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# Family Health Behaviors[†]

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*We study how health behaviors are shaped through family spillovers. We leverage administrative data to identify the effects of health shocks on family members' consumption of preventive care and health-related behaviors, constructing counterfactuals for affected households using households that experience the same shock but a few years in the future. Spouses and adult children immediately improve their health behaviors and their responses are both significant and persistent. These spillovers are far-reaching as they cascade even to coworkers. While some responses are consistent with learning information about one's own health, the evidence points to salience as a major operative explanation.* (*JEL* D15, D83, I12, J12)

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, Glaeser, and Rosen 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, Chiappori, and Weiss

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[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.

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 datasets on health and consumption of health care 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.

We begin by documenting the importance of spillovers in health behaviors. Our primary empirical setting is the effects of nonfatal heart attacks and strokes, which are commonly studied as sudden and severe events whose particular timing is likely unpredictable (see, e.g., Chandra and Staiger 2007, Doyle 2011). In the context of these health shocks, we study as our main outcome the consumption of the cholesterol-lowering medication statin, a class of disease-specific risk-reducing preventive care that is among the most widely prescribed medications in the developed world (Redberg and Katz 2016).

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: by the end of our analysis horizon of four years, the impact of the family shock on the consumption of preventive care amounts to an increase of 15 percent among prime-age spouses and to increases of 37 percent and 16 percent among younger and older adult children, respectively. We further find that these spillovers in health care consumption are far reaching across one's network with a meaningful implied multiplier, as they cascade even to coworkers, who exhibit long-lasting responses of the same order of magnitude as spouses.

In support of these findings, we provide additional analysis of spillovers in behaviors within a complementary setting of fatal events, focusing on spouses. We show that fatal family events lead to considerable behavioral improvements in the form of

[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, Stimpson, and Peek 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.

decreases in key harmful habits (smoking and usage of prescription opioids), as well as to increases in general awareness regarding own health.[4]

In the second part of the paper, we investigate the mechanisms by which shocks can trigger the changes in health behaviors that we identified among family and network members. We offer various tests to distinguish between two major classes of mechanisms: learning new information about one's own health risks, and the salience of health shocks itself. Our focus on these channels is guided by the literature and the empirical patterns; of course, alternative mechanisms could also be at play, some of which we show can be likely ruled out.

We find some evidence in support of learning as an explanation. We show that spouses across predictable risk types are similarly prompted to seek information about their own cardiovascular risk (via medical testing), but that those with higher baseline risk end up having larger statin responses. 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 responses in preventive behaviors. Likewise, as premature parental cardiovascular shocks carry more information on a child's biological risk (De Backer et al. 2003, Pencina et al. 2014), we find that children whose parents were younger at the time of shock are more likely to increase their consumption of statins as preventive care.

More so, there is consistent evidence in support of salience as a key operative channel. We exploit the richness of the data to provide three sets of supportive findings. First, we study settings with distinctly limited scope for learning new information. We show similarly large increases in the consumption of statins by spouses who had been tested for cholesterol levels in the pre-shock period and hence had already gained access to the information relevant for their cardiovascular risk. We also provide various examples within our complementary application of spousal death, where the exact nature of the event is unlikely to reveal new information.

Second, we show that family members' attention is particularly drawn to the local nature of the health event, so their behavioral responses target the domain of the specific experienced risk. Individuals whose spouse died of cancer, as compared to any other cause, 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. We further show that the behavioral changes are local to the vicinity of the experienced risk's domain by studying cross-condition responses. Interestingly, in the context of fatal cancer, we find some evidence of 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 health event, 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.

[4] 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, e.g., a review in Stahl and Schulz 2014). These papers use designs different from ours and generally compare outcomes before and after bereavement 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 behaviors, where an exception is Jin and Christakis (2009).

Third, we utilize circles in one's network that are purely social to investigate the role of the degree of exposure to the shock and its prominence, by exploiting various measures of "distance." We show that sons- and daughters-in-law increase their take-up of preventive care following the parent-in-law cardiovascular shock, and that, consistent with exposure intensity, this effect is driven by those who live closer to their in-laws. We also show that whereas there are meaningful spillovers in health care consumption among close coworkers (defined by similarities in age/occupation), there are no such effects on socially distant coworkers.

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). Our results point to a role of one's network in health-related habit formation and underscore the importance of incorporating interpersonal interactions across different family generations and peers in analyzing health behaviors, investments, and the consumption of health care. The response patterns also highlight learning and, specifically, salience and attention as key modeling components (as in, e.g., DellaVigna 2009; Bordalo, Gennaioli, and Shleifer 2012, 2013; and Gabaix 2019),[5]and point to event-driven responses, in line with frameworks such as Bernheim and Rangel's (2004) cue-triggered decision-making model. More generally, the results additionally advance our understanding of the nature and scope of broader network effects in consumption, here through the lens of health care utilization.[6]

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 health care 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 I we describe our empirical strategy. Section II outlines the data sources we use, the empirical setting, and the analysis sample. The evidence on spillovers in health behaviors is presented in Section III, and Section IV investigates their underlying mechanisms. Section V discusses the implications of our findings and concludes.

[5]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 on how limited attention and salience affect economic behavior in a variety of settings; see DellaVigna (2009) and Gabaix (2019) for reviews.

[6]De Giorgi, Frederiksen, and Pistaferri (forthcoming) 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.

## I. 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.

### A. *Primary Quasi-Experiment*

The ideal experiment for identifying behavioral responses to family health shocks would randomly assign shocks to families and track responses in health behaviors over time. Such an experiment would compare the ex post responses to shocks by affected households to a counterfactual behavior of ex ante similar unaffected households. To approximate this ideal experiment, we utilize a quasi-experimental research design that exploits the potential randomness of the timing of a severe health shock within a short period of time. 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 $\tau$, and a matched control group, composed of family members from the same cohorts in households that experience the same shock but in year $\tau + \Delta$. 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 $\Delta$ weighs households' comparability (which is greater when $\Delta$ is smaller) against analysis horizon (which is greater when $\Delta$ is larger).[7] Our choice of $\Delta$ is five years, such that we can identify effects up to four years after the shock (since the control group becomes "treated" in period $\Delta$). We assess the robustness of our analysis to this choice and find that local perturbations to $\Delta$ provide similar results.[8]

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 $\Delta$, 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 comovement 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.

[7] For more details on the research design and its underlying trade-offs, see Fadlon and Nielsen (2017).
[8] This is reported in online Appendix Table 8.

