

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

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Abstract

Individuals learn about their health risks from witnessing family members' health experiences. I assess how the (mis)-interpretation of this risk information limits the potential welfare gains from these spillovers, using household health shocks as quasi-random risk signals. Adverse health events generate strong, persistent informational spillovers within a family, increasing spending by about 10% annually among unaffected household members. I show that these responses are more consistent with households reevaluating their health risks than other potential mechanisms; however, responses include increased utilization of both high- and low-return services. To evaluate welfare effects, I estimate a structural model of health choices in which individuals learn about their health risks over time. The model suggests that consumers over-respond to health information by over-weighting their health risks, resulting in welfare losses averaging \$2,788 from the new information. Placing bounds on how consumers update their beliefs in response to risk information improves welfare for 80% of households and suggests the revelation of health risk information can be optimally targeted on household demographics to improve social welfare gains.

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 health care choices. Through connections with family, friends, and neighbors, individuals form expectations of their own 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, both due to the close proximity of family members and the high relevance of their experiences due to shared genetic profiles and lifestyle choices, both of which influence expected health care consumption. Understanding how individual health experiences shape family health behaviors is important for policies aiming to improve public health, such as those attempting to address high levels of health care spending or the takeup of high-value health services.

One especially salient dimension of health information individuals may learn from family health experiences is knowledge about health risks, including both current and expected future health care needs. The extent to which individuals receive and utilize risk information derived from health events is an important driver in potentially life-saving decisions, such as to receive screenings for important conditions or be vaccinated against COVID-19. While previous work has provided evidence that health events within a household generate spillover effects altering these behaviors (Hodor, 2021; Fadlon and Nielsen, 2019), questions remain as to whether individuals are updating their beliefs about health risks or responding to other forms of health information. Furthermore, the welfare effects of transmitting new health information depend critically on whether individuals interpret new information correctly. In particular, health events provide salient new evidence that may lead consumers to “over-correct” their beliefs about their health risks, resulting in suboptimal changes in health behaviors and, ultimately, welfare.

In this paper, I examine how consumers who receive health risk signals through witnessing a major health event—such as a diagnosis with a new chronic condition—within their household modify their assessments of their own risks and alter their choices accordingly. I study households with employer-sponsored insurance (ESI) obtained through large employers between 2006 and 2018. Highly-detailed claims data provides insight into how individuals respond to quasi-random health events in their family, including overall responses in plan choices and health spending as well as decisions about the use of specific services. Additionally, these data include rich variation in coverage generosity and plan characteristics among enrollees, an important fact I can leverage to separate changes in household beliefs about risk from other, potentially confounding effects of health events.

I show that major health events generate strong informational spillovers among non-

diagnosed household members. Those exposed to new health information significantly and persistently increase both their overall health care utilization and their investments in preventive care—particularly for services that are specific to the condition just diagnosed in their household. I show that these spillover effects are more consistent with individual learning than other potential mechanisms. The magnitude of these increases is constant across insurance plan designs—including plans without deductibles—suggesting that moral hazard concerns are not driving changes.¹ Additionally, chronic events induce stronger and more persistent behavior changes than acute health events, suggesting that salience effects arising from a traumatic health experience do not fully explain observed results (Dalton et al., 2020; Fadlon and Nielsen, 2019). Finally, I show that even individuals who are most familiar with the health care system—such as those taking regular preventive medications for cardiovascular health—are responsive to major health events, implying that learning about health *systems*, rather than health *risk*, is not the main driver of observed results.²

In general, one would expect receiving new information about one’s risk to lead to improvements in decision-making and welfare. Surprisingly, however, I demonstrate that the welfare effects of new information are not obvious from reduced-form analysis alone. Affected household members increase their use of “low-value” health services, procedures that are generally agreed to be cost ineffective due either to their reach (e.g., benefiting few patients) or their average returns (e.g., low levels of benefits relative to costs) (Colla et al., 2015). Households responding to chronic diagnoses are most likely to increase their utilization of low-value services that appear, from their perspective, most closely related to preventive care, including cardiac screenings before low-risk surgeries or imaging services for headaches or lower back pain. In addition, households do not meaningfully alter their insurance plan choices, even after large expected increases in health costs from managing chronic conditions. Both of these findings cast doubt on the extent to which new health information ultimately improves choice quality.

