An Ounce of Prevention or a Pound of Cure? The Value of Health Risk Information *

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Abstract

Individuals infer their health risk from experiences of people around them, particularly family. I assess how household health events affect this learning. When someone is diagnosed with a chronic condition, their family members increase healthcare spending by 10%. Informational spillovers lead to higher utilization of both high- and low-return care; responses align with individuals reassessing health risks given new information. To evaluate welfare implications, I estimate a structural decision-making model where individuals learn about risk from family. Results reveal consumers over-respond to diagnoses, over-weighting their risks *ex-post* and crowding out potential welfare gains from information for almost half of individuals.

Keywords: Health spillovers, consumer learning, behavioral health economics, discrete choice models, chronic illness

JEL codes: I12, I13, D83, D91, D12

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1 Introduction

Social networks provide important information for consumers making healthcare choices. Through connections with family, friends, and neighbors, individuals form expectations of their health risks, learn about the value of specific medical practices, and identify how or from whom to receive care. Family relationships provide particularly influential sources of health information due to their proximity and the high relevance of their health experiences, as both shared genetic profiles and lifestyle choices influence expected healthcare consumption. Understanding how individual health experiences shape family health behaviors is essential for policies aiming to improve public health, such as those attempting to address high levels of healthcare spending or the takeup of high-value health services.

One especially salient dimension of information that healthcare shocks communicate is knowledge about individual health risks, particularly if the shock highlights dimensions of risk that are correlated across family members. Affected individuals may alter their expectations about future health care needs, and even choose to increase take-up of high-value health services after witnessing a family member's health experience. For example, individuals may choose to become vaccinated against COVID–19 after witnessing the infection of a family member (Chen, 2021; Giardinelli, 2021; Salcedo, 2021).

There is evidence that family members react to the health events of their loved ones (Fadlon and Nielsen, 2019; Hodor, 2021), but it remains unclear what drives these reactions. Health events may lead individuals to reassess their health risks, but may also change the expected price of household medical care, alter household preferences for health consumption, or provide knowledge about the availability of health services. Understanding the role that social connections play in altering individual health choices—including the use of both high-return care and the propagation of low-value services—relies on separating these competing effects. In particular, assessing the welfare effects of transmitting new health information requires both understanding whether individuals respond to information itself and the extent to which they update their beliefs correctly.

In this paper, I examine how a health shock affecting a household—in particular, a new diagnosis of a chronic condition—changes individual assessments of health risks, and how these changes disrupt healthcare consumption. Using claims data for U.S. households with employer-sponsored insurance (ESI) between 2006 and 2018, I show that health events generate strong informational spillovers among non-diagnosed household members. Affected individuals significantly and persistently increase both overall utilization and investments in disease-specific preventive care, novel evidence of responsiveness to new risk information.

Further, I show that individual risk updating appears to drive observed spillover utilization, rather than alternative mechanisms. I observe that spillover responses are consistent even when the marginal price of future care is unaffected by a diagnosis, suggesting that *ex-post* moral hazard concerns do not drive the results.[1] Chronic diagnoses also induce larger changes in spending than similarly intensive acute health events, suggesting that general salience effects—absent risk information—are insufficient to describe the observed results. Finally, I separate responses to risk information from responses to a broader set of institutional knowledge, exploiting adherence to existing prescriptions to show that learning about health *systems*, rather than health *risk*, does not drive my results.

In general, one would expect new risk information to improve individual decision-making and welfare. Surprisingly, however, the welfare effects of chronic diagnoses are ambiguous using only reduced-form evidence. I observe that affected household members increase take-up of "low-value" health care, including services that do not benefit the marginal patient or have low levels of benefits relative to costs (Colla et al., 2015). These responses are most likely to include increased utilization of low-value services that appear, from a patient's perspective, closely related to preventive care, including extraneous pre-operative screenings or imaging services. Importantly, this suggests individuals may have trouble interpreting risk information signals from a chronic diagnosis or may not appropriately choose high-quality services conditional on their specific medical histories.

[1] As is common in the health economics literature, I use the phrase "moral hazard" to denote induced-demand effects arising from changes in the price an individual faces for care Einav et al. (2013).

These findings motivate a structural approach to model how risk beliefs evolve following a household health shock and how such transitions affect individual welfare. I write and estimate a dynamic model where forward-looking individuals learn about health risks through preventive screenings and household diagnoses. The model combines dynamic investments in health production (Grossman, 1972) with consumption decisions trading off risk protection and consumption (Cardon and Hendel, 2001); this provides identifying variation pinning down individual learning from new diagnoses separately from dynamic effects, moral hazard, or salience. Importantly, the model allows me to estimate the welfare effects of observed responses and compare them to responses with more targeted risk signals.

Counter to expected thought, information transmitted from household health shocks is not welfare-improving for a large subset of affected individuals. Nearly half of those presented with new risk information would be willing to pay to avoid the resulting change in their beliefs, with welfare losses averaging 90% of baseline expected utility. The central insight of the model is that there is a tension between the seriousness of a major health event and the appropriate level to which individuals should update their beliefs: new diagnoses in a household spur overly large changes in an individual's assessment of their health risks, resulting in average posterior beliefs that are well above the average in-sample risk of diagnosis. This result is robust to multiple iterations of the model and does not appear to be sensitive to any single modeling assumption. Counterfactual simulations suggest that bounding these changes in risk beliefs substantially increases consumer welfare: over 90% of individuals in my sample would find health information welfare-improving were their responses mitigated.

My analysis contributes to an important discussion on the spillover effects of health information within social networks, particularly the family. Family relationships provide important information for economic decisions, including labor supply and education (Browning et al., 2014; Altmejd et al., 2021); however, their role in forming health behaviors is not as well understood. Previous work has suggested that an individual's social network informs their decision-making following acute health events (Bouckaert et al., 2020; Hodor, 2021;

Song, 2021), health trials (Archibong and Annan, 2021), and infectious disease outbreaks (Agüero and Beleche, 2017).[2] I contribute to this discussion in three ways. First, I present novel evidence on the role of learning about risk from family, highlighting chronic diagnoses as a new form of information transmission to which individuals respond more than even other acute health events. Second, I show that this learning is the primary mechanism causing observed spending changes. Finally, I integrate this evidence to show that risk information on its own does not improve the quality of consumed care and may ultimately lead to individual welfare losses, perhaps due to its lack of interpretability.

Beyond information transmission within families, I also contribute to the literature on non-Bayesian learning in models of health behavior (Barseghyan et al., 2018; Bundorf et al., 2021a). My model combines findings from two distinct threads of the learning literature. First, I emphasize the role of disproportionate weight individuals place on high-cost, low-probability events (Kahneman and Tversky, 1973; Holt and Smith, 2009; Goldstein et al., 2023). Such disproportionate responses have been found to rationalize individual choices that would otherwise require unreasonably high levels of risk aversion to justify (Ortoleva, 2012; Paserman, 2008; Spinnewijn, 2015). I combine these results with quickly-evolving literature studying the role of peer signals in learning. My work highlights how individuals over-emphasize peer signals relative to their own, similar to work studying how individuals value signals from sufficiently related distributions (Dasaratha et al., 2022).

I incorporate these disparate findings into a new structural model of health risk belief formation. This model is similar to other models underscoring the relationship between health expectations and behaviors (Darden, 2017), but is novel in that it allows for individuals to inappropriately overweight noisy signals of risk. My model incorporates a fully flexible specification for misinterpreting information (Hauser and Bohren, 2021), and provides a

[2] A rich literature has highlighted how individuals respond to information about their own health risks, including their own diagnosis. For an in-depth review of this literature, see Alalouf et al. (2019). Some previous work has demonstrated that certain diagnoses can have dramatic impacts (Almond et al., 2010); however, examinations of other diagnoses revealed a lack of noticeable responses (Dupas, 2011; Kim et al., 2019).

micro-foundation for how individuals form beliefs in a setting of largely small-probability events. The model results highlight that even fully forward-looking consumers with rational expectations may over-respond to health shocks *ex-ante* in a quasi-Bayesian framework, resulting in welfare penalties from new information. The model provides additional insight into the development of subjective health beliefs, including why consumers may be better at predicting relative, rather than absolute, risk (Bundorf et al., 2021b) and why biases in assessing risks may arise (Arni et al., 2021).

Finally, my work is relevant to the well-established literature exploring sub-optimal health decisions made by many consumers (Abaluck and Gruber, 2016a; Baicker et al., 2015; Handel and Kolstad, 2015).[3] This includes discussions about the extent to which improving health information generally may improve decision-making (Abaluck and Gruber, 2016b; Cutler and Zeckhauser, 2004; Gruber et al., 2020). My analysis reveals that overcoming information frictions is not simply a matter of increased access to health information.[4] Rather, individual responses to some information may not improve the quality match of patients and services, but simply shift consumers from one type of poor decision-making to another while increasing total health spending.

I present my empirical setting and data in Section 2. Following a discussion of major health events, I provide evidence of their spillover effects and the potential mechanisms driving them in Section 3. Then, to evaluate the welfare effects associated with these responses, I present the details of my model in Section 4 and its results in Section 6. Finally, I discuss the relevance of my findings and directions for future work in Section 7.

2 Empirical Setting & Data

Data on household health utilization and major medical events come from the IBM/Truven Marketscan *Commercial Claims and Encounters* Data from 2006 to 2018. These data contain

[3] See Abaluck and Compiani (2020) for a more thorough discussion of these results.

[4] Finkelstein et al. (2022) find a similar result when considering drivers of adherence to medication guidelines for high-return pharmaceutical treatments.

detailed inpatient, outpatient, and pharmaceutical claims for households enrolled in an ESI plan provided through participating insurance carriers to several medium and large U.S. firms. which contracted with participating payers. Throughout, spending data has been normalized to 2020 USD using the Consumer Price Index for All Urban Consumers series.

I limit the sample to only households with two or more members observed for two or more years and insured with one of eight large firms. I required that each household have full eligibility and continuous enrollment across their window of observation. My final sample consists of 353,403 households and 5,439,482 individual-year observations.[5]

Table 1 presents summary statistics for the full sample as well as for households in which an individual is affected by a chronic condition. In general, households are comprised of one to two adults and one to two children, with relatively generous insurance coverage. The average (median) household pays out-of-pocket for roughly 18% (16%) of their annual health consumption, and 21% of individuals in the sample do not face any cost-sharing during a year. In addition, roughly a quarter of households are enrolled in plans with no deductibles.[6]

2.1 Major Health Events

Major health events, which communicate information about health risk to households, are identified by diagnostic codes in the data, following the Department of Health and Human Services' Hierarchical Condition Categories (HCC) diagnostic codes. HCCs are typically used in risk adjustment models and identify a basic set of chronic illnesses that alter overall

[5]The average household is continuously observed for 7 years in the data. Note that although the results presented in the main text utilize an unbalanced panel—potentially contributing to increased levels of uncertainty at the tails of event study specifications—the results are robust to specifications requiring a fully balanced panel across 6 years.