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 window.

The way the control group is defined means that family members in the control group experience the same shock as those in the treatment group, but at a later age, which generates a systematic difference between the treatment and control groups. To address this issue, we construct as a robustness check an alternative control group that matches households on observable pre-shock risk factors. This approach expands the control group to a broader set of comparable households, which includes those who never experience the health shock despite having similar underlying risk, as well as those who experience the shock but within other $\Delta$ bandwidths. A detailed description of the matching procedure that we use is provided in online Appendix B. This alternative design leads to similar findings, which are reported in online Appendix Table 9. The remainder of this subsection formalizes the main research design and describes our estimator.

*Formal Description of the Main Design and Estimating Equation.*—Fix a group of cohorts, denoted by $\Omega$, and consider estimating the treatment effect of a shock experienced at some point in the time interval $[\tau_1, \tau_2]$ by individuals whose family members belong to group $\Omega$. We refer to these individuals' family members as the treatment group and divide them into subgroups 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 $\tau$ the family members (from the same cohort group $\Omega$) of individuals who experienced the same shock but at $\tau + \Delta$, for a given choice of $\Delta$. 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 $t = \Delta$).

Our estimating equation is a dynamic difference-in-differences specification of the following form:

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

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 $\delta_r$, which estimate the period $r$ treatment effect ($r > 0$) relative to the pre-period $-1$. Validation of the parallel trends assumption requires that $\delta_r = 0$ 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 level.

To quantify mean treatment effects or for increased precision in the analysis of distant circles and smaller subsamples, we estimate the standard

difference-in-differences equation of the following form, which averages over years before and after the shock:

$$y_{i,t} = \alpha + \beta treat_i + \gamma post_{i,t} + \delta treat_i \times post_{i,t} + \lambda X_{i,t} + \varepsilon_{i,t}. \tag{2}$$

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

### B. *Response Heterogeneity*

There are two related specifications that we employ when analyzing heterogeneity in treatment effects to shed light on mechanisms.

*Heterogeneity Specification 1.*—We use the first specification 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 (2) in the following way:

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

where

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

We adjust the basic difference-in-differences design by allowing the treatment effect, $\delta_i$, to vary across households and model it as a function of the household's characteristic $z_i$. Our parameter of interest is $\delta_1$, which captures the extent to which the family member's response correlates with $z_i$.[9]

*Heterogeneity Specification 2.*—We use the second specification 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:

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

Equation (4) is similar to equation (1) but where $treat_i$ is replaced with $z_i$, such that the treatment and control groups are now defined as households with different values of $z_i$ within the group of treated households. The key parameters of interest are $\delta_r$ for $r > 0$, which estimate how the outcomes for treated households with varying

[9] In the estimation of (3) we always include in the vector $X_{i,t}$ the variables in $z_i$ as well as their interaction with $treat_i$ and $post_{i,t}$.

levels of $z_i$ differentially evolve around the event (relative to the baseline period $-1$). The same identifying assumption is required in the analysis of these experimental groups, i.e., that absent the event, the outcomes of households with different values of $z_i$ would run parallel. As before, an implied necessary condition is that $\delta_r = 0$ for $r < 0$. Lastly, the corresponding equation that estimates how responses vary on average across treated households with different levels of $z_i$ takes the form

$$y_{i,t} = \alpha + \beta z_i + \gamma post_{i,t} + \delta \times z_i \times post_{i,t} + \lambda X_{i,t} + \varepsilon_{i,t}, \tag{5}$$

where $\delta$ is the parameter of interest.

## II. Data, Setting, and Analysis Sample

We use administrative data on all Danish households from several registers that span the years 1980 to 2011. The Danish setting 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 health care 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 health care system is the provision of near-complete and universal health care coverage. Importantly, this enables us to identify effects that are not confounded by the availability of health insurance. Detailed institutional background is provided in online Appendix A. Below, we describe our data sources, empirical setting, and analysis sample.

### A. *Data Sources*

For utilization outcomes that measure health behaviors, we use three databases that encompass both primary and secondary health care utilization records with exact dates and codes. These include (i) the *Prescription Drug Database*, covering all prescribed drugs that were purchased from 1995–2011, with detailed information on classification of medicine (using the Anatomical Therapeutic Chemical (ATC) classification system); (ii) the *Health Insurance Registry*, covering all individual contacts with primary care physicians and medical care specialists outside of hospitals from 1985–2011; and (iii) 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).

For identifying family health events and their timing, we use hospitalization dates and diagnoses from 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 health care 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.

### B. *Empirical Setting and Analysis Sample*

In our primary setting of nonfatal health shocks, the sample is comprised of all households in which an individual experienced a heart attack or a stroke for the first time and survived for the four-year analysis horizon (that corresponds to $\Delta = 5$). We focus on heart attacks and strokes as our leading health events since they are commonly studied as sudden and severe events whose particular timing is likely unpredictable, and are therefore 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 statin, 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.[10] The main close family circles that we study are spouses (based on matches of all married and cohabiting couples prior to the shock) and their adult children (based on matches available for individuals born after 1960).

Our focus on nonfatal shocks stems from the opportunity to study a setting in which behavioral responses take the form of health investments; that is, of preventive actions against existing underlying risks. This is because some preventive care can be also consumed for managing own severe health conditions and we want to analyze a setting in which shocks do not induce such conditions. The literature (see, e.g., Stroebe, Schut, and Stroebe 2007 for a review) and our own investigation show that death events can lead to severe health conditions, specifically among spouses. This is in contrast to nonfatal cardiovascular shocks, where we find no evidence for such effects on family members. We verify that family members' statin consumption responses are preventive health investments by showing there is no increase in the incidence of own cardiovascular shocks (or of hospital contacts of any type) among spouses or children following the nonfatal cardiovascular shocks that we analyze. See online Appendix Table 7 for details on these analyses. We note, however, that all the shocks we study can certainly have various health effects, e.g., on the mental health of family members due to stress induced by the adverse family health event.

We additionally study two distant circles of family members and peers. The first is the sample of sons- and daughters-in-law (to whom we collectively refer as "children-in-law"). The second is the sample of 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

[10] 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 United States (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).

interact, we focus on *close* coworkers in the following way.[11] 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 twenty-fifth percentile (of approximately 20). We then identify their coworkers who have been employed in a similar occupation class,[12] and who are close to these individuals in terms of age (with an age gap of 5 years or less).[13] We exclude from this sample any coworker who is also a family member.