These findings motivate a structural approach to model the evolution of household decisions following health events and quantify the associated welfare effects of receiving health information. I write and estimate a model in which households form beliefs about their health risks over time. In my model, households first make decisions about their insurance coverage prior to receiving information about their health state in a period; once this information is realized, households choose health spending (Cardon and Hendel, 2001; Einav

¹As 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. For a more in-depth discussion of this abuse of notation, see Einav et al. (2013).

²This general learning may include systematic learning about health care organizations, the process of receiving insurance coverage for care, or building physician relationships (Sabety, 2020).

et al., 2013; Marone and Sabety, 2020). Novel to my model, health shocks take two forms: major health events and non-chronic health states. Major health events occurring in a household induce other household members to update beliefs about their own health risks, but also affect consumer choices by potentially lowering the conditional cost of non-chronic care and increasing household risk aversion. A structural approach allows me to separately identify these competing effects, resulting in a clearer estimation of the welfare effects from receiving health information.

A key challenge in my model is identifying changes in an individual’s beliefs about their health risks separate from these alternative explanations. I use multiple sources of variation in the data to decompose the effects of household health events. First, I use a broad set of health events which vary in their expected treatment costs to identify the effects of price changes resulting from a new diagnosis on spending decisions. More expensive conditions (e.g., cancers) are associated with stronger price effects than cheaper ones (e.g., asthma) and therefore are expected to induce stronger moral hazard responses. Second, I exploit variation in the availability and generosity of plans offered to households to separately identify changes in household risk aversion at the time of plan choice. Here, the intuition is that individual beliefs about risk determine optimal medical spending and coverage levels, while household risk aversion also determines the gradient of preferred coverage as the price or generosity of plans varies (Ericson et al., 2020). I complement this approach with additional information about the circumstances of a diagnosis (e.g., whether a hospitalization occurred) to further model risk preferences and risk beliefs separately. Finally, I use both acute and chronic health events to assess the extent to which individuals learn more generally about the health care system, rather than the causal effect of new information about health risks.

Counter to expected thought, the new information gained from health events is not welfare-improving for many households. Households would be willing to pay an average of about \$2,788 per household per year in order to *avoid* the health risk information communicated by a major medical event. 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. Counterfactual simulations suggest that bounding these changes in risk beliefs substantially increases consumer welfare. 80% of the households in my sample would find health information welfare-improving were their responses to such information mitigated. Finally, I demonstrate that the societal value of communicating health information can be improved by selectively revealing it to specific groups, such as those with higher *ex-ante* risk. This suggests that population health infor-

mation campaigns—including genetic testing programs and screening practices for important conditions such as COVID-19—can benefit from targeting specific groups.

My analysis contributes to a burgeoning discussion on the spillover effects of health information within social networks, including for family members responding to acute health events (Hodor, 2021; Song, 2021; Bouckaert et al., 2020; Fadlon and Nielsen, 2019) and community members responding to community-level health information, including vaccine compliance (Archibong and Annan, 2021) and infectious disease outbreaks (Agüero and Beleche, 2017).³ I contribute to this discussion in three ways. First, I illustrate that individuals are even more responsive to chronic diagnoses within their family than to acute events. Second, I explore the mechanisms behind these responses, showing that changes to how individuals assess their health risks appear to drive observed spending changes. Finally, I provide evidence that while health events increase investments in high-value care, they are also associated with large errors in risk assessments and the takeup of low-value care, resulting in welfare losses for households on average.