[6]While insurance contracts are defined by a complicated set of cost-sharing measures—including copayment and coinsurance rates that vary widely across provider specializations, networks, and procedures—the structural model described in Section 4 uses only a family deductible, a simplified non-specialist coinsurance rate, and a family OOP maximum, consistent with prior work (Marone and Sabety, 2022). These measures are constructed using the empirical distributions of payments in the claims data, and described in detail in Appendix Section A.1 (Zhang et al., 2018). I find that these simplified measures capture a wide degree of variation in my data and harmonize well with measures from earlier work.

Table 1. Household Summary Statistics

	Full Sample	Households Affected by Chronic Events
Panel A: Household Demographics		
Family size	2.84 (0.001)	3.11 (0.004)
Employee age	45.01 (0.007)	43.61 (0.039)
Enrollee age	30.87 (0.008)	29.37 (0.041)
% female employees	41.57 (0.037)	41.04 (0.190)
% female enrollees	50.17 (0.021)	50.11 (0.109)
Risk Score	0.95 (0.001)	1.51 (0.008)
Panel B: Household Medical Utilization		
Total medical spending	\$2,504 [\$680] (4.51)	\$4,546 [\$1,130] (73.13)
OOP medical spending	\$443 [\$110] (0.53)	\$614 [\$175] (4.39)
% enrollees w/ 0 spending	15.39 (0.015)	10.35 (0.067)
% enrollees w/ 0 OOP	21.04 (0.017)	14.68 (0.077)
Household deductible	\$415 (0.619)	\$419 (3.094)
% w/ 0 deductible	28.04 (0.032)	32.47 (0.180)
Panel C: Individual Major Medical Events		
Total cost, Diagnosis	—	\$4,164 [\$1,319] (156.81)
OOP, Diagnosis	—	\$532 [\$212] (27.36)
OOP, Recurring	—	\$489 [\$190] (24.78)
$N_{\text{households}}$	353,403	62,528
$N_{\text{individuals}}$	1,087,353	194,844

Notes: Values based on Marketscan claims data, 2006–2018. Enrollees are employees plus their covered dependents. Spending values are reported in 2020 USD. Standard errors are reported in parentheses and sample medians (when reported) are in brackets. Column 2 limits the sample to only household-years in which a chronic diagnosis occurred (see Appendix A for diagnostic codes).

health utilization and spending. I limit my classification of health events to non-pregnancy HCCs that occur with high frequency.[7]

Table 1 shows how households affected by these chronic conditions differ from the full population. The second column of the table limits the sample to only household-years in which a chronic diagnosis occurred. Affected households are riskier, on average—note that this is mechanical, as HCCs are directly used in the calculation of a risk score. In the year of

[7]See Appendix Section A.2 for details. To ensure that I identify new diagnoses, I require that relevant diagnosis codes appear during or after an individual's second observed year.

diagnosis, the average (median) household spends about 82% (66%) more on health services than the corresponding household in the full sample. In contrast, affected households look very similar to the full sample in terms of insurance enrollment and plan generosity.

An important feature of my analysis is the separate treatment of health costs for major medical events, including the costs associated with maintaining the health of someone with a chronic condition. To measure these costs, I collaborated with Rebecca Hughes, MD, to identify a set of disease-specific procedures and prescriptions associated with each health condition in my sample.[8] I then identify household spending on these health events based on the claims for these procedures and prescriptions, both in the year of diagnosis and following years. As reported in Table 1, the average (median) household in my sample spends $532 ($212) OOP on the associated diagnosis, and then $489 ($190) each year that follows on recurring costs needed to care for chronic conditions.

2.2 Additional Variable Definitions

The rich variation of the data allows me to evaluate the impact of new chronic diagnoses on a wealth of utilization and quality measures. In particular, I define four additional outcome variables which will be useful in identifying the mechanisms by which new health information changes household behavior: preventive health services, acute health events, adherence to prescription medication, and the use of low-value health services. Appendix A contains a full set of all diagnostic information, procedure codes, and therapeutic classes used in the construction of each of these variables.[9]

First, I define a set of health services typically considered to be preventive in nature, consistent with previous work and federal guidelines (Hoagland and Shafer, 2021; USPTF, 2022). Preventive screenings and wellness visits constitute an important point of entry for the identification of other health concerns (Jiang et al., 2018) and are generally considered to be an important form of high-value care (Tong et al., 2021). For each enrollee, I identified

[8] Appendix Section A.3 lists the relevant codes used for each diagnosis.
[9] See sections A.3 through A.6 for details.

individual preventive services based on commonly used code combinations recommended by the United States Preventive Task Force (USPTF).

Second, I define a set of *acute* health events, which capture health events of a similar level of seriousness to new chronic diagnoses, but which are transient in nature. These events are used to compare how households respond to new risk information to responses to a health event wtihout information, but which may change future marginal utilities of health consumption (see Section 3.3.1). I identify acute health events as new HCCs within households for conditions which typically do not persist past a year, including hospitalizations for severe viral infections or other non-chronic conditions. Appendix Section A.4 compares acute and chronic events by pre-event spending, event cost, and hospitalization incidence; overall, these events are comparable to new chronic diagnoses, as discussed in Section 3.3.1.

Third, I define adherence to prescription medication. This measure is used to separately identify how changes in new risk information may alter household behaviors independent of other forms of information about the health system as an institution (see Section 3.3.2). I measure adherence to cardiovascular preventive drugs as the proportion of days covered in a year, in keeping with prior literature (Choudhry et al., 2009).[10]

Finally, I define categories of medical utilization which are frequently labeled as "low-value" by medical professionals and health officials (Chua et al., 2016; Colla et al., 2015).[11] Low-value services include both those whose cost typically outweighs the benefits to an average patient (e.g., some surgeries, such as arthroscopy) and services which are chronically over utilized in ways that dramatically lower their return (e.g., some imaging services, such as MRI for migraines). I define instances of low-value consumption based on an individual's diagnosis and procedure codes as well as their diagnostic history, based on previous work (Colla et al., 2015). I subdivide these services into 5 categories: pediatric services, including

[10]This measure is standard in the literature on adherence, and corresponds to the fraction of the year after a patient's first prescription fill for which the patient has a supply of the medication. Appendix Section A.5 contains a detailed list of the therapeutic classes used in my sample.

[11]These health services are based on recommendations made with the Choosing Wisely initiative, directed by the American Board of Internal Medicine Foundation and other physician specialty organizations (Bhatia et al., 2015; Wolfson et al., 2014).

imaging services and the early use of medications such as antibiotics; adult prescription drugs, such as the use of opiates to treat migraines; unnecessary imaging services for adults; extraneous screening services for adults, including cardiac testing before low-risk surgeries; and adult surgical procedures.

3 Spillover Effects of Household Health Events

To estimate the causal impact of health shocks on health choices, I use a "stacked" regression design, which avoids potential bias from naive staggered treatment designs in the presence of heterogeneous treatment effects within units over time (Goodman-Bacon et al., 2019; de Chaisemartin and D'Haultfoeuille, 2019; Cengiz et al., 2019).[12] I create event-specific cohorts $r \in \{1, ..., N^1\}$ based on the year of diagnosis for each affected household, including both appropriately not-yet-treated households and those who are never diagnosed in the control group for each cohort. I then stack cohorts and estimate a single regression with cohort-specific time and household fixed effects:

$$Y_{tf,r} = \alpha_{f,r} + \tau_{t,r} + \sum_{k=-T}^{T} \gamma_k \mathbb{1}\{t - E_{ft,r} = k\} + \epsilon_{ft,r}. \tag{1}$$

In Equation 1, f denotes a household, t a year, and r a cohort. To assess spillover responses, $Y_{ft,r}$ aggregates health utilization across a household *excluding* those who experience the major health event. I measure these outcomes both in counts (e.g., number of visits) and spending (both total and OOP), and adjust for the skewed nature of these distributions by using Poisson regression for count outcomes and the inverse hyperbolic sine transformation for spending outcomes.[13] Throughout, reported coefficients can be interpreted as approxi-

[12] My results are robust across several alternative specifications, including the standard two-way fixed-effects (TWFE) model and alternative "doubly robust" estimators (Callaway and Sant'Anna, 2018; Sant'Anna and Zhao, 2020). I do not find any evidence that my regression results suffer from concerns of negative weighting (Goodman-Bacon et al., 2019). These and other robustness checks are reported in Appendix Sections B.1 and B.2.

[13] I use the inverse hyperbolic sine transformation for spending data to accommodate the approximately 15% of individual-years in my data with 0 spending (Harris and Stöcker, 1998; Bellemare and Wichman,

mate percentage changes in the outcome variable, relative to the year before the shock, $t-1$. Standard errors are clustered at the household level.

A stacked approach allows me to identify the potentially time-varying effects of health shocks without contamination from an inappropriately selected control group. Previous work has restricted comparisons to only households who have yet to experience a similar diagnosis ("not-yet-treated" households), to improve the *ex-ante* match quality between groups on unobservable characteristics. In addition to not-yet-treated households, I also include never-treated households in the estimation. The central tradeoff in doing so is that including these households allows for separate identification of dynamic treatment effects from time fixed-effects (Sun and Abraham, 2020), but may come at the cost of introducing violations in the parallel trends assumption.[14] I discuss this tradeoff more in Appendix Section B.3, and include robustness checks showing balance across the groups and using only not-yet-treated households in estimation.