In our secondary setting of death events the sample includes all families in which a member died between 1985 and 2011. For these events we study spouses and children, whose respective samples are constructed in the same way as before. Online Appendix Table 1 summarizes the various analysis samples that we analyze and reports summary statistics.

## III. Spillovers in Health Behaviors

We now turn to our analysis of how health behaviors are shaped through family spillovers by studying intra-generational effects on spouses and inter-generational effects on adult children. To further investigate the breadth of spillovers, we also analyze whether and to what degree the effects cascade to peers. In the core part of our analysis, we study the effects of nonfatal heart attacks and strokes on family members' consumption of preventive care that is specific to these cardiovascular conditions. In complementary analysis, we study the effects of general fatal events on family members' behaviors by analyzing overall changes in consumption of health care that is indicative of health behaviors and awareness. We focus on reporting and assessing the estimated effects in relative terms compared to counterfactual or baseline levels, in order to account for the underlying prevalence of the health care utilization outcomes we analyze within each subpopulation.

### A. *Effects of Severe Nonfatal Health Shocks*

Cardiovascular shocks are directly tied to a disease-specific risk-reducing medication, statin, so we focus on its consumption as a main behavioral outcome. This class of medication is composed of prescription drugs taken to lower cholesterol that are pervasively used to prevent cardiovascular disease. We therefore proceed with analyzing how individuals' cardiovascular shocks affect the consumption of statins by members of their network.

*Intra-Generational Effects on Spouses*.—We begin with studying spouses to identify family spillovers that cannot be attributed to biological channels. To visualize the estimation strategy, we provide figures that plot the raw data for the main

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

[12] 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).

[13] Estimations that perturb the thresholds of workplace size and age gap are provided in online Appendix Table 5.

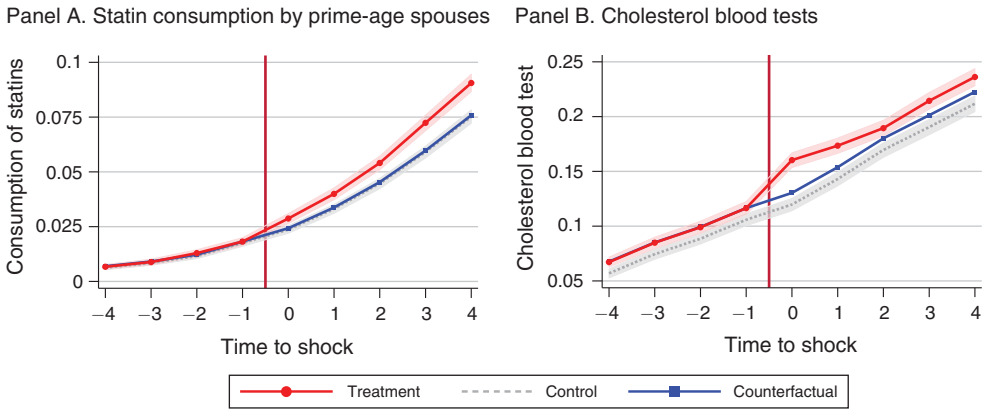


FIGURE 1. EFFECTS OF CARDIOVASCULAR SHOCKS ON SPOUSAL CONSUMPTION OF PREVENTIVE CARE

*Notes:* These figures display changes in spouses' consumption of preventive care in response to family cardiovascular shocks by plotting means of the raw data. 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 (along with the corresponding 95 percent confidence intervals). 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 (along with the corresponding 95 percent confidence intervals).

circles of spouses and children where ties are strongest and samples are largest, and we then turn to regression estimations for the remainder of the analysis. 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 (1) 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 about 15 percent, an increase of 1.17 percentage points (pp) on a base of 7.86 pp.

Table 1—Dynamic Family Effects of Cardiovascular Shocks

| | Spouses' statin consumption | | | Adult children's statin consumption | |
|---|---|---|---|---|---|
| | Prime age (Ages 25–55) (1) | Older (Ages 55–85) (2) | Spouses' cholesterol testing (3) | Younger (Ages 25–40) (4) | Older (Ages 40–65) (5) |
| Time to shock: | | | | | |
| −4 | 0.00052 | −0.00072 | −0.00138 | 0.00012 | −0.00000 |
| | (0.00104) | (0.00158) | (0.00441) | (0.00024) | (0.00065) |
| −3 | 0.00026 | −0.00063 | −0.00114 | 0.00008 | −0.00014 |
| | (0.00096) | (0.00141) | (0.00456) | (0.00023) | (0.00060) |
| −2 | 0.00102 | −0.00072 | −0.00056 | 0.00015 | 0.00071 |
| | (0.00079) | (0.00113) | (0.00447) | (0.00020) | (0.00049) |
| −1 | 0 | 0 | 0 | 0 | 0 |
| | 0 | 0 | 0 | 0 | 0 |
| 0 | 0.00386 | 0.00234 | 0.03033 | 0.00067 | 0.00150 |
| | (0.00095) | (0.00128) | (0.00498) | (0.00023) | (0.00061) |
| 1 | 0.00511 | 0.00930 | 0.02052 | 0.00173 | 0.00429 |
| | (0.00134) | (0.00179) | (0.00516) | (0.00033) | (0.00089) |
| 2 | 0.00702 | 0.01093 | 0.01100 | 0.00245 | 0.00433 |
| | (0.00169) | (0.00219) | (0.00545) | (0.00043) | (0.00113) |
| 3 | 0.01012 | 0.01036 | 0.01572 | 0.00279 | 0.00765 |
| | (0.00203) | (0.00255) | (0.00570) | (0.00053) | (0.00135) |
| 4 | 0.01166 | 0.01230 | 0.01720 | 0.00433 | 0.00799 |
| | (0.00234) | (0.00284) | (0.00624) | (0.00063) | (0.00159) |
| Treat | −0.00120 | −0.00115 | −0.00043 | −0.00030 | 0.00005 |
| | (0.00119) | (0.00187) | (0.00393) | (0.00025) | (0.00075) |
| Counterfactual at $t = 4$ | 0.07863 | 0.22842 | | 0.01180 | 0.04933 |
| Percent change | 14.83 | 5.38 | | 36.69 | 16.20 |
| Counterfactual at $t = 0$ | | | 0.12998 | | |
| Percent change | | | 23.33 | | |
| Observations | 441,720 | 667,980 | 214,793 | 1,179,387 | 647,667 |
| Number of clusters | 44,302 | 65,661 | 20,997 | 67,460 | 40,690 |

*Notes:* This table reports dynamic difference-in-differences estimates for the evolution of household responses to cardiovascular shocks using specification (1). It displays estimates for the $\delta_r$ 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 also report the estimate for the coefficient $\beta$ on the variable $treat_i$. Counterfactual levels, calculated using specification (1), are reported for assessing response magnitudes relative to underlying levels. We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report in parentheses robust standard errors clustered at the household level.