I also contribute to a growing literature estimating learning and preferences in structural models of health behavior (Barseghyan et al., 2018; Bundorf et al., 2021a). I incorporate the findings of this literature into the first dynamic structural model addressing the value of health information spillovers, and highlight the particular behaviors—such as information misinterpretation—that dampen potential welfare gains. My model encompasses previous work highlighting the role major health events play in inducing demand responses by changing spot prices for other care (Eichner, 1997; Kowalski, 2016). Additionally, I make use of previous identification results to simultaneously estimate weighted probabilities and standard risk aversion parameters in a nonlinear framework (Ericson et al., 2020).⁴

More broadly, I contribute to the literature on non-Bayesian learning, which emphasizes the disproportionate weight put on recent, and particularly salient, events (Kahneman and Tversky, 1973). Models that incorporate such ideas include Holt and Smith (2009), who find in an experimental setting that individuals significantly overweight new evidence (relative to typical Bayesian predictions) when it had a lower *ex-ante* probability of occurring. Other important models draw attention to biased beliefs in models of consumer choices, including their role in rationalizing choices that would otherwise require unreasonably high levels of risk aversion (Spinnewijn, 2015; Ortoleva, 2012; Paserman, 2008). My model builds on these by

³A 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; Oster et al., 2013); however, examinations of other diagnoses revealed a lack of noticeable responses (Kim et al., 2019; Dupas, 2011).

⁴See Barseghyan et al. (2013) and their later review paper Barseghyan et al. (2018) for a more thorough discussion of the literature estimating models of probability weighting in other settings in economics.

simultaneously estimating biased beliefs and risk preferences, providing a micro-foundation of how individuals form health beliefs in a setting of largely small-probability events. My model provides additional insight into the development of subjective health beliefs; in particular, I provide new evidence that explains why consumers may be better at predicting their relative risk rather than their absolute risk (Bundorf et al., 2021b), and how biases in assessing their own health risks may arise (Arni et al., 2021).

Finally, my work is relevant to the well-established literature exploring suboptimal health decisions made by most consumers (Abaluck and Gruber, 2011, 2016a; Abaluck and Compiani, 2020; Baicker et al., 2015; Iizuka et al., 2021; Ketcham et al., 2012; Handel, 2013; Handel and Kolstad, 2015). This literature includes an ongoing discussion about the extent to which improving health information generally may improve decision-making (Abaluck and Gruber, 2016b; Gruber et al., 2020; Cutler and Zeckhauser, 2004). My analysis reveals that some health signals—such as major health events—do little to align household choices with the value of medical care, and may instead lead to an increase in the over-utilization of services that provide little or no benefit to households. Hence, simply improving access to health information may shift consumers only from one type of poor decision-making to another, while increasing total health spending. Additionally, my paper underscores the role of behavioral economics in structural models assessing the quality of consumer choices. I show that including factors such as belief discounting may help to explain why overcoming information frictions is not simply a matter of increased access to health information.

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 5. The model output informs several counterfactual analyses assessing the role of consumer responsiveness to information, which I present in Section 6. Finally, I discuss the relevance of my findings and directions for future work in Section 7.

2 Empirical Setting & Data

My primary data on household plan choice, health utilization, and major medical events come from the IBM/Truven MarketScan *Commercial Claims and Encounters Data*[®]. These data contain detailed inpatient, outpatient, and pharmaceutical claims for a sample of households enrolled in ESI through large U.S. firms which contracted with participating payers. Each observation includes diagnostic, procedural, and payment information, as well as household, firm, and insurance plan identifiers. I obtained data from 2006 to 2018, with the exception

of plan identifiers which are only available until 2013. Throughout, spending data has been normalized to 2020 USD using the CPI-U series.

My final sample includes households with two or more members observed for two or more years, each of which is employed by (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.

Table 1 presents summary statistics for the full sample as well as the subset of the sample with insurance plan identifiers. It is important to ensure that the two samples are relatively balanced given that I use only the plan-identified sample in my structural estimation (Section 4). In general, the two groups have similar demographics, spending trends, and health states. A notable exception is that households in the plan-identified sample incur lower OOP costs than the full sample, suggesting that they possess more generous insurance coverage on average. However, this is likely due to time trends arising from the fact that the plan-identified sample runs only through 2013. Medical spending, as expected, is highly skewed, with average annual household spending in the range of \$2,500 compared to a median of about \$400. Observed switches in plan choices are low, consistent with prior work (Handel, 2013).

