the number of hospitalization days. Similar results are found if severity is defined by hospitalization days being above or below the median, and if we further interact the treatment effect with the sick spouse's age at the time of the shock. Column 3 and 4 jointly show that while those who experience more severe shocks are more likely to drop out of the labor force (and potentially require more caregiving), spouses' health investments do not vary by this dimension. Column 5-8 investigate responses by income losses. First, columns 5-6 estimate equation (3) and show that the investments in spousal health are present even though households experience very small income losses (taking into account all income sources and government transfers). Second, columns 7-8 further show that while households in which the sick person was the primary earner experience larger income losses, spousal health investments do not vary by this dimension. These regressions include prime-age sick spouses (of ages 25-55) who were more likely to participate in the labor force in the pre-period, but similar results are found when we include all households and define income shares for each household member using income from any source (not only from labor earnings). 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 4: Family Effects of Cardiovascular Shocks—Different Physicians

	Spouses' Statin Consumption					Adult Children's Statin Consumption				
	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 this table, we analyze only households in which the family members whose behaviors we study do not share the same doctor with the person who 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 healthcare 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 number (where the average number of patients per GP is 1,279), 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 a 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: Robustness Checks 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 and workplace size in our definition of "close" coworkers, which were chosen to balance 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 6: 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 that by individuals whose parent died of any other cause; column 6 compares expenditure on diagnostic radiology by individuals whose parent died of cancer to that by individuals 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 7: Health Conditions following Family Shocks

	Cardiovascular Shocks								Fatal Spousal Shocks	
	Spouses				Adult Children					
	Cardiovascular Disease		Any Hospitalization		Cardiovascular Disease		Any Hospitalization		Any Hospitalization	Major Conditions
	Prime Age	Older	Prime Age	Older	Younger	Older	Younger	Older		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Treat x Post	-.0002	-.0003	.0031	.0006	.00019	.0002	.0016	.0012	.0203***	.0036***
	(.0004)	(.0006)	(.0028)	(.0023)	(.00014)	(.0003)	(.0018)	(.0023)	(.0013)	(.0008)
Counterfactual	.0053	.0194	.3035	.3724	.00144	.0040	.3122	.2700	.3743	.1054
Number of Obs.	441,720	667,980	441,720	667,980	1,179,387	647,667	1,179,387	647,667	2,230,731	2,230,731
Number of Clusters	49,080	74,220	49,080	74,220	67,460	40,690	67,460	40,690	248,126	248,126

Notes: This table reports mean differences-in-differences estimates using equation (3) for the effects of shocks on family members' hospital contacts. The outcome "cardiovascular disease" represents an indicator for contacts related to cardiovascular conditions; the outcome "any hospitalization" represents an indicator for any hospital contact with either an inpatient or an outpatient department; the outcome "major conditions" represents an indicator for a hospital contact related to any severe condition included in the Charlson Comorbidity Index (Charlson et al. 1987): Acute Myocardial Infarction, Cerebrovascular Disease, Chronic Pulmonary Disease, Congestive Heart Failure, Cancer, Dementia, Diabetes with chronic complications, Diabetes without complications, AIDS/HIV, Hemiplegia or Paraplegia, Metastatic Carcinoma, Mild Liver Disease, Moderate or Severe Liver Disease, Peptic Ulcer Disease, Peripheral Vascular Disease, Renal Disease, and Rheumatologic Disease (Connective Tissue Disease). This index is a weighted sum of the number of specific diagnoses in a given year, which was originally designed to predict ten-year mortality and is now widely used as a measure of adverse health (see, e.g., Ho and Pakes 2014 and Finkelstein et al. 2016). Similar results are found when we narrow the analysis to conditions included in the Iezzoni Chronic Conditions (Iezzoni et al. 1994), another widely-studied set of illnesses (see, e.g., Welch et al. 2011 and Finkelstein et al. 2016), as well as when we study the Charlson numerical index itself (instead of illness indicators). 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 References

Charlson, M. E., P. Pompei, K. L. Ales, and C. R. MacKenzie (1987). A new method of classifying prognostic comorbidity in longitudinal studies: Development and validation. *Journal of Clinical Epidemiology 40*(5), 373–383.

Finkelstein, A., M. Gentzkow, and H. Williams (2016). Sources of geographic variation in health care: Evidence from patient migration. *The Quarterly Journal of Economics 131*(4), 1681–1726.

Ho, K. and A. Pakes (2014). Hospital choices, hospital prices, and financial incentives to physicians. *The American Economic Review 104*(12), 3841–3884.

Iezzoni, L. I., T. Heeren, S. M. Foley, J. Daley, J. Hughes, and G. A. Coffman (1994). Chronic conditions and risk of in-hospital death. *Health Services Research 29*(4), 435–461.

Welch, H. G., S. M. Sharp, D. J. Gottlieb, J. S. Skinner, and J. E. Wennberg (2011). Geographic variation in diagnosis frequency and risk of death among medicare beneficiaries. *JAMA 305*(11), 1113–1118.