Older spouses have much more frequent interactions with the medical system (see online 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 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 online Appendix Figure 2).

For a subset of our sample, specifically those who reside in Greater Copenhagen, the data also include indicators for blood tests of cholesterol levels. As these tests are

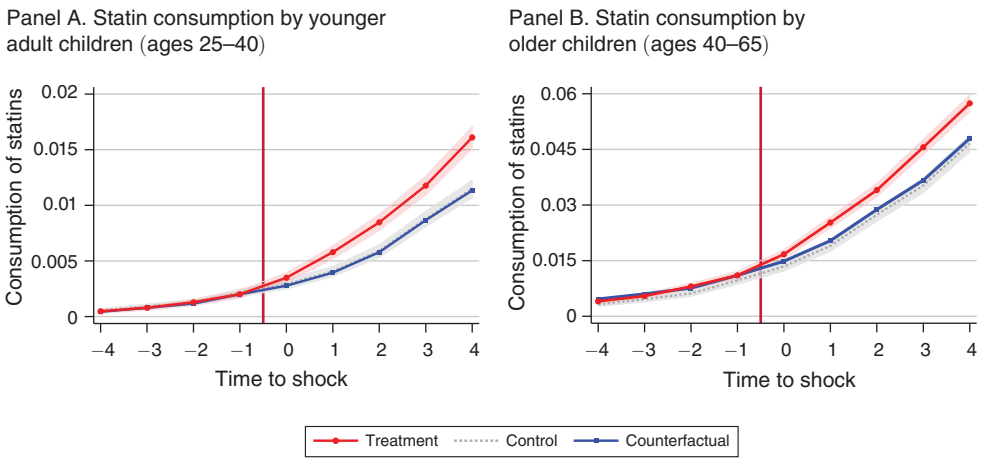


FIGURE 2. EFFECTS OF CARDIOVASCULAR SHOCKS ON ADULT CHILDREN'S CONSUMPTION OF PREVENTIVE CARE

*Notes:* These figures display changes in adult children's consumption of preventive care in response to family cardiovascular shocks by plotting means of the raw data. 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 (along with the corresponding 95 percent confidence intervals). 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 (along with the corresponding 95 percent confidence intervals).

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 23 percent. 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 higher also in the following years. This is in line with continuous monitoring tests that are common in medication maintenance for those who have started consuming statins, and also mirrors the growing post-shock share of spouses who take up statins, as manifested by the gradual growth in the estimated statin consumption effect.

*Inter-Generational Effects on Adult Children.*—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. Specifically, we analyze adult children's statin consumption responses to parental heart attacks or strokes at different stages of the life cycle. 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

Table 2—Effects of Cardiovascular Shocks on Statin Consumption by Coworkers

| | | Distant coworkers | | |
|---|---|---|---|---|
| | Close coworkers | Larger workplaces | Large age gap | Different occupation |
| | (1) | (2) | (3) | (4) |
| Treat × post | 0.01349 | 0.00143 | 0.00297 | 0.00350 |
| | (0.00559) | (0.00330) | (0.00335) | (0.00524) |
| Counterfactual | 0.08442 | 0.08249 | 0.06040 | 0.08871 |
| Percent change | 15.98 | | | |
| Observations | 49,336 | 131,488 | 87,704 | 53,312 |
| Number of clusters | 3,498 | 4,057 | 4,744 | 4,071 |

*Notes:* This table reports difference-in-differences estimates for coworkers' statin consumption responses to cardiovascular shocks using specification (2). Counterfactual levels in the periods following the shock are calculated using this estimation. *Close coworkers* are defined as coworkers within the same occupation class and age range (with an age gap of 5 years or less) in smaller workplaces (in which the number of employees was equal to or lower than the sample's 25th percentile). *Distant coworkers* are defined as workers within the same workplace who are more "distant" along at least one of these dimensions (as indicated at the top of columns 2–4). We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report in parentheses robust standard errors clustered at the workplace level.

over time. Estimating equation (1), 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 37 percent. 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 percent by the fourth year after the shock (see column 5 of Table 1).

*Effects on Peers*.—To study how far the spillovers of health shocks to improved health behaviors can reach, we analyze the effects on coworkers. We approximate peers with whom individuals are more likely to interact and focus the analysis on close coworkers (as defined in Section IIB). Column 1 of Table 2 displays the average treatment effect of health shocks on close coworkers' consumption of preventive care. The results indicate an increase of 1.35 pp, which amounts to an effect of 16 percent. As this effect is economically considerable, it is also meaningful to estimate the dynamic regression despite the small sample. Column 1 of online Appendix Table 10 indicates that 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.

### B. *Effects of Fatal Health Events*

Lastly, we investigate family spillovers in health behaviors in the extreme context of death. 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 health care 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 online Appendix Table 6).

*Increased Awareness of Health.*—We begin by analyzing the effects of death events 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 alarms." For visual clarity of the dynamic patterns that we find in this subsection, we report our findings by plotting the $\delta_r$ coefficients from specification (1) along with their 95 percent confidence intervals. We also indicate on the figures the counterfactual outcome levels for periods $t = 0$ and $t = 4$, the beginning and end of the analysis period, to gauge response magnitudes relative to underlying levels.[14]

Panel A of Figure 3 displays spousal responses using our first proxy for health alertness. The figure shows that, in the immediate years just after their spouse's death, individuals' propensity to visit hospitals on account of suspected health conditions clearly and meaningfully increases. This effect seems to dissipate over time, although some 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.[15] Panel B of Figure 3 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 event.

*Declines in Harmful Behaviors and Medication.*—A large share of the literature on adverse health behaviors has focused on smoking and on 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 health events as additional measures for improvements in health behaviors.