2.1 Major medical events

I model the ways households respond to information about their health risk communicated through major health events within the family. I identify these events based on observed diagnostic codes in the claims data, using a subset of the Department of Health and Human Services' Hierarchical Condition Categories (HCCs). These HCCs, which are typically used in risk adjustment models, identify a basic set of chronic illnesses that may alter overall health utilization and spending. I limit my classification of health events to non-pregnancy HCCs that occur with high frequency as discussed in Appendix A.2.

To ensure that I identify new diagnoses, I require that relevant diagnosis codes appear during or after an individual's second observed year. Additionally, I drop households for which the diagnosed individual is not present for at least a full year after their medical event to exclude individuals who might have passed away during or shortly after their event.

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 a physician to identify a set of disease-specific procedures and prescriptions associated with each health condition in

	Full Sample	Plan-Identified Sample
Panel A: Household demographics		
Family size	3.0 (0.00)	3.0 (0.00)
Employee age	45.0 (0.01)	44.4 (0.01)
Enrollee age	30.9 (0.01)	30.4 (0.01)
% female employees	41.6 (0.00)	42.4 (0.00)
% female enrollees	50.2 (0.00)	50.3 (0.00)
Panel B: Medical spending & plan choices		
Total medical spending	\$2,504.41 [\$679.75] (4.51)	\$2,454.88 [\$624.16] (7.12)
OOP medical spending	\$443.07 [\$109.66] (0.53)	\$337.98 [\$80.33] (0.89)
% individuals w/ zero spending	15.4 (0.00)	16.6 (0.00)
% individuals w/ zero OOP	21.0 (0.00)	22.2 (0.00)
% switching plans	—	5.3 (0.00)
Panel C: Major medical events		
% experiencing chronic diagnosis	6.3 (0.00)	5.2 (0.00)
% experiencing acute event	1.0 (0.00)	0.6 (0.00)
Diagnosis OOP, chronic	\$1,082.05 [\$464.69] (11.59)	\$854.62 [\$329.90] (17.72)
Diagnosis OOP, acute	\$2,494.42 [\$1,419.91] (68.05)	\$2,107.09 [\$964.62] (122.50)
Recurring OOP, chronic	\$983.03 [\$521.39] (17.32)	\$683.60 [\$446.69] (19.20)
Years	2006–2018	2006–2013
N_{families}	353,403	179,044
$N_{\text{individuals}}$	1,087,353	555,733

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.

Table 1. Household Summary Statistics

my sample.⁵ 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 \$683.60 (\$446.69) out-of-pocket on recurring costs needed to care for chronic conditions.

2.2 Plan characteristics.

Heterogeneity in each household’s choice of plans provides a plausibly exogenous source of variation in how major medical events and chronic health costs impact household spending decisions. I exploit the claims data to estimate the characteristics of each plan in my households’ choice sets, which will be important inputs in my theoretical model.

I define a household’s plan choice set at the firm-state-year level, and limit attention to plans covering at least five percent of all covered lives within a firm-year to rule out executive plans.⁶ In reality, health plans are defined by a complicated set of cost-sharing measures, including copayment and coinsurance rates that vary widely across provider specializations, networks, and procedures. For tractability, my structural model takes in a simplified version of these measures: a family deductible, a simplified non-specialist coinsurance rate, and a family OOP maximum. I construct measures for each plan’s individual and family deductibles based on the empirical distribution of payments in the claims data (Zhang et al., 2018). I then estimate the other two cost-sharing parameters as those that minimize the sum of squared residuals between predicted and observed OOP spending for households within each plan year (Marone and Sabety, 2020). Appendix A.1 describes this methodology in more detail and evaluates the quality of these inferences. I find that these simplified measures capture a wide degree of variation in my data as well as matching well with measures used in earlier work. Finally, I estimate each plan-year’s family premium as the average cost of all households enrolled in the plan over a year, and assume that employee premium contributions are consistent with the national averages for household coverage, on average about 28% of the household premium (Foundation, 2020).

There is substantial variation across firms, regions, and years in the generosity of coverage offered to employees, which I describe in Table 2. As I describe in Section 4.3.2, such variation provides an intuitively useful means of attributing household behaviors to changes in risk *preferences* versus risk *beliefs*; households who are more risk averse tend to minimize their

⁵Appendix A.3 lists the relevant codes used for each diagnosis.