Figure 1 presents the time-varying causal effect of a health shock on household OOP spending for all non-diagnosed individuals. The first panel illustrates that non-diagnosed household members increase their annual OOP spending by about 10% relative to the year before the event. For the median (average) household, this corresponds to an increase of about \$50 (\$115) annually. This effect begins in the year of the health event and persists following the diagnosis.[15]

While health events may generate spillovers in household spending for many reasons, households responding to the information contained in a diagnosis may be more likely to seek out preventive screenings. The second panel of Figure 1 estimates the effect of chronic diagnoses on the total utilization of preventive services, as defined in Section 2.2. Here, too,

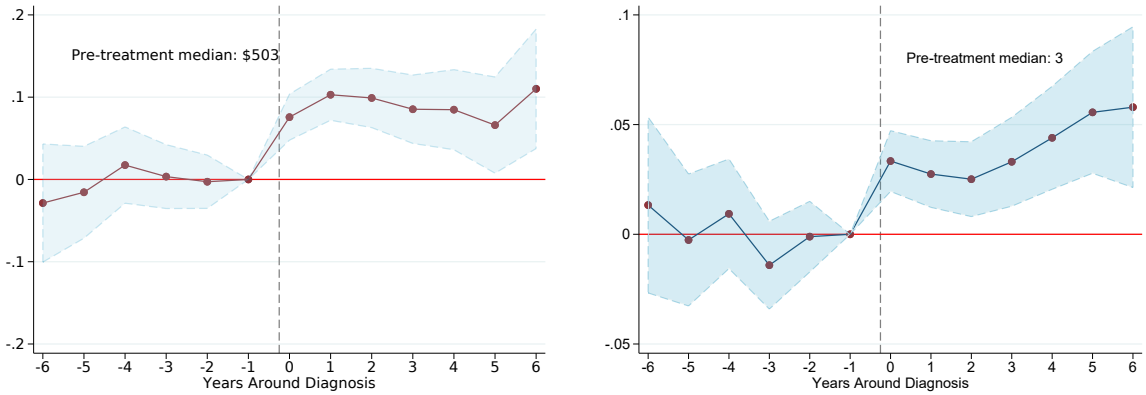
2020). I show in Appendix Section B.1 that my results are not substantively altered when using alternative transformations, including the more typical $\log(y+1)$ or Poisson regression.

[14]Namely, that in the absence of major health events, the treated and control groups would continue to have similar spending and utilization trajectories.

[15]These results are robust to the measurement of the outcome variable, including total billed spending or number of unique health encounters; see Appendix Section B.4. Some specifications do not suggest that the results persist for a full five years after diagnosis, but all consistently find short-term effects lasting at least 3-4 years following a diagnosis.

Figure 1. Effect of Chronic Diagnoses on Other Household Members' Healthcare Utilization

(a) $\sinh^{-1}$(Total OOP Spending) (b) # of Preventive Visits



Notes: Figures show regression coefficients from "stacked" TWFE regressions, with 95% confidence intervals. Regressions estimate the effect of a new chronic diagnosis on medical utilization of other (non-diagnosed) household members. In panel (a), the dependent variable is the inverse hyperbolic sine of total OOP spending; panel (b) estimates the effect on the number of household preventive services per year using Poisson regression. Coefficients are presented relative to the year prior to diagnosis. Standard errors are clustered at the household level.

I find that new diagnoses in a household are associated with strong responses. Affected, non-diagnosed household members increase their overall use of wellness visits by about 5% relative to a median of 3 visits annually.[16]

A principal concern threatening the identification of these effects is that anticipation of a diagnosis—through, for example, deteriorating health—may introduce unobserved pre-trends into the empirical analysis, even for adjacent household members. For comparison, I present results in Appendix Section B.4 illustrating how diagnoses affect the focal individuals; this both benchmarks the spillover results and allows me to observe any anticipation directly. I find that diagnosed individuals more than double their annual OOP spending in the year of diagnosis but not thereafter; additionally, I do not observe spending increases pre-diagnosis.[17]

[16] Appendix Section B.4 shows that these results are robust to alternative outcome measures, including spending. Here, I show the main outcome measured in the number of visits rather than spending to account for the fact that the Affordable Care Act (ACA)'s cost-sharing exclusion took effect in 2010 (or 2012 for certain women's health services), disrupting the costs for preventive services for those with ESI (Hong et al., 2017).

[17] This makes sense in the context of the events in my sample, which are mainly unexpected diagnoses affecting children (e.g., sudden-onset diabetes). Such events are not typically associated with a pre-diagnosis "anticipation" stage, especially not one lasting longer than a year.

3.1 Changes as Responses to New Health Risk Information

These results suggest a meaningful, persistent change in how an individual diagnosis affects the utilization of an entire household. Next, I turn to exploring the mechanisms behind such responses. I first show that observed responses are indicative of affected individuals reassessing their health risks in light of new health information from a major diagnosis; I consider alternative explanations in Section 3.3.

I show that households respond to the risk information contained in a diagnosis by estimating the effect of a new diagnosis on the use of *disease-specific* preventive services. The intuition I rely on is that household exposure to risk information is more targeted than other forms of information, leading individuals to select into specific screenings based on a focal diagnosis. For example, households with a newly diagnosed diabetic may use diabetes screenings more post-diagnosis than households affected by a non-diabetes diagnosis.[18]

To assess the causal effect of diagnoses on the utilization of disease-specific preventive care, I use a triple-differences approach. This approach separates the disease-specific effect of risk information from any general effects resulting from a new chronic diagnosis (e.g., salience effects). I estimate the effect of a new chronic diagnosis on a household f's decision to screen for a specific diagnosis d during time t, as summarized in Equation 2:

$$\begin{aligned} Pr(\text{Screening})_{fdt} =& \beta_{\text{DD}}(\text{post}_t \times \text{chronic}_f) + \beta_{\text{DDD}}(\text{post}_t \times \text{chronic}_f \times \mathbb{1}\{\text{chronic}_f = d\}) \\ &+ \alpha_f + \tau_t + \varepsilon_{fdt}, \end{aligned} \tag{2}$$

where $chronic_f$ is a dummy variable indicating whether *any* chronic diagnosis occurred within the household and $post_t$ indicates periods following a diagnosis. The triple interaction variable includes an additional constraint that the chronic diagnosis $chronic_f$ matches the specific diagnosis d (e.g., a diabetes diagnosis when the outcome variable is a diabetes

[18] This is exactly what is shown in the raw data in Appendix Figure B.6.

screening). Hence, β_{DD} identifies the effect of any chronic diagnosis on screening, while β_{DDD} identifies the effect of the specific diagnosis of interest relative to other diagnoses.[19]

The triple difference approach is advantageous because it allows me to compare the causal effect of diagnoses on the use of preventive care across multiple control groups. When the outcome variable is a screening for a specific service (e.g., diabetes), Equation 2 estimates the effect of a "matching" diagnosis relative to other diagnoses where the screening would not be informative. The identifying assumption for the triple differences approach is the same as for the earlier regressions: that spending differences between diagnosed and undiagnosed households would have evolved similarly over time in the absence of treatment.[20]

I estimate several versions of this regression for various diagnosis-screening pairs. I select diagnoses and screenings which are commonly utilized and for which there are clear diagnostic codes available. I examine the impact of new diabetes and cancer diagnoses on their respective screenings, as well as the effect of diabetes diagnoses on cholesterol screenings. I also assess the impact of any new chronic diagnosis in a household on the rate of new hypertension diagnoses, relative to all major health events.[21]

Table 2 presents the estimation results from these regressions in two panels. First, I highlight that new chronic diagnoses alter specific preventive behaviors in cases where they transmit important information about health risk. The occurrence of any chronic diagnoses in a household is associated with a 19.4% increase in the rate of hypertension diagnoses among other affected household members. Furthermore, specific diagnoses such as cancer

[19]The sum of the coefficients $\beta_{DD} + \beta_{DDD}$ identifies the diagnosis-specific effect of receiving a diagnosis, relative to all non-diagnosed households in my sample. Notice that, in Equation 2, all requisite interaction terms for the triple differences are either subsumed in the fixed-effects or colinear with the included variables given the unique structure of my treatment variables.

[20]When adding the triple interaction, the identifying assumption is modified only to include the assumption that spending differences between households diagnosed with one condition and households diagnosed with another would have evolved similarly in the absence of treatment, a statement which is subsumed in the initial identifying assumption. Appendix Section B.5 includes standard difference-in-differences regression results that corroborate the findings reported here.

[21]Given that there is no procedure code for hypertension screenings, this approach proxies the effect of the risk information associated with chronic diagnoses on new general wellness screenings, relative to the other forms of health information accompanying acute events. Coding practices reduce my ability to test this finding for each individual diagnosis in my sample; for example, there are no diagnostic or procedure codes used exclusively for asthma screenings.

and diabetes increase the likelihood that a non-diagnosed household member will seek out screening by 13.2% and 21.1%, respectively. Finally, diabetes diagnoses are associated with an increase in cholesterol screenings of 7.2%.[22]

The second panel of Table 2 reports results for two "placebo" regressions for cases where health events communicate no useful *risk* information, and hence are expected to change disease-specific screenings little. These include the effect of new diabetes diagnoses on obesity diagnoses (a diagnosis which, while an important risk factor for some types of diabetes, is externally verifiable prior to a household diagnosis), and the effect of a new household mental health condition on screenings for depression. I find no evidence that these events affect screenings, an important finding given that they may provide less useful risk information to households. For more discussion, see Appendix Section B.6.[23]

3.2 Quality of Induced Spending Changes

Given the high degree of responsiveness to chronic diagnoses, a natural question is whether new information improves the overall quality of care received. While new diagnoses could feasibly cause individuals to substitute consumption towards high-value preventive services, affected individuals may choose to increase consumption overall, with less regard for the risk-mitigating benefit of a service. I examine how diagnoses spillover into household consumption of low-value care, as defined in Section 2.2.[24]

[22]Similar to previous work, I also find evidence that new diagnoses reduce the rate of other, unrelated screenings (Fadlon and Nielsen, 2019); for example, a non-diabetes chronic diagnosis is associated with a 7.4% *decline* in the rate of diabetes screenings among non-diagnosed household members. These effects, however, are typically smaller than the estimated increases in disease-specific screenings, suggesting that this crowding out is not necessarily one-to-one.

[23]Additional results include leveraging within-family variation in relationships and corresponding risk to show that households are selective in which members receive screenings following a diagnosis. For example, when households are affected by a chronic illness with a strong genetic component—such as type 1 diabetes—children and siblings of the affected individual are more likely to be screened than other household members. On the other hand, diagnoses such as type 2 diabetes—which has a stronger lifestyle component than a genetic one—are associated with more frequent screenings for spouses.

[24]In addition to the utilization of low-value care, I explore other ways health events alter the quality of consumers' health care decisions, including their plan choices (Appendix Section B.8). In general, I do not find that major health events prompt households to switch their health insurance plans.