We first explore the consumption of medication that treats nicotine dependence.[16] This class of medication is prescribed to individuals engaged in chronic smoking who wish to cease their unhealthy behavior or to switch to a less damaging substitute (Siu 2015; Swift and Aston 2015).[17] Panel C of Figure 3 displays estimates of

[14] In this subsection, we analyze a variety of utilization codes that cover different calendar years (due to institutional changes in data reporting). Therefore, we rely here on specification (1) 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 that correspond to Figure 3, which we provide in panels A–D of online Appendix Figure 3.

[15] 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.

[16] Relatedly, Khwaja, Sloan, and Chung (2006) study associations between smoking and spousal health in the Health and Retirement Study.

[17] This class of prescription drugs for smoking cessation is labeled under ATC code N07BA. It includes medications and nicotine replacement therapies (such as nicotine chewing gum and patches), which are widely recommended to all adults as part of cessation regimens.

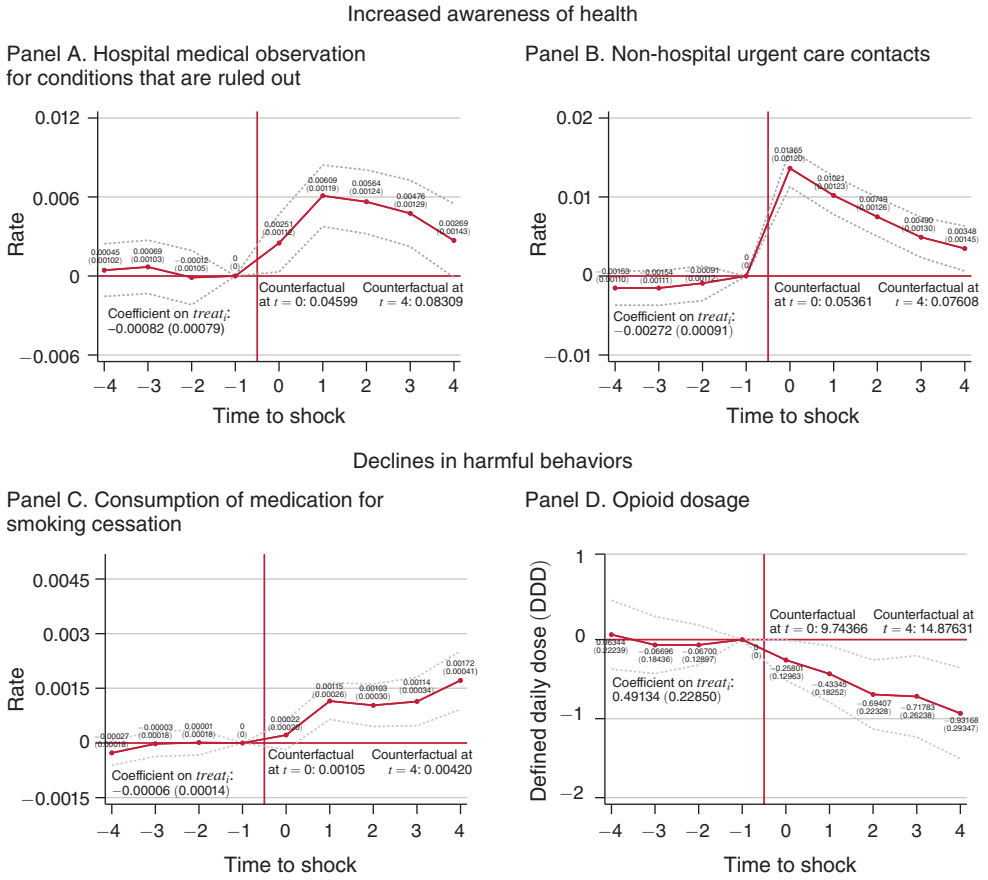


FIGURE 3. EFFECTS OF SPOUSAL DEATH ON HEALTH BEHAVIORS

*Notes:* These figures display changes in health behaviors in response to spousal death by plotting the dynamic difference-in-differences estimator of equation (1). The figures plot the estimates for $\delta_r$ along with their 95 percent confidence intervals. We also report the estimate for the coefficient $\beta$ on the variable $treat_i$. We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report in parentheses robust standard errors clustered at the household level. In each panel, we indicate benchmark levels for period $t = 0$ (the beginning of the analysis period) and period $t = 4$ (the end of the analysis period) based on counterfactual calculations from specification (1), to gauge response magnitudes relative to underlying levels.

equation (1), where the outcome variable is an indicator for the purchase of a prescription drug for smoking cessation or smoking substitutes. The estimates show increases in individuals' consumption of this class of medication in the years following a spousal death, which average to about 50 percent in the post-shock period (years 1–4) compared to the counterfactual.

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). In this case, health-promoting behavioral responses would translate into reductions in consumption. Since opioid dose reductions should be gradual due to withdrawal symptoms (Miller and Kipnis 2006, Volkow 2014), we

study a continuous measure of dosage. 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.[18] Panel D of Figure 3 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 gradually reaches a decrease of 0.93 days by the fourth year after the shock.

## IV. Mechanisms

Having documented the importance of spillovers in health behaviors, we now move on to their interpretation through the lens of possible models. Guided by the literature and the empirical patterns, we conceptualize two major classes of mechanisms by which adverse health events within the network can trigger changes in health behaviors. The first channel is potential learning and revelation of new information about one's *own* risk. For example, adult children may be induced to learn of their genetic risk or about shared environmental risk while growing up, whereas spouses may be exposed to information on joint behavioral risks. The second channel is health salience itself, where one's attention may be drawn to the health domain even when the family event is unlikely to reveal new information.

In this section, we explore the nature of the spillovers by offering various ways to distinguish between these two mechanisms. We show that while learning information about one's own health seems to be at play, there is consistent evidence in support of salience as a key operative explanation. Of course, alternative mechanisms could be at play, which we investigate at the end of this section.

### A. *Evidence Consistent with Learning New Information*

We begin by providing evidence in support of learning as an explanation for the effects of family cardiovascular shocks on improved health behaviors among spouses. We first ask the following 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 our 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 available 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

[18] 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 health care environments.