⁶My data does not distinguish whether there exist plan “tiers” within firms (for example, a university that offers one set of plans to its faculty and a different set to its graduate students). These unobserved barriers may cause measurement error in the plan choice sets used in the structural model in Section 4; however, such error would not affect any of my primary results, which focus on how new health information alters spending choices conditional on the choice of plan.

overall variation in *ex-ante* expenditures by choosing more generous health plans, while households who are less risk averse but believe they are at higher risk for major health events may choose less-generous plans overall that instead provide more targeted coverage. The average household has between 2 and 4 plans to choose from in a given year, with a wide degree of variation in the average family deductible. This variation is comprised of both heterogeneity in the frequency with which firms offer zero-deductible health plans as well as in the size of nonzero deductibles. Similar variations exist in other plan characteristics, including copayment rates and OOP maxima.

	Firm							
	A	B	C	D	E	F	G	H
# of plans offered	3.5	2.5	3.0	2.0	2.0	2.6	2.8	3.0
Family premium (\$000s)	12.7	9.8	9.7	10.2	9.3	8.9	9.1	11.5
Family deductible (\$000s)	0.4	0.4	2.1	1.0	1.0	0.7	0.9	0.5
% of 0-deductible plans	64.3	46.7	0.0	0.0	0.0	22.2	31.8	38.9
Family OOP max. (\$000s)	3.5	4.6	5.1	5.9	4.3	4.1	5.2	3.9
HHI of all plans	0.4	0.6	0.4	0.6	0.9	0.6	0.7	0.4

Notes: Averages are pooled across all plans and years in a given firm.

Table 2. Average Plan Characteristics, 2006–2013

3 Spillover Effects of Household Health Events

This section presents my main reduced-form empirical results. I first show that after experiencing a chronic major health event, households increase their overall medical utilization by about 10% annually, as well as increasing their investment in billed spending on preventive care. I illustrate that the observed responses are consistent with a reevaluation of one’s own risk by showing that households are more likely to invest in preventive care that is specific to the illness their family member experienced. I then consider other potential mechanisms, including financial incentives, salience effects, and general learning about the health care system. Finally, I show that household members increase their utilization of “pseudo-preventive” low-value services—such as extraneous screenings and imaging services—showing that while health events generate strong spending responses, these responses are not necessarily targeted at high-return services.

3.1 Induced Spending Changes

To estimate the causal impact of health shocks on health choices, I first estimate two-way fixed effects “event study” regressions of the following form:

$$\sinh^{-1}(y)_{ft} = \alpha_f + \tau_t + \sum_{k=-T}^T \gamma_k \mathbb{1}\{t - E_{ft} = k\} + \epsilon_{ft}. \quad (1)$$

The variable y_{ft} represents a spending outcome for a household f in year t ; in my main specification, this outcome is annual OOP payments made by all family members *except* those who experience the major health event. I adjust for highly-skewed distributions of spending variables by using the inverse hyperbolic sine transformation.⁷ An added advantage of this transformation is that the resulting regression coefficients can be interpreted as approximate percentage changes in the outcome variable, relative to the year prior to the shock. I include household and year fixed effects, as well as dummy variables indicating when an observation occurred relative to E_{ft} , a household’s event year.⁸ The coefficients on these indicator variables, $\{\gamma_k\}$, are the coefficients of interest. I also adjust for potentially correlated responses within a household by clustering standard errors at the household level.