Table 2. Effect of Chronic Diagnoses on Take-Up of Disease-Specific Preventive Care

Own Screening (Dependent Variable)	Household Diagnosis	Pre-Diagnosis Average	Effect of Any Diagnosis (β_{DD})	Effect of Specified Diagnosis (β_{DDD})
Panel A: Main Effects				
Hypertension[1]	Any Chronic[2]	2.01	-0.27**	0.39***
		(0.007)	(0.102)	(0.110)
Cancer	Cancer	20.72	-0.01	2.74***
		(0.021)	(0.113)	(0.509)
Diabetes	Diabetes	6.21	-0.46***	1.31***
		(0.012)	(0.086)	(0.279)
Cholesterol	Diabetes	17.01	-0.22	1.23***
		(0.019)	(0.126)	(0.389)
Panel B: Placebo Regressions				
Obesity[1]	Diabetes	1.04	0.02	0.10
		(0.005)	(0.035)	(0.110)
Depression	Depression	0.36	-0.01	-0.08
		(0.003)	(0.037)	(0.077)

Notes: Table presents six triple-difference regressions for how diagnoses affect household investments in disease-specific preventive care (Equation 2). Outcome variables are binary indicators for the screening in the first column; treatment variables are a binary indicator for the diagnosis in the second column. Difference-in-differences coefficients (β_{DD}) indicate the effect of *any* chronic diagnosis on screenings, while triple differences coefficients (β_{DDD}) indicate the (additive) effect of *specific* diagnoses. Standard errors clustered at the household level shown in parentheses. [1]Due to unavailability of procedure codes, these outcomes are measured using diagnostic codes. [2]Here, the reference group is all acute major health events. $^{*}p < 0.05$,$^{**}p < 0.01$,$^{***}p < 0.001$

I find that new chronic diagnoses are associated with an increase in total low-value spending of about 5% (Appendix Section B.7). However, these results mask significant heterogeneity across services; separating them yields useful intuition as to what information households react to. Households may seek out different types of low-value care if they are responding to new risk information—e.g., by demanding low-value screenings such as imaging services or preoperative screenings—or responding to marginal price changes following a diagnosis—e.g., by opting for more elective surgeries.

Table 3 presents results estimating the effect of a new chronic diagnosis in each of five categories using a standard difference-in-differences framework.[25] New chronic diagnoses

[25]Stacked regressions with dynamic effects are included in Appendix Section B.7.

Table 3. Estimated Effects of Chronic Illness on Low-Value Care Utilization

Population	Pediatric	Adult Services			
Service Category	All Services	Prescriptions	Imaging	Screening	Surgery
$\text{Post}_t \times \text{Diagnosis}_f$	0.051*	-0.004	0.029***	0.103***	-0.096***
	(0.017)	(0.000)	(0.013)	(0.014)	(0.012)
R^2	0.349	0.309	0.293	0.326	0.379

Notes: Table shows estimated difference-in-difference regression coefficients for the effect of a new chronic diagnosis (N=1,538,161). Outcome variables are the inverse hyperbolic sine of billed spending in each category. See Appendix A for service definitions. Spending is measured in 2020 USD. Standard errors clustered at the household level are reported in parentheses.
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

increase household utilization of low-value screenings, imaging services, and pediatric care, with effect sizes ranging from ten percent for screenings to three percent for imaging. I find no effect on the misuse of prescription drugs among adults.[26] Taken with the previous results, these findings suggest that households affected by new chronic conditions increase their utilization of a broad set of preventive and "psuedo-preventive" services, without regard to the average return on those services.[27]

3.3 Alternative Explanations for Spending Changes

My results suggest that individuals are highly responsive to new risk information. In particular, the fact that individuals seek out disease-specific preventive care provides strong evidence that individuals are responding to the informational component of a household health shock. However, individuals may be separately responding to other characteristics of new diagnoses, which I explore in this section.

[26]The results also provide preliminary evidence that major health events provide a deterrent from low-value elective surgeries. However, Appendix Table B.6 highlights the strong presence of pre-trends in these models, which obfuscates the true causal effect of the diagnosis.

[27]Although I observe households utilizing more of these services—such as preoperative screenings or imaging services—it is unclear whether these are decisions made at the household level or by a physician who knows the family history and hence deems these services as appropriate (Finkelstein et al., 2021).

3.3.1 Moral Hazard & Salience

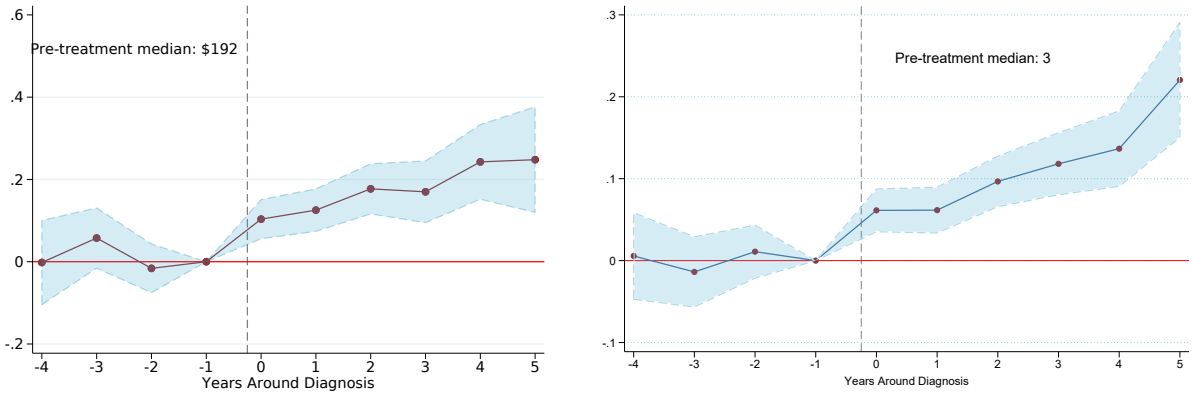
A natural response to Figure 1 is to conclude that the spending increase is driven by induced demand responses among the non-diagnosed individuals. A chronic diagnosis—such as diabetes—implies consistent, predictable costs on a household—such as through insulin prescriptions and endocrinologist visits. These additional costs, which are largely fixed for the individual, effectively reduce cost-sharing for the rest of the household, lowering future spot prices of (non-chronic) health care (Eichner, 1998; Kowalski, 2016).

Two features of the results suggest that these induced-demand responses are unlikely to be the principal driver of the results. First, the costs of a chronic diagnosis are typically larger in the year of diagnosis than in future years, especially when a hospitalization is required to diagnose the illness or there are acute complications that must be dealt with. This would suggest that if other household members were responding to changes in care prices alone, their responses would be much larger closer to the diagnostic event, and more muted in following years. Figure 1 does not show this to be true, either for overall utilization or the use of wellness visits specifically. Second, in Figure 2 I show that even households for whom a focal diagnosis does not meaningfully change marginal prices for other household members (e.g., those enrolled in plans with zero deductible), health shocks continue to promote strong spillover responses. Were moral hazard the principal mechanism, households in plans with no deductibles would have no incentive to adjust spending decisions (Anderson et al., 2023).

It may also be that the intensity of major health events realigns household preferences to prioritize medical care. Individuals who experience the hospitalization of a household member may (over-)respond to the trauma of the event itself, changing their health consumption behaviors in order to avoid future hospitalizations. These "salience effects" are different from effects capturing the salience of a specific risk condition, which leads to meaningful behavior changes particularly in the case of preventive care investments, as discussed above (Fadlon and Nielsen, 2019; Hodor, 2021). Rather, the critical difference here is that these general

Figure 2. Effect of Chronic Diagnoses on Utilization: Households Facing Zero Deductible

(a) $\sinh^{-1}$(Total OOP Spending) (b) # of Preventive Visits



Notes: Figures show regression coefficients (and 95% confidence intervals) from "stacked" TWFE regressions estimating the effect of a new chronic diagnosis on utilization of other (non-diagnosed) household members. The sample is restricted to households enrolled in ESI plans with zero deductible at the time of the event. In panel (a), the dependent variable is the inverse hyperbolic sine of total OOP spending; panel (b) estimates the effect on the number of household preventive services per year using Poisson regression. Coefficients are presented relative to the year prior to diagnosis. Spending is measured in 2020 USD. Standard errors are clustered at the household level.

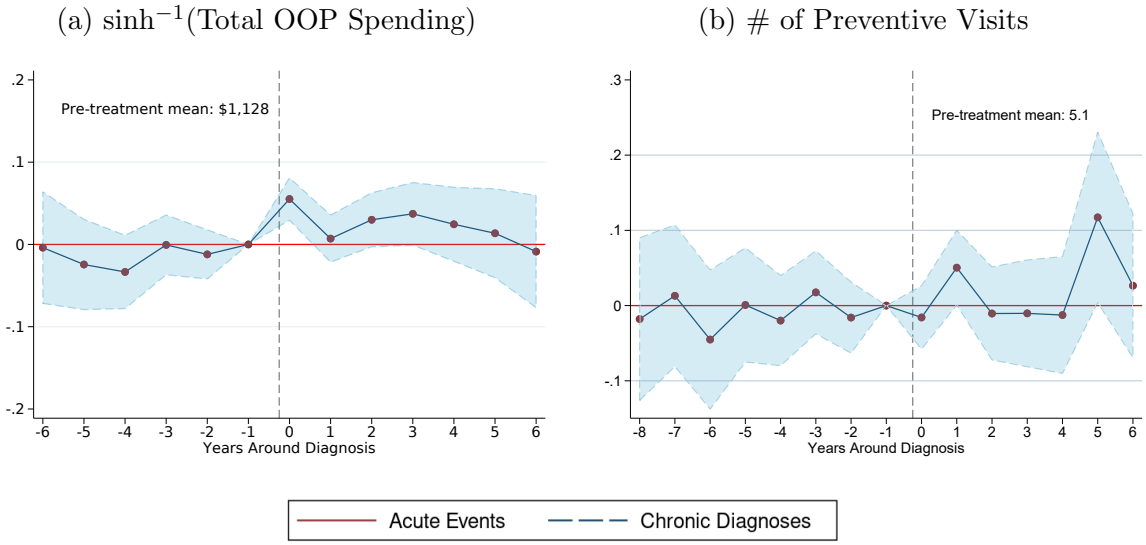
salience effects capture individual responses to overall negative health experiences that alter future risk *preferences*—by affecting the marginal utility of care—rather than risk *beliefs*.