Table 3—Heterogeneity in Family Effects of Cardiovascular Shocks

| | Spouses' statin consumption (1) | Spouses' statin consumption (2) | Spouses' statin consumption by previously tested (3) | | Adult children's statin consumption (4) |
|---|---|---|---|---|---|
| Treat × post | 0.00305 | 0.00432 | 0.01748 | Treat × post | −0.00023 |
| | (0.00158) | (0.00204) | (0.00883) | × parent's age | (0.00008) |
| Treat × post | 0.01089 | 0.01075 | | Treat × post | 0.00059 |
| × high risk | (0.00242) | (0.00303) | | × own age | (0.00012) |
| Observations | 954,256 | 715,692 | 43,664 | | 1,548,616 |
| Number of clusters | 103,859 | 103,859 | 5,170 | | 84,794 |

*Notes:* This table studies heterogeneity in family responses to cardiovascular shocks along different dimensions. Column 1 estimates equation (3) 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 provides an analysis similar to that in column 1, but where we define the post-shock period as years 3 and 4 when the responses in statin consumption level out (as seen in Figure 1). Column 3 estimates equation (2) and analyzes statin consumption responses by spouses whose cholesterol levels had been already tested for in the pre-shock periods. In column 4 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 (3), 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 in parentheses robust standard errors clustered at the household level.

probability of a cardiovascular shock in the baseline period $t = -1$, and split the sample into high and low risk using the population median.[19]

Column 1 of Table 3 estimates equation (3), where $z_i$ denotes an indicator for whether the spouse's predicted risk is above the median. We find that even low risk spouses exhibit some increase in their take-up of statins as preventive care in response to the family shock. However, at the same time, high risk spouses are much more likely to increase their statin consumption, by an additional 1.09 pp. Analogous patterns are displayed in column 2, where we define the post-shock period as years 3 and 4 when the responses in statin consumption level out (as seen in Figure 1). Nonetheless, we find no such risk gradient in the spouses' information-seeking behavior through cholesterol testing (see column 1 of online Appendix Table 2). 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 underlying risk end up having larger statin responses, consistent with learning induced by the family shock about own health risks. 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 online 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.

[19] Specifically, we use data on the entire adult population to estimate a probit model for the one-year probability of experiencing a first heart attack or stroke. We include as right-hand-side variables age fixed effects, gender, year fixed effects, education, and lagged hypertension and diabetes statuses (based on consumed medications). For each individual, we then predict the probability of experiencing the first heart attack or stroke based on this model.

We find similar patterns in the responses of children. 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 4 of Table 3 estimates equation (3), 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.

### B. *Evidence Consistent with Salience and Attention*

We now turn to present evidence in support of salience and attention as a key operative channel underlying the spillovers in health behaviors within one's network. We provide three sets of supportive findings with analyses that aim to proxy for: the scope of pre-shock knowledge of health risks and of potential learning; the extent to which responses are directed to the particular experienced risk and are local to it; and the degree of exposure to the shock and its prominence.

*Limited Scope for Learning*.—We begin by further exploiting the data on cholesterol testing in the context of spousal cardiovascular shocks. 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 have discussed, 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.[20] 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 3 of Table 3). 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.

In support of these findings, we additionally provide several examples in the context of spousal death, where the event itself is not likely to directly carry new information on the studied health behavior outcome. In particular, we find that husbands whose wives died of female cancers considerably increase their expenditure on cancer diagnoses, although the spouse's cancer type is not likely informative of their own cancer risk (which we verify in the data). We provide details below within the analysis of condition-specific preventive care. We also study the effects of spousal death on the consumption of medication for smoking cessation for a small class

[20] This is not the case for family circles with first-degree biological links, such as children, for whom the guidelines indicate that eligibility should also incorporate family history.

of causes of death, autoimmune diseases, that the medical research has not been able to link to any risky behavior and specifically to smoking (NIH 2016). We find that even in these cases, survivors engage in treatments to reduce smoking (see column 1 of online Appendix Table 6).[21]

*Locality of Behavioral Responses to Experienced Risk*.—Next, we present evidence that agents' attention is specifically drawn to the local nature of the experienced family health event so they take actions particular to its risk's domain. To investigate the directedness of spillover responses, we study the consumption of condition-specific preventive care by exploiting variation in causes of death among treated households in our death event setting.[22] 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 event? 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 (4) 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 nonfatal cardiovascular shocks. Column 1 of Table 4 estimates equation (4) and clearly shows that following spousal death, 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. 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 4).

Next, we study our second class of preventive care and analyze health care costs associated with visits to diagnostic radiologists in the context of cancer death. Since visits to radiologists are for diagnosis purposes only (rather than for continuous health risk management), behavioral responses in the current application should likely translate to transitory effects. We estimate the corresponding regression in column 3 of Table 4 and find that following a spousal death of cancer, individuals' health care expenditure on diagnostic radiology significantly increases compared to

[21] In addition, we show that spousal death induces declines similar to those documented in Section IIIB in the consumption of opioids when we exclude prescription opioid poisoning as a cause of death, so that the event itself is not directly tied to the studied behavioral response (see panel E of online Appendix Figure 3).

[22] This also allows us to take a step toward isolating behavioral responses in the context of spousal death, where the event can lead to severe health conditions (see online Appendix Table 7). The underlying idea is that all spouses among treated households are exposed to the main effects of spousal death, but their experience may differ by the cause of death. Among different alternative explanations, this analysis reduces the plausibility that potential complementarities between treatment and preventive care explain the findings of the effects of spousal death on health behaviors.

Table 4—Condition-Specific Preventive Care Following Fatal Events

| | Spousal statin use when cause of death is cardiovascular (1) | Children's statin use when cause of death is cardiovascular (2) | Spouses' diagnostic radiology when cause of death is cancer (3) | Spouses' hospital contact with family cancer code when C.O.D. is cancer (4) |
|---|---|---|---|---|
| Time to shock: | | | | |
| −4 | 0.00208 | −0.00076 | −0.50692 | −0.00005 |
| | (0.00204) | (0.00056) | (0.52221) | (0.00006) |
| −3 | 0.00007 | −0.00060 | 0.17280 | −0.00006 |
| | (0.00181) | (0.00051) | (0.52134) | (0.00007) |
| −2 | 0.00135 | −0.00022 | −0.22732 | −0.00000 |
| | (0.00140) | (0.00042) | (0.51411) | (0.00008) |
| −1 | 0 | 0 | 0 | 0 |
| | 0 | 0 | 0 | 0 |
| 0 | 0.00357 | 0.00196 | 0.63333 | −0.00002 |
| | (0.00148) | (0.00053) | (0.53163) | (0.00007) |
| 1 | 0.00466 | 0.00203 | 1.54639 | 0.000192 |
| | (0.00197) | (0.00071) | (0.54724) | (0.000085) |
| 2 | 0.00496 | 0.00333 | 1.23071 | 0.00000 |
| | (0.00228) | (0.00088) | (0.55681) | (0.00008) |
| 3 | 0.00618 | 0.00491 | 0.92725 | −0.00000 |
| | (0.00252) | (0.00103) | (0.56761) | (0.00009) |
| 4 | 0.00710 | 0.00661 | 0.59471 | 0.00011 |
| | (0.00274) | (0.00117) | (0.58510) | (0.00010) |
| Baseline at $t = -1$ | 0.06520 | 0.01364 | 12.22532 | 0.000119 |
| Observations | 874,661 | 2,922,141 | 2,382,999 | 1,524,096 |
| Number of clusters | 124,824 | 167,586 | 303,192 | 213,925 |
| Households with condition | 13,589 | 38,076 | 107,565 | 76,101 |