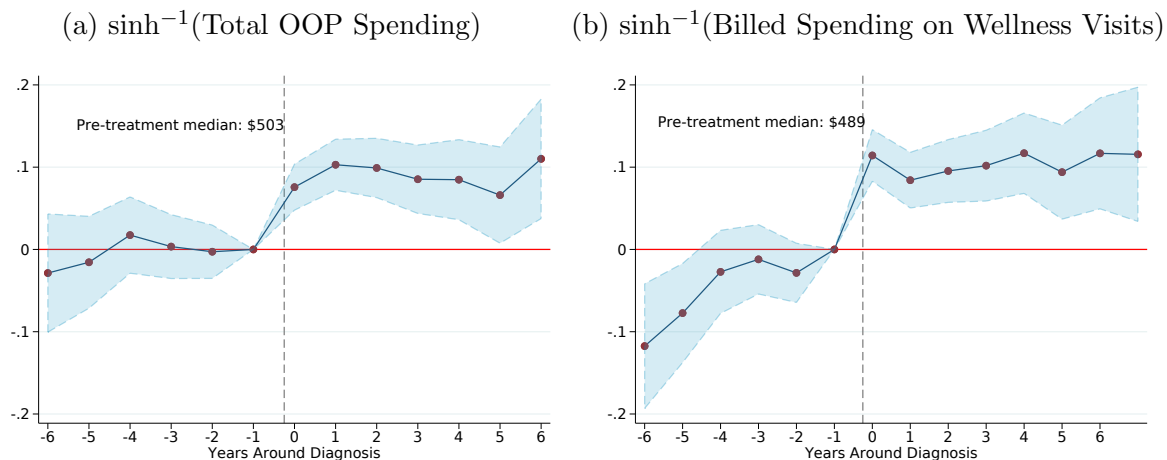
This approach allows me to identify the potentially time-varying effects of health shocks—which might have decaying influence on household choices over time—while simultaneously controlling for any unobserved household- or year-specific deviations in behavior. However, recent work has highlighted that two-way fixed-effects estimators can be difficult to interpret without strong modeling assumptions (Imai and Kim, 2020). In particular, the presence of heterogeneous treatment effects may obscure interpretation of both pre-trends and estimated time-varying treatment effects (Sun and Abraham, 2020; de Chaisemartin and D’Haultfoeuille, 2019; Goodman-Bacon, 2018). In addition to the traditional event study results shown here, I present a number of additional specifications in B.6, including simple recentered time series graphs, standard difference-in-differences regression coefficients, and the robust alternative estimator proposed by de Chaisemartin and D’Haultfoeuille (2019). Taken together, these results provide a consistent picture of a strong household response to health shocks.

Figure 1 presents the time-varying causal effect of a health shock on household OOP

⁷I use the inverse hyperbolic sine transformation to accommodate the approximately 15% of individual-years in my data with 0 spending (Harris and Stöcker, 1998). Bellemare and Wichman (2020) show that for a model with continuous variables x and y and specification $\sinh^{-1}(y) = \beta x + \varepsilon$, the elasticity of y with respect to x is $(\beta x / y) \sqrt{y^2 + 1} \approx \beta x$ whenever $y \geq 2$. Bellemare and Wichman (2020) also discuss the ways using this measure may refine estimates using the more common $\log(y + 1)$ transformation. I show in Appendix B.6 that my results are not substantively altered when using the logarithm transformation.

⁸Important for this approach, I utilize a large control group in my sample, allowing me to separately identify the time-varying treatment effects from yearly fixed effects (Borusyak and Jaravel, 2016).

Figure 1. Effect of Chronic Diagnoses on Non-Diagnosed Household Members' Spending



Notes: These figures show estimated coefficients and 95% confidence intervals for the effect of a new chronic diagnosis on medical spending. In both panels, the sample includes spending for all household members without major health events. In panel (a), the dependent variable is the inverse hyperbolic sine of total OOP spending; panel (b) estimates the effect on total spending (insurer spending + OOP spending) on wellness visits only. Spending is measured in 2020 USD. Standard errors are clustered at the household level.

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 prior to 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 for at least five to seven years post-diagnosis. Additional results in Appendix B.6 corroborate this finding with other outcome variables including total billed spending or visit frequencies.