To examine the impacts of salience effects in the absence of new health risk, I compare household responses to the acute events defined in Section 2.2. The intuition is that these events—for example, hospitalizations for viral infections—provide transient shocks that ought not to meaningfully change individuals' risk beliefs but may alter the relative valuation of health services (e.g., seeking vaccinations to reduce the likelihood of future hospitalizations). Hence, differences in responses across these two events provide a sense of how health risk information—over and above salience effects—drives the observed spillover responses.[28]

Figure 3 presents the results. Unlike in Figure 1, acute hospitalizations spur few changes in health behaviors among other household members. Acute hospitalizations are associated with a short-term increase in spending of about 5% in the year of diagnosis, but effects are

[28]Recall that acute events are comparable to chronic events as discussed in Appendix A. In fact, acute hospitalizations tend to be slightly more expensive and require longer hospital stays than chronic diagnoses; hence, were salience effects alone driving household responses, I should observe stronger household responses among the sample affected by acute diagnoses. I do not observe this.

Figure 3. Effect of Acute Health Events on Other Household Members' Healthcare Utilization



Notes: Figures show regression coefficients from "stacked" TWFE regressions, with 95% confidence intervals. Regressions estimate the effect of an acute hospitalization on medical utilization of other (non-diagnosed) household members (see Section 2.2). In panel (a), the dependent variable is the inverse hyperbolic sine of total OOP spending; panel (b) estimates the effect on the number of household preventive services per year using Poisson regression. Coefficients are presented relative to the year prior to diagnosis. Standard errors are clustered at the household level.

transitory. Acute health events do not cause increased spillover investments in preventive care. Given that acute hospitalizations make healthcare at least as salient—if not more so—than chronic diagnoses, the findings suggest that changes in risk preferences arising from a "health scare" are insufficient to entirely explain changes in behavior. Rather, new health risk information, such as about one's inherent genetic risk for a chronic condition, appears to drive observed changes.[29]

3.3.2 Health Information

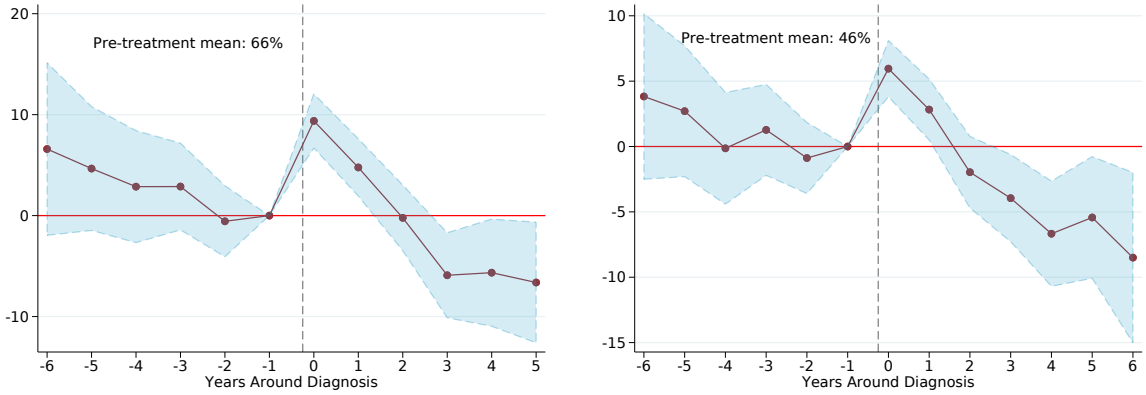
In addition to new health risk information, a diagnosis may give families institutional knowledge, such as about the value of medical care, the process of obtaining insurer-covered care, or how to establish provider relationships. It may be difficult to disentangle effects of this knowledge from new risk information, as the two generally co-move.

[29] As an alternative test for salience effects, I use variation in pre-diagnosis risk among household members affected by a new chronic condition. The results of this analysis—which likewise confirm that households respond to information over salience—are reported in Appendix B.6.1.

I separate these by focusing on a unique case where a diagnosis provides information about risk but not institutions: adherence to existing prescriptions, including preventative cardiovascular medications such as statins. These drugs are extremely common and are known to be effective in preventing future health problems (O'Connor, 2006). Importantly, individuals with active prescriptions already have sufficient institutional knowledge to receive this care; hence, when affected by a new household diagnosis, individuals may update risk beliefs without gaining new knowledge about *how* to obtain medication.

Figure 4. Effect of Chronic Diagnoses On Adherence to Existing Preventive Medications

(a) Pr(Refill Old Prescription) (b) Proportion of Days Covered



Notes: Figures show regression coefficients from "stacked" TWFE regressions, with 95% confidence intervals. Regressions estimate the effect of a new chronic diagnosis on adherence to preventive medications whose prescriptions were first written prior to the major health event. In panel (a), the dependent variable is a binary indicator for whether the prescription was refilled during the year; panel (b) estimates the effect on the proportion of days in a year covered by the medication (see Section 2.2). Standard errors are clustered at the household level.

Figure 4 presents the effects of a new chronic diagnosis on adherence to existing preventive prescriptions.[30] As expected, in the absence of new health information, individuals become less adherent to prescriptions over time. However, diagnoses in the household spur a resurgence in multiple measures of adherence, with affected individuals around ten percent-

[30]See Section 2.2 for a description of the measure of prescription adherence. I limit the sample to those who have prescriptions for these medications at least once annually for two years pre-diagnosis. An identification concern in these regressions is that adherence decays over time in response to financial or knowledge barriers (Slejko et al., 2014). As this may occur at different rates across individuals, these trends are not accounted for by household and year fixed effects. I therefore control for the number of years an individual has been in the sample.

age points more likely to fill a prescription immediately after a household health event. This illustrates that individuals respond to updated risk beliefs communicated by a diagnosis, not simply new information about the logistics of obtaining care.

4 Empirical Model of Belief Formation

Based on the empirical results above, I estimate a dynamic model of belief formation for households learning about health risks. In the model, one individual's health shock propagates health information across a household, leading each member to update their belief about subsequent health risks. The goal of the model is to identify implied health expectations based on observed health utilization choices—separate from other potential mechanisms—and measure changes in welfare associated with potentially under-informed beliefs.

My model takes into account how major health events in a home may alter both individual investments in health production—e.g., through the use of preventive health services (Grossman, 1972)—as well as other drivers of health utilization, such as the spot price of care (for moral hazard). I write a model that captures both the dynamic returns to investment in health and the tradeoff between risk protection and moral hazard (Cardon and Hendel, 2001).[31] The key parameters of interest—namely, how major health events alter beliefs about health risks—are identified based on observed spending and preventive care utilization (see Section 5.1 for more discussion on identification).

Formally, consider a household comprised of a set of individuals i, who in each year t choose medical spending m_{it} and preventive care investments s_{it}.[32] Although medical

[31]The model presented here is a simplified version that does not take into account household insurance plan choice or changes in risk aversion arising from major health events. The empirical evidence presented in Section 3 suggests that these are second-order concerns; however, I present the details of an enriched two-stage model of consumer choice that takes into account these features in Appendix C.2. Although static, the results of that model are qualitatively similar to those presented here.

[32]Note that throughout, the unit of analysis is an individual solving their own problem in isolation, with information exogenously arriving from other household members. The model is robust to considering this as a household problem, as discussed in Appendix Section C.2.

spending overall does not affect a person's future health needs, preventive care utilization is an investment that potentially improves one's health over time (e.g., reducing future spending). Below, I outline each decision-making process separately.[33]

4.1 Decision 1: Static Spending

Individuals make healthcare spending decisions based on some information about their health needs in a given period and their beliefs about their future health risks. Each year, an individual draws a transient health shock λ_{it}^{TR} (measured in dollars) from a stationary distribution $F(\cdot)$; this represents year-to-year fluctuations in health needs for acute conditions (e.g., illnesses and injuries). Additionally, individuals have an *ex-ante* belief at the start of the year that with some probability p_{it}, they will incur a chronic health shock of cost λ_{it}^{CH}.[34] An individual's *ex-ante* expected annual health spending, therefore, is given by

$$\mathbb{E}[\lambda_{it}] = \mathbb{E}[\lambda_{it}^{\text{TR}}] + p_{it}\mathbb{E}[\lambda_{it}^{\text{CH}}]. \tag{3}$$

Individuals make static decisions about total health spending m_{it} to match their realized health needs λ_{it} given a quadratic loss function that takes into account the OOP costs

[33] In keeping with previous work, m_{it} is measured in dollars (Einav et al., 2013; Marone and Sabety, 2022); however, s_{it} is measured as the discrete number of visits annually. This is done to be consistent with the empirical work in Section 3, as well as to more closely capture individual decision-making—as total billed spending for preventive care is generally unobservable to the individual, particularly since OOP spending for preventive care is almost zero—and to make the model's state space more tractable. As in previous work, I abstract away from within-year claim timing (Einav et al., 2013; Marone and Sabety, 2022).

[34] As discussed below, the primary model results are unaffected by allowing these shocks to be correlated and estimating the correlation parameter ρ; hence, in the simplest version of the model, they are assumed to be independent.

$c(m_{it}, s_{it})$ of chosen spending levels,[35]

$$u_{it} = (m_{it} - \lambda_{it}) - \frac{1}{2\omega_i}(m_{it} - \lambda_{it})^2 - c_{it}(m_{it}, s_{it}). \tag{4}$$

Individual heterogeneity in moral hazard—indicating differing levels of demand elasticity for health services—is modeled using the parameter ω_i. That is, individuals with greater values of ω_i increase their demand for m_{it} by more than others following an unexpected change in $c(\cdot)$. Note that this includes how individuals respond to static health prices for both transient and chronic spending, given the construction of λ_{it}; hence ω_i captures individual responsiveness to the marginal cost of health services relative to the expected marginal benefits of health production in the future.[36]

Solving the expected-utility maximization problem is straightforward; however, as the marginal OOP cost changes based on where it is evaluated, the solution depends on which "region" of OOP costs an individual finds themselves in conditional on their health shocks. If the realized acute health shock is negative (or sufficiently small relative to the shift parameter), individuals will choose $m^*_{it} = 0$ as spending is required to be non-negative; otherwise, optimal spending follows the condition:

$$1 - \frac{1}{\omega_i}(m_{it} - \lambda_{it}) - c_{ijt} = 0 \Rightarrow m^*_{it} = \max\left[0, \lambda_{it} + \omega_i(1 - c_{ijt})\right]. \tag{5}$$

That is, medical expenses in each period are chosen so that the marginal utility of those services is equal to the marginal (known) OOP cost. Equation 5 highlights two impor-

[35]Note that although this function appears to be a symmetric loss function, the additional restriction that OOP costs are always (weakly) less than total costs—meaning that $c'_{it}(\cdot) \leq 1$ at all points in an insurance contract—implies that individuals prefer a constant amount of over-spending (e.g., $\lambda_{it} + \delta$) than a constant amount of under-spending (e.g., $\lambda_{it} - \delta$). This is consistent with the notion that all else equal, an individual would prefer to spend slightly more than their health shock than slightly less, which is reasonable in the context of health spending. Appendix Section C.1.1 contains a proof of this result and additional discussion.