*Notes:* This table reports dynamic difference-in-differences estimates for the evolution of household responses around death events using specification (4). The regressions are estimated on treated households only, where we divide the sample by cause of death. We indicate baseline levels in period −1 among the subsample of households with the studied cause of death. The table displays estimates for the $\delta_r$ parameter vector of the interaction between cause of death indicators and the indicators for time with respect to the event 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 health care 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 in parentheses robust standard errors clustered at the household level.

a spousal death of any other cause. This temporary differential response amounts to an increase of 12.6 percent relative to the baseline period ($t = -1$). 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 4).

To further investigate how directed the responses are toward particular risks, we look into specific types of cancer. For this analysis, as we investigate thinner subsamples, we estimate the average difference-in-differences type specification of equation (5) that aggregates pre- and post-shock years, where now $z_i$ further splits

the sample by particular cancer types. When we study expenditure on diagnostic radiology as an outcome we define the post-shock period as years 1 and 2 in which the dynamic specification identified differential behavior. 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. We find that individuals whose spouse died of lung cancer are indeed more likely to engage in smoking cessation following the event compared to those whose spouse died of any non-cancer cause (column 1 of Table 5). 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 5).[23]

The second class of cancer types that we study is female cancers, which in our sample include ovarian, cervical, and breast cancer.[24] 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. A key advantage of this context is the limited potential for information revelation regarding husbands' own risk of cancer, as we verify in the data.[25] Still, we find responses that are very similar in magnitude to what we have found so far, so that husbands whose wife has died of female cancer meaningfully increase their expenditure on diagnostic radiology in the years just after the event relative to those whose wife has died of any non-cancer cause (column 3 of Table 5). 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 5).[26]

Whereas the within-condition analysis suggests that spillover responses target the experienced risk, cross-condition analysis can further indicate how local they are to its vicinity.[27] 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

[23] Not surprisingly, the baseline consumption rate of smoking cessation medication, which is related to the underlying prevalence of the smoking behavior itself, is higher among individuals whose spouse died of lung cancer as compared to those whose spouse died of non-lung cancer which could reflect joint behaviors and correlated risks.

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

[25] Specifically, we study in the cross section of households with deceased wives whether a wife's (future or experienced) death of female cancers can be predictive of husbands' contacts with inpatient or outpatient hospital departments for any cancer-related reason (beyond potential diagnostic tests). Regressions that include our set of controls find quite precise zeros, whether we use the post-shock period (−0.0003, SE 0.0015) or the pre-shock period (−0.0014, SE 0.0013); regressions of raw correlations with no controls actually produce negative estimates (−0.0045 with SE of 0.0012 for post-shock periods; −0.0096 with SE 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.

[26] We were unable to conduct 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.

[27] This analysis can also serve as 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 risk.

Table 5—Health Behaviors Following Spousal Death

| | 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. × post | 0.00062 | 0.00017 | 1.51393 | 0.00027 | 0.106547 | −0.004494 |
| | (0.00031) | (0.00020) | (0.75458) | (0.00154) | (0.669330) | (0.001508) |
| Baseline $t = -1$ | 0.00060 | 0.00035 | 7.033021 | 0.02001 | 10.76727 | 0.07928 |
| Observations | 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 difference-in-differences estimates for responses in health behaviors around spousal death using different specifications of equation (5). 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 indicate baseline levels in period −1 among the subsample of households with the studied cause of death. We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report in parentheses robust standard errors clustered at the household level.

cause, but where the outcome variable is instead a measure of preventive care that is particular to a different disease $x'$.[28]

Column 5 of Table 5 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 risk. Moreover, for cross-condition responses in the context of cancer, we even find some evidence of declines (see column 6 of Table 5). That is, individuals whose spouse died of cancer exhibit decreased propensity to consume preventive care against cardiovascular disease following the spousal death. Note that this response is economically small and is on the order of 5.7 percent relative to baseline levels. Nonetheless, we view its importance as pointing 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 health event, even when it is 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 only suggestive, can have implications for pitfalls in leveraging salience as a policy tool, which we discuss in the conclusion.

[28] 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 disease are less likely to visit diagnostic radiologists, it could be driven by the fact that 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).

*Degree of Network Member's Exposure*.—Lastly, we exploit the more distant circles in one's network, who are only socially tied as adults, to explore how the extent of the shock's prominence can play a role in health behavior spillovers. We use different measures of "distance," either physical or social, to proxy for the shock's salience and the degree to which network members are exposed to the shock within our main setting of cardiovascular health events.

We begin with the children's households by analyzing sons- and daughters-in-law, who comprise a next generation circle that is not subject to potential learning about biological risk or about risk related to the family environment shared in childhood. Column 1 of Table 6 estimates the spillover effect using equation (2). It shows that, following the cardiovascular shock to their spouse's parent, children-in-law exhibit an average increase in statin consumption that amounts to about one-quarter of that of their spouses, i.e., the adult children (for whom the estimate is reported in column 2 of Table 6). Whereas this effect is small in magnitude, its importance lies in revealing the breadth of the spillovers that we identify, in a context where salience and increased awareness could be a likely explanation. To explore this conjecture, we further test whether distance across households matters. As the simplest measure for distance that may capture the degree of exposure to the shock and its salience, we look at variation in geographical distance based on municipality of residence in the baseline period $t = -1$.[29] Specifically, we divide the sample into children-in-law who live closer to or further from their parents-in-law using the median distance.[30] Column 4 of Table 6 provides the spillover effect for each subsample split by distance, calculated using a regression of specification (3). The results reveal that the spillover effect on in-laws is driven by the next generation households who live closer to their spouses' parents, who respond with a meaningful increase of about 12.5 percent (on a counterfactual of 1.96 pp).[31] Interestingly, distance and exposure do not seem to play an additional more refined role among this sample of nearby in-laws, who already live within the short distance of the median of 14 miles (as seen when we cut the sample further by the twenty-fifth percentile in column 5 of Table 6).