Importantly, this increase in utilization encompasses an increased investment in preventive care. The second panel of Figure 1 illustrates this by limiting the scope of the analysis to household spending only on wellness visits. Wellness visits are non-problem-based visits with a family or primary care physician that are generally recommended about once a year; these visits include routine screenings for important chronic conditions including cancers and mental health conditions. These visits constitute an important jumping-off point for the use of other preventive services (Jiang et al., 2018) and are therefore generally considered to be an important form of high-value care (Tong et al., 2021). Here, too, I find that new diagnoses in a household are associated with strong responses. Affected, non-diagnosed household members increase their overall spending on wellness visits by about 10%, matching the increase in overall utilization.⁹

⁹Even before the ACA's cost-sharing exclusion took effect in 2010 (or 2012 for certain women's health services), OOP costs for preventive care were steadily declining for those with ESI (Hong et al., 2017). Once the ACA took effect, the majority of wellness visits should be free to enrollees in my sample (Shafer et al.,

3.2 Changes as Responses to New Health Risk Information

These results suggest a profound and persistent change in how non-diagnosed household members engage with the health care system. I first show that these responses are indicative of household members updating their beliefs about their own health risks following the receipt of health information from a major event. Such observed responses could also be driven by factors beyond changes in a household’s assessment of their health risks, including changes in the price of care, salience effects, overall exposure to the health care system, or improved physician relationships (Sabety, 2020), ; I explore these potential mechanisms in more detail in the following section.

To more explicitly explore the link between major health events and risk beliefs, I estimate the causal effects of health shocks on preventive services that are specific to an affected household’s diagnosis. Here, the intuition I rely on is that household exposure to risk information is more targeted than other forms of health information; hence, the extent to which I observe households selecting into preventive services that are disease-specific rather than general provides evidence of responses specifically to new risk information.

For example, individuals who have learned that they are at increased risk for developing diabetes may have a higher likelihood of seeking out screenings for abnormal blood glucose levels than individuals who have learned that they are at increased risk for another chronic condition. Figure 2 plots re-centered time series that depict the associations between household diagnoses and the takeup of diabetes screenings for children within a household.¹⁰ The figure plots average utilization rates of diabetes screenings among household members under 18 years old for two groups: those who are exposed to a diabetes diagnosis in their home and those who are exposed to a different chronic diagnosis. The horizontal dashed line indicates the average utilization of these screenings in unaffected households. Children whose family members are diagnosed with conditions other than diabetes do not appear to significantly alter their screening behaviors; on the other hand, siblings of those diagnosed with diabetes increase screenings following the diagnosis, being over twice as likely to be screened for diabetes as other children.¹¹

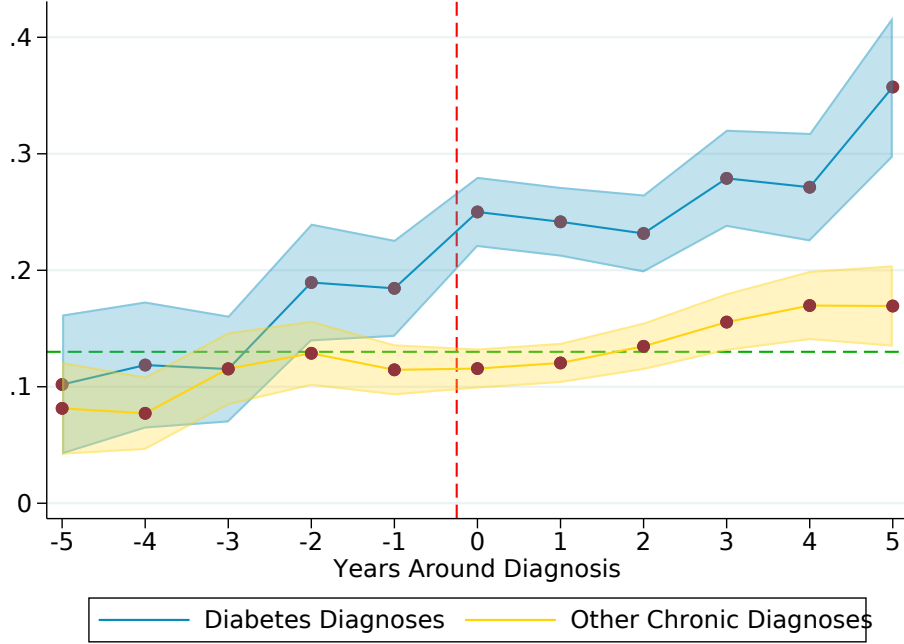