[36]As the main goal of this model is to identify the effects of major health events on health risk beliefs while adjusting for individual moral hazard effects, I parameterize these values based on observable demographics using the results of Einav et al. (2013) rather than estimating them directly (Section 4.4). Model results are robust to estimating these directly, as is done in Appendix Section C.2.

tant features of the model: first, new chronic diagnoses may generate moral hazard effects among price-sensitive individuals by exogenously lowering $c'(\cdot)$ for other household members. Second, despite the apparent symmetry of the quadratic loss function, the features of marginal cost-sharing in insurance contracts ensure that individuals will always choose to over-spend—rather than under-spend—relative to a realized health shock λ_{it}.[37] This is a desirable feature in models of health spending with imperfect information, where the utility costs of over-spending might be systematically lower than under-investing in health care

4.2 Decision 2: Preventive Care Investments

While the choice of spending (m_{it}) is a static one, individuals' choices of preventive services (s_{it}) influence future shocks λ_{it} and provide information on p_{it}. Both of these effects are specified in the model.

First, preventive care may directly affect an individual's future expected health spending, by both reducing the likelihood of future expensive procedures and ensuring that chronic diagnoses are discovered early in a primary care setting, avoiding costly hospitalizations. This return on investment is modeled by linking expectations for future shocks $\lambda_{i,t+1}$ to decisions today:

$$\mathbb{E}\left[\lambda_{i,t+1}\right] = \gamma\left(\sum_{\tau<t} s_{i\tau}\right) \cdot \left(\mathbb{E}\left[\lambda^{\text{TR}}_{i,t+1}\right] + p_{i,t+1}\mathbb{E}\left[\lambda^{\text{CH}}_{i,t+1}\right]\right), \tag{6}$$

where $\gamma(\cdot)$ relates previous preventive care use to percentage reductions in expected future spending.[38] An alternative approach to the one modeled here would be to allow preventive care to directly shift the mean or variance of underlying distributions for (λ^{CH}, λ^{TR}), with the intuition that preventive care makes future diagnoses *less likely*. However, for the chronic diagnoses most common in my sample, preventive care prioritizes risk monitoring and early

[37] See Appendix Section C.1.1 for a proof of this fact.

[38] As discussed in Appendix C.1.2, the primary model results are robust to various degrees of flexibility in specifying $\gamma(\cdot)$. These include a single parameter γ, estimated in the primary model, as well as linear, exponential, and second-order spline functions relating primary care visits to reductions in future spending.

diagnosis (e.g., reducing costs) over mitigating risk (e.g., shifting distributions). This is particularly true given the heritability and rapid onset of the conditions in my sample. Additionally, the selected approach allows for separate identification of changes in beliefs and $\gamma(\cdot)$: the returns from preventive care are identified using diagnosed individuals and never-diagnosed households, while belief updating relies only on the diagnosed households.

Preventive care also provides individuals with information about their health risks, p_{it}. This is modeled by a Bayesian process, with each individual's prior specified as a normal distribution (in log-odds space) with mean $\mu_{p0,i}$ and variance $\sigma^2_{p0,i}$. Future health signals are also normally distributed in log-odds space in order to obtain closed-form solutions for belief updating while containing probabilities in the unit interval:

$$\log\left(\frac{s_{it}}{1-s_{it}}\right) \sim \mathcal{N}(\overline{p}_i, \sigma^2_s). \tag{7}$$

That is, each preventive visit's signal is distributed around some observable proxy for individual true risk $\overline{p}_i$ with noise σ^2_s.[39] Absent data on actual underlying health risks, I proxy $\overline{p}$ using logistic regressions predicting each individual's probability of a new chronic diagnosis in a year as a function of observable demographics, past acute and chronic health events in the household, and family medical history (including pre-existing conditions). Although imperfect, these proxies are similar to the information a medical professional might convey in a preventive visit, as true health risks are not observed perfectly in that setting either.[40]

[39]Hence the evolution of the mean and variance parameters can be written as: $\sigma^2_{pi,t+1} = \frac{\tilde{\sigma}^2_{it}\sigma^2_{pi0}}{\tilde{\sigma}^2_{it}+s_{it}\sigma^2_{pi0}}$ and $\mu_{pi,t+1} = \frac{\tilde{\sigma}^2_{it}\mu_{pit}+\sigma^2_{pit}\tilde{\mu}_{it}}{\tilde{\sigma}^2_{it}+\sigma^2_{pit}}$, where the variable s_{it} indicates how many health signals an individual has received by the end of period t (Crawford and Shum, 2005). In estimation, I model σ^2_s as being inversely proportional to an individual's average chosen annual spending on preventive care in order to accommodate the potential stacking of visits.

[40]Specifically, for an individual i and diagnosis d, the underlying risk is the predicted probability from the logistic regression $\mathbb{1}\{d=1\} = \vec{\beta}(agesex_i) + \gamma_1 \text{Past Acute Event}_i + \gamma_2 \text{Past Chronic Event}_{-i} + \gamma_3 \text{Past Acute Event}_{-i} + \vec{\delta}(familyhistory_i) + \varepsilon$ for a vector of age-sex bins and dummies for pre-existing conditions in a family's medical history. Individual risk probabilities are then pooled across diagnoses with $\overline{p_i}$ set as the maximum probability of a diagnosis.

4.3 The Role of Major Health Events

A single individual's health shock propagates throughout a household, providing each household member with new information about p_{it}. Given the quasi-randomness of these diagnoses, I model these responses as a discrete shift p_{it}:

$$\Delta(p_{it}) = \pi_1(\text{Chronic Event})_{f,-i} + \pi_2(\text{Acute Event})_{f,-i} + \pi_3(\text{Acute Event})_{f,i}, \tag{8}$$

where f denotes the household members affected by person i's diagnosis. Both chronic and acute health shocks are allowed to propagate across a household, even if the signal should rationally provide little health risk information. The choice to model belief evolution as a discrete shift in this setting allows the model to match persistent changes in behavior observed in Section 3; however, I also estimate a version of this model that allows these effects to decay over time, and estimate that decay to be near zero (Appendix C.2).[41]

4.4 Summary

Taken together, individuals respond to their own beliefs p_{it} and both individual and household shocks by choosing $\{m_{it}, s_{it}\}_{t=1}^{T}$ to solve the dynamic Bellman equation

$$V_{it} = \max_{\{m_{it}, s_{it}\}_{t=1}^{T}} \left\{ (m_{it} - \lambda_{it}) - \frac{1}{2\omega_i}(m_{it} - \lambda_{it})^2 - c_{it}(m_{it}, s_{it}) + \delta \mathbb{E}[V_{i,t+1}]) \right\}, \tag{9}$$

where households discount future years consumption at a rate of $\delta = 0.95$. Equation 9 highlights that individuals are forward-looking in choosing preventive care investments as a central form of health production. Rewriting the Bellman equation to separate out the static choice of m_{it} in each period assists in highlighting the role of belief formation in the

[41] The main predictions of the model are also robust to alternative specifications, including a Bayesian learning framework with signal means as the equilibrium parameters of interest and an adaptive learning framework where individual beliefs are specified as an AR(1) with some dependence $\rho < 1$ on the previous period's beliefs. For a more in-depth review of the relative strengths and weaknesses of Bayesian or adaptive learning in structural modeling, see Aguirregabiria and Jeon (2020).

dynamic decision-making process:

$$V_{it} = \max_{\{m_{it}, s_{it}\}_{t=1}^{T}} \left\{ u_{it}^{\text{TR}} + p_{it} u_{it}^{\text{CH}} + \delta \mathbb{E}[V_{i,t+1} | \Delta(p_{it})] \right\}, \tag{10}$$

where u_{it}^{TR} and u_{it}^{CH} each represent the components of the overall utility index (Equation 4) arising from transitory and chronic health shocks. Equation 10 underscores two consequences of family health shocks: first, exogenous changes to p_{it} may drastically change overall medical spending m_{it}, as each period's expected utility exogenously shifts down (due to increased anticipation of negative utility shocks from chronic conditions). Second, households may respond to this new risk information by consuming additional preventive care; in the model, this allows individuals to recenter their beliefs about health risks gradually over time. Both of these results are consistent with the evidence presented in Section 3.

5 Parameterization & Estimation

The key parameters of interest in this model are the vector of response coefficients $\vec{\pi}$, which capture how individuals adjust their own beliefs about health risks in response to household signals. Additional parameters include individual prior beliefs about health risks $\{\mu_{p0,i}, \sigma^2_{p0,i}\}$, parameters governing the distributions for health shocks $\{\lambda_{it}^{\text{TR}}, \lambda_{it}^{\text{CH}}\}$, and heterogeneity in price responsiveness, ω_i. As I focus here on the evolution of health beliefs, I calibrate individual parameters governing underlying health shocks and price responsiveness based on previous work. I model λ_{it}^{TR} as a shifted lognormal distribution governed by a mean, variance, and shift parameter $(\mu_{\lambda,i}, \sigma^2_{\lambda,i}, \kappa_i)$. That is, each individual in each period draws λ_{it}^{TR} from a distribution $F(\mu_i, \sigma_i, \kappa_i)$ such that

$$\log(\lambda_{it} - \kappa_i) \sim \mathcal{N}(\mu_i, \sigma_i^2). \tag{11}$$

This choice of distribution accommodates the skewed nature of spending data while also allowing for a nontrivial fraction of individuals to choose zero spending in a given year (matched by κ_i). I calibrate the three hyper-parameters $(\mu_{\lambda,i}, \sigma^2_{\lambda,i}, \kappa_i)$ using the empirical distribution of annual spending of a matched sample of patients *not* included in the structural estimation, including both individuals enrolled in other firms in Marketscan and spending data for in-sample individuals between 2014–2018.[42] Similarly, I calibrate expected spending on chronic conditions λ_{it}^{CH} based on the empirical distributions of disease-specific spending at the HCC level; I modeled separate distributions for the year of diagnosis and follow-up years to differentiate between diagnostic and maintenance costs.[43]

Finally, I calibrate ω_i using the estimated regression coefficients predicted by Einav et al. (2013). This variation—which captures how price sensitivity differs across individuals—is likely a second-order effect in the takeup of preventive care and risk belief updating, particularly as noted in Section 3. The model continues to adjust for individual-level variation in price sensitivity, even though individual coefficients are not equilibrium objects in my structural estimation.[44] These calibrations dramatically reduce the state space of the model and allow for tractable estimation.