The second distant circle that allows us to explore the role of exposure intensity are coworkers. While considerable 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 2). More interestingly, we find no effects on coworkers within smaller

[29] 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).

[30] For this analysis we constrain the sample to include only parents- and children-in-law who do not share the same doctors, for whom the overall effect is reported in column 3 of Table 6, to verify variation by distance would not be attributable to that.

[31] The time pattern of their responses in statin consumption using the dynamic specification of equation (1) is provided in online Appendix Table 10. Note that another potential explanation for variation by distance could be environmental health risks (such as air pollution). In such a case, one may expect similar variation by distance in the responses of the adult children married to these sons- and daughters-in-law, for which we find no evidence in the data.

Table 6—Effects of Cardiovascular Shocks on Statin Consumption by Sons- and Daughters-in-Law

| | | | Children-in-law by distance | | |
|---|---|---|---|---|---|
| | Children-in-law (1) | Adult children married to sample of column 1 (2) | Subsample mean (3) | By median distance (4) | By 25th percentile of distance (5) |
| Treat × post | 0.00114 | 0.00485 | 0.00159 | | |
| | (0.00058) | (0.00056) | (0.00062) | | |
| Further from parents-in-law | | | | 0.00096 | 0.00243 |
| | | | | (0.00081) | (0.00153) |
| Closer to parents-in-law | | | | 0.00245 | 0.00247 |
| | | | | (0.00097) | (0.00124) |
| Counterfactual | 0.02337 | 0.01768 | | | |
| Percent change | 4.89 | 27.43 | | | |
| Observations | 1,129,928 | 1,124,320 | 958,768 | 958,768 | 402,544 |
| Number of clusters | 73,268 | 73,135 | 67,261 | 67,261 | 33,752 |

*Notes:* This table reports difference-in-differences estimates for statin consumption responses to family cardiovascular shocks by sons- and daughters-in-law. Column 1 estimates equation (2) for children-in-law. For comparison, column 2 estimates equation (2) for the adult children married to them. The slight difference in samples across the two columns is driven by a small set of households for whom we have information on education for only one spouse. Counterfactual levels in the periods following the shock are calculated using these estimations. The remainder of the table studies the spillover effect on sons- and daughters-in-law by the distance to their parents-in-law. Column 3 first replicates the overall effect on in-laws from column 1 for the subsample of families in which parents- and children-in-law do not share the same doctors, to verify variation by distance would not be attributable to that. In column 4 we then 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 effect for each subsample split calculated using a regression of specification (3). Column 5 cuts the sample further and includes only those whose distance from their in-laws is shorter than the sample median, and then splits the remaining sample by the 25th percentile of the unconditional distance distribution. We include as controls age fixed effects, calendar year fixed effects, gender, and education, and we report in parentheses robust standard errors clustered at the household level.

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 2).[32] The patterns of spillover impacts on health care 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.[33]

## C. *Alternative Explanations*

The analysis so far suggests roles of family health events in inducing learning about own health and in making health salient. In this subsection, we investigate

[32] 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 either.

[33] An additional piece of evidence in line with these findings is the lack of spillovers among a sample of former spouses. See column 6 of online Appendix Table 2, where we define former couples as individuals who were linked through marriage or cohabitation in period $t = -5$ but are no longer linked in period $t = -1$.

and discuss the potential role of leading alternative explanations for the estimated responses of spouses, and we also show that several such explanations do not appear to be operative. 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 online Appendix Table 3 for these results. In addition, the evidence suggests that the spillover is not likely to be driven by responses of a family physician who may provide primary care to several members of the household and aggregate information across them. We analyze households in which husbands and wives do not share the same doctor (defined in several ways), and we find similar-magnitude responses among them (see online Appendix Table 4).[34] Of course, we note that the respective physicians of the spouses themselves (or of the other network members) can naturally act as a mechanism, e.g., if they learn from their patient about the relative's health shock and consider or recommend some measures of preventive care.

## V. Discussion and Conclusion

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 can be far reaching, as they also spill over to the consumption of health care by 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 health care which constitutes a large share of household spending. 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 health events 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'

[34] In this table, we also reach similar conclusions from an analogous exercise for adult children.

attention is particularly drawn to the local nature of the health event 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 interpersonal interactions, suggesting we should analyze health behaviors, the demand for health investments, and the consumption of health care 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. To put the effect magnitudes in perspective, it may be useful to compare them to benchmarks of under-utilization of preventive care. Within our main application of cardiovascular shocks, a recent paper relevant to the population that we study provides us with a useful benchmark for the recommended population share that should consume statins for prevention. Mortensen and Nordestgaard (2018) assess this share for the Danish population based on the Copenhagen General Population Study (a representative medical cohort study of the Greater Copenhagen population) for adults of ages 40–75. They estimate according to the European guidelines, which are the relevant guidelines for our Danish application, a recommended share of 15 percent. To compare the baseline utilization of statins among our studied population, we adjust it to reflect similar households based on age, gender, and residence. We find that the baseline utilization share of spouses is well below the recommended benchmark and is approximately 6.9 percent (SE 0.27 percent). This is also the case for spouses in our sample with high cardiovascular risk, for whom the corresponding share is 8 percent (SE 0.32 percent). As these simple statistics point to baseline under-utilization of preventive care among the population that we study, they are suggestive that family shocks may induce beneficial health behaviors that should have been practiced in the absence of the adverse event. Gauging the magnitudes of our estimated effects, we find that the spillovers from the family shock alone close 16 percent $(= 1.3/(15 - 6.9))$ of the under-utilization gap in statin consumption among spouses by the end of the analysis period. While Mortensen and Nordestgaard's (2018) study is not applicable to our younger sample of adult children, *if* one is willing to further assume that the proportional baseline under-utilization is similar for this sample, then children's required response for reaching recommended levels would be 1.28 percentage points. In that case, the spillovers would close 42 percent $(= 0.54/1.28)$ of the potential under-utilization gap among them.

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 health care 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.[35] However, our findings also point more generally to potential pitfalls in using salience as a policy tool (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 possible unintended crowd out of non-salient dimensions, as these would be made less relevant by design.

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[35] 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. 2014), which recommend family screenings of high risk individuals to identify additional family members who would benefit from assessment and treatment.

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