Estimation is done using the generalized method of moments and forward induction (Hansen, 1982). Given that the choice of the continuous variable m_{it} is static and the state space of the dynamic variable s_{it} is small, estimation is feasible with minimal assumptions.[45] Equilibrium parameters are chosen based on moments that match the model predictions

[42]Parameters are calibrated by binning individuals based on including age, sex, risk score quartile, and enrollee relationship, and then constructing moments based on the observed empirical spending distribution. This is done using three properties of a shifted lognormal distribution: $\overline{\lambda} = \exp(\mu+\frac{1}{2}\sigma^2)+\kappa$, $\lambda^M = \exp(\mu)+\kappa$, and $\frac{\text{sd}(\lambda)}{\overline{\lambda}} = \sqrt{\exp(\sigma^2)-1}$, where λ^M denotes the median. The solution to this system of equations given the moments of the empirical distribution of λ identifies the three hyperparameters μ, σ, κ. In order for shocks to be meaningful, we restrict $\lambda_{\mathcal{I}t} < m_{\mathcal{I}t}$ in each period when drawn.

[43]Note that in the primary specification, these shocks are uncorrelated; however, the primary model results are robust to allowing for correlations between these two shocks, as discussed in Appendix Section C.1.2.

[44]Appendix C.2 presents a more detailed model that does estimate variation in ω_i across individual demographics and medical histories, and finds that the key model results are unchanged.

[45]The only restriction made on the state space transition matrix is that individual investments in preventive care cannot *drop* by more than 2 visits from period t to $t+1$.

to observed data, including the reduced form results presented in Section 3. I match the model on the predicted levels of overall health spending and preventive care utilization at the individual level—including the mean, median, and RMSPE of predictions—and regression coefficients for simple difference-in-differences regressions estimating the effect of a chronic diagnosis in the home on spillover health spending and spillover preventive visits.[46]

5.1 Identification

The main identification challenge is to pin down the elasticity of beliefs to information, $\vec{\pi}$. To do so, I use two sources of variation. First, differences in observed spending decisions for spillover household members, compared to not-yet-treated and never-treated household members, generate the needed variation to identify the effect of a diagnosis. I link the model to key moments estimated in the regressions presented in Section 3 via indirect inference in order to leverage this variation. Second, in order to separate the evolution of risk beliefs from alternative mechanisms such as price and salience effects, the model leverages variation across households enrolled in different plans (e.g., households facing large marginal price changes compared to others with no relevant changes) and within-household variation in diagnostic risk (e.g., differences between affected members with the appropriate intrafamilial relationship for a risk signal to be relevant).

An additional feature of the dynamic model is its link between current healthcare consumption (s_{it}) and future health returns (λ_{it}). Modeling this link explicitly separates two sources of variation in the data: health shocks both affect individual beliefs—thus changing health expenditures—and generate dynamic effects through the return of health inputs on future health. Hence, the main source of variation for belief updating comes from intertemporal relationships between health shocks and the choices of health investments. My dynamic model accounts for these two channels separately, allowing me to pin down changes in static belief formation π_1 separate from the dynamic effects of increased health consump-

[46] As in Section 3, I appropriately transform the dependent variables of total health spending and preventive care takeup.

tion. The dynamic links between current investments in preventive care and future health risks also place additional restrictions on the model preventing unreasonable belief divergence post-diagnosis (e.g., beliefs about risk that grow excessively over time).

Despite this identifying variation, residual concerns may remain that there are unobserved mechanisms that causally affect spending after a diagnosis and inappropriately load onto the estimated belief elasticities. Although addressing all such concerns is impossible, I present evidence in Section 6 and Appendix C that the estimated values for $\vec{\pi}$ and the subsequent welfare calculations are robust to a suite of modeling choices. These include incorporating additional flexibility into parameterizing assumptions, estimating calibrated parameters as structural objects, and integrating additional features of the data-generating process such as household utility maximizations and insurance plan choices.

6 Structural Results

Table 4 presents the equilibrium model parameters estimated by GMM. Standard errors are calculated as discussed in Cocci and Plagborg-Møller (2021), and hence represent conservative, "worst-case standard errors" for calibrated structural parameters.[47]

Prior to individual learning about risk—either through household health events or preventive screenings—I estimate that individual beliefs about a future major diagnosis affecting them are centered at about 1.4%. There is considerable heterogeneity in prior beliefs: the 10th percentile of the distribution has prior beliefs of 0.25%, while the 90th percentile has prior beliefs of 11.7%. However, considering a relative in-sample diagnosis rate of about 2.7%, roughly 80% of the individuals in my sample under-estimate their true risk of a major health event occurring. Preventive care is therefore an important vehicle for individual

[47] These standard errors allow for arbitrary correlations across empirical moments, and balance the tradeoff of computational feasibility—particularly in a setting where bootstrapping is difficult given that some of the moments are the result of high-dimensional fixed-effect regression estimation—against accurate coverage in conditions where correlations across moments cannot be derived analytically.

Table 4. Estimated Structural Parameters

		Dynamic Model	
		Estimate	95% Confidence Interval
Panel A: Initial Beliefs			
μ_{p_0}	Prior Mean	1.433	[0.553, 2.314]
$\sigma^2_{p_0}$	Prior Variance	2.389	[1.756, 3.023]
Panel B: Learning from Preventive Care Investments			
μ_s	Signal Mean	0.023	—
σ^2_s	Signal Variance	0.982	[0.880, 1.083]
γ	Health Returns from Prevention	0.705	[0.648, 0.762]
Panel C: Learning from Major Health Events			
π_1	Family Chronic Event	9.152	[8.952, 9.351]
π_2	Own Acute Event	3.497	[2.046, 4.949]
π_3	Family Acute Event	1.023	[0.830, 1.227]

Notes: Table presents estimated equilibrium parameters of the model estimated via GMM on a sample of $N = 387,216$ enrollees in $149,938$ households between 2006 and 2013. All average parameters are expressed in terms of probabilities (%), while variances are expressed in log-odds. Signal mean μ_s is not estimated via GMM, but rather through individual-level risk predictions. Standard errors are calculated following Cocci and Plagborg-Møller (2021).

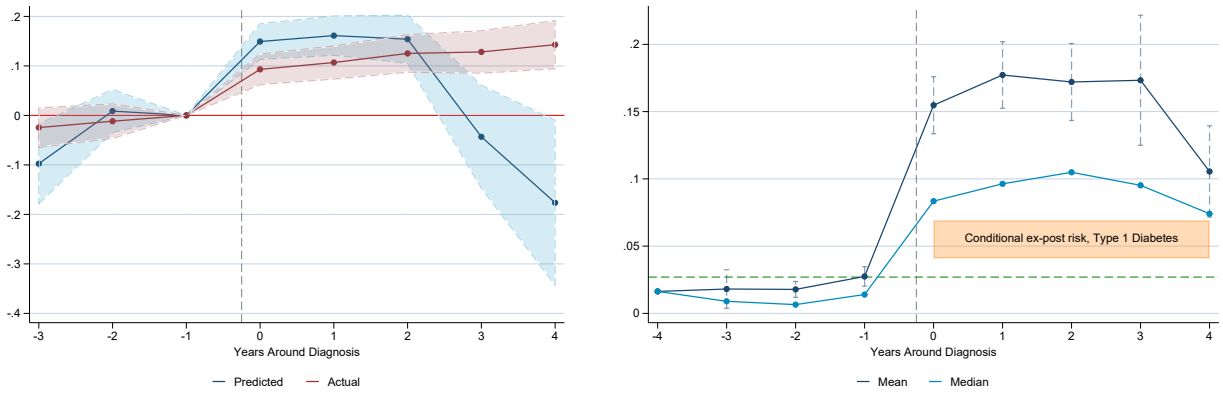
learning: the variance of preventive signals is roughly 40% of the variance of prior beliefs, suggesting that preventive care improves the beliefs of the representative individual.

The key result of the model is that household health events—particularly chronic diagnoses—are major drivers in individual health beliefs. Chronic diagnoses increase individual risk beliefs by 9.2 percentage points, a more than six-fold increase from the average prior belief. This effect is much larger than that of acute events, either for oneself (3.5pp) or other household members (1.0pp). This central result is robust to numerous alternative specifications allowing additional flexibility, discussed in Appendix C.

Figure 5 presents key takeaways from estimation. In the first panel, I highlight the overall match quality of the model, reporting TWFE coefficients for the effect of a new diagnosis on the inverse hyperbolic sine of spillover utilization. I show coefficients for both observed spending and spending predicted by the model. In general, the model captures the increase in spending for the first 3 years following a diagnosis.

Figure 5. Model Predictions: Non-Diagnosed Spending and Beliefs Around a New Diagnosis

(a) TWFE Regression Coefficients, Total Spending (b) Average and Median Health Risk Beliefs



Notes: Figures show key predictions of the structural model described in Section 4. Panel (a) reports TWFE coefficients for the causal effect of a new chronic diagnosis on both observed spillover household utilization and spending predicted by the model (similar to Figure 1; see Equation 1 for the specification). Panel (b) reports recentered time series indicating average and median individual risk beliefs for the same population (individuals affected by a new chronic diagnosis in their home), averaged over draws from individual posterior distributions. The green horizontal line in Panel (b) illustrates the average in-sample rate of diagnosis (∼2.7%); the orange range indicates the estimated *ex-post* risk of a diagnosis of type 1 diabetes following a sibling's diagnosis ([4.1%,6.9%]) (Harjutsalo et al., 2005).

The major implication of the equilibrium parameters is presented in the second panel of Figure 5. Here, I present changes in predicted beliefs for affected individuals in a recentered time series. Prior to diagnosis, individuals tend to slightly underestimate their risks relative to the in-sample diagnosis rate. Following the diagnosis, beliefs about the probability of a future health shock increase to between 15% and 20% for the average individual (roughly 10% for the median). These shifts persist for the first four years after the event, after which investments in preventive care (which provide more moderated risk signals to individuals) cause both average and median beliefs to decline slightly.

However, even by the end of year five, both the median and average individuals have risk beliefs that are higher than an expected *ex-post* conditional probability of a diagnosis, given the clinical information of a diagnosis. To see this, the second panel of Figure 5 includes the estimated range of a sibling's risk of developing type 1 diabetes, conditional on knowing another sibling is already diagnosed (Harjutsalo et al., 2005). This range is an instance where there is large conditional risk-sharing—hence, where a signal is particularly informative—

yet even in this case, estimated beliefs exceed the standard measures of conditional health risk even five years post-diagnosis. I explore the welfare implications of these facts in the following section.

6.1 The Welfare Effects of Health Shocks

Based on the estimated structural parameters, I can construct a measure of each individual's expected utility gain from new health risk information. These utility differences constitute a willingness to pay (WTP) measure which allows me to benchmark the welfare effects of providing health risk information and consider how welfare changes as the type of information an individual receives changes.

Throughout this section, I focus only on individuals affected by a chronic diagnosis in their household. WTP comparisons are made against a counterfactual state in which household members continue to invest in and learn from their own preventive screenings, but have no discrete shift in their beliefs following a major health event. As Equation 9 is estimated in dollar terms, this comparison is then simply the difference:

$$WTP_{it} = V_{it}(\text{information}) - V_{it}(\text{no information}). \tag{12}$$

I present results comparing WTP in the period of a diagnosis, excluding follow-up welfare effects; including later periods does not qualitatively change the results. Equation 12 measures how much individuals would be willing to pay for the information contained in a diagnosis, given individuals' interpretation of that information.

In general, I find that any potential welfare gains from risk information are overshadowed by the interpretation of that information for a substantial fraction of the population.[48] I estimate that roughly 48% of the affected individuals in my sample would be willing to pay

[48] Appendix Figure D.1 shows the full distribution of WTP across the affected individuals in the sample.

to *avoid* new information. Among this population, losses are skewed: the average (median) welfare loss is \$100 (\$29), constituting a nearly 89.3% (7.4%) decline from baseline utility.

How much would someone need to react to a health shock so that the information is *ex-ante* welfare-enhancing? I assess this by examining the maximum allowed shift in beliefs following a chronic diagnosis that increases welfare for each affected individual. The median (average) such allowable increase is 2.77 (7.46) percentage points.[49] This implies that the estimated average shift in beliefs from new information is roughly 33% larger than the allowable amount, potentially driving the estimated welfare penalties.

At first glance, a welfare penalty associated with new information is counter-intuitive. This result, however, highlights the discrepancy between how informative a signal actually is and how informative an individual interprets it to be. Given that individuals appear to over-update in response to information (Figure 5), the welfare results are consistent with the findings presented in Section 3. Over-updating reduces individual expected utility through two mechanisms: first, increased expected spending with lower expected health returns (e.g., over-utilization of low-value services), and second, greater uncertainty in spending, especially for more risk-averse individuals.[50] Placing information into context could mitigate utility losses through both channels.

6.2 The Role of Belief Updating in Welfare Penalties

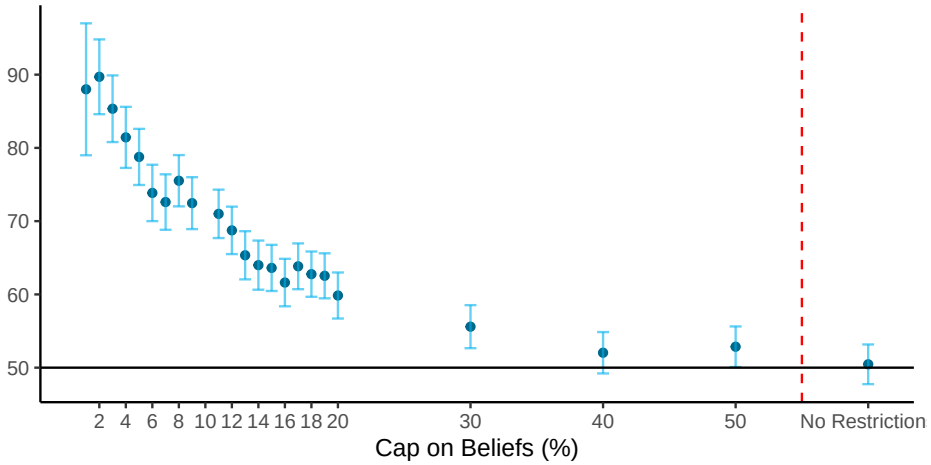
Given these results, I consider how limiting responsiveness to chronic risk information (π_1) might change its value. I perform simple counterfactual scenarios imposing arbitrary upper bounds $\overline{p}$ on post-diagnosis beliefs.[51] This exercise intuitively illustrates how valuable one might expect health information to be in contexts where individuals more appropriately interpret information based on their conditional risks.

[49] See Appendix Figure D.2 for the full distribution.

[50] The second mechanism is similar to an anticipatory "disutility" discussed by Oster et al. (2013). Appendix Figure D.3 highlights the correlation between welfare results and household characteristics, such as pre-diagnosis risk and risk aversion.

[51] Results are qualitatively similar when allowing this threshold to vary, for example as a multiple of individual-specific predicted risks.

Figure 6. Bounding π_1 Increases the % of Individuals Valuing Health Risk Information



Notes: Figure shows counterfactual simulations estimating differences in WTP for information across states where (i) individuals update beliefs with a post-diagnosis belief cap set at $\overline{p}$, and (ii) individuals do not learn from diagnoses. Figure shows the percentage of individuals with positive WTP at each value of $\overline{p}$. On the far right, observed equilibrium values are plotted for comparison. Utility gains are shown in Appendix Figure D.4. Utility differences are measured according to Equation 9 in 2020 USD, and are calculated at the year of diagnosis. Error bars represent 95% confidence intervals.

Figure 6 presents the results as the belief cap $\overline{p}$ varies. Each point plots the average estimated difference in *ex-ante* expected utility when information is transmitted, conditional on the cap (Equation 12). The figures include the estimated baseline utility gains on the far right for comparison. Panel (a) illustrates that the share of individuals who value health information is sharply increasing as $\overline{p}$ prohibits large swings in underlying risk beliefs. Without restrictions, about half of individuals have a positive WTP for information; however, even restricting beliefs of future chronic risk to be 10% or fewer—a relatively generous bound, given true conditional risks are generally below 7%—expand this share to be almost 70%. The value of information increases further as caps become more restrictive; ultimately, 90% of individuals value information when beliefs are capped at 3% or fewer, consistent with the analysis reported above. As the cap becomes excessively restrictive, the value of information diminishes, reflecting that health information has value insofar as it persuades individuals to update beliefs to a "correct" posterior.[52]

[52]Appendix Figure D.4 highlights the relative magnitude of these utility gains. I find that gains are maximized at $\overline{p} \approx 3\%$, with a roughly 15% increase in baseline *ex-ante* expected utility.

6.3 Additional Policy Simulations

The structural model estimated here allows us to move beyond welfare estimation into policy simulations comparing alternative approaches to improve the value of risk information. Health-related spillovers—especially within families—can be leveraged as policy tools to improve screening and public health, and have therefore attracted recent research. However, welfare implications of differing approaches are not obvious, particularly given limited understanding of individual reactions to information; using the estimated model allows for careful comparisons of these options.

My model results are consistent with the empirical findings presented above and additional work studying the role of selection into screenings (Einav et al., 2020). In particular, I find that the value of new risk information—without corrections to belief updating rules—is greatest for individuals with low *ex-ante* risk scores, who may have large existing errors between underlying and perceived risk.[53] Similarly, the model predicts that medical histories are particularly valuable for those with salient familial relationships; as discussed in Section 3, individual WTP for information exhibits strong within-family variation, with some relationships (e.g., siblings for Type 1 diabetes and spouses for Type 2 diabetes) exhibiting greater returns for new health risk information. Together, this suggests value in policies leveraging medical histories and machine learning, among other techniques, to construct a more targeted approach to screenings and the transmission of new risk information.

However, the model suggests that interventions seeking to improve information *interpretation*, rather than simply information *access*, may be more valuable and effective. Health literacy programs that either improve the precision of risk signals or more clearly underscore the value of specific health services for a risk condition may dramatically improve patient welfare while reducing overall health spending. This may include improving the return on primary care investments as a way to correct inappropriate health beliefs or to limit the use

[53] Appendix Figure D.5 shows the results of a simulation using the structural model to estimate the optimal revelation of health risk information based on observable demographics and taking into account individual belief responses.

of pseudo-preventive low-value services. In fact, these policies may have broad impacts on both spending and welfare.

The model results could be extended in several meaningful ways to improve its use for policy evaluation. First, future work could relax the assumption that individuals have no control over their chronic care health costs. This is particularly interesting in non-ESI populations, including uninsured or Medicaid-enrolled individuals for whom chronic diagnoses may impose large financial burdens (Hadley, 2007). This is related to the effect of liquidity constraints on spending adjustments (Gross et al., 2020), another important consideration to be integrated into the model. Finally, future work might integrate this model with other costs incurred through living with a chronic condition, including earnings penalties and job lock (Biasi et al., 2019; Eriksen et al., 2021; Garthwaite et al., 2014).

7 Conclusion

This paper assesses the extent to which information about one's health risks alters individual decision-making in health care. I demonstrate that an individual diagnosis propagates across household members, who respond to new health risk information by altering their use of both high- and low-return services. These changes in behavior are best explained by individuals reassessing health risks, rather than responding to financial incentives or salience effects. However, while access to new health information changes behavior in meaningful ways, it does not necessarily leave individuals better off.

I use a structural approach to quantify the welfare effects of new health information. I find that for a large subset of the population, gains from information are swamped by extremely large shifts in estimated *ex-post* risks. Bounding the extent to which individuals increase their risk beliefs post-diagnosis makes information welfare-improving for over 90% of individuals. The findings of the model are robust to multiple model specifications, and the

model results can be meaningfully used to consider policies leveraging health information to improve social welfare.

Increasing an understanding of how consumers interpret new information is at least as vital as improving their access. Family health experiences are powerful forces in shaping individual behaviors and decisions; however, witnessing these experiences may lead individuals to "over-react" when making future consumption decisions. Individuals and families living with the risk of chronic illness may be better off as they are taught to seek out high-value medical care and temper high expectations of negative outcomes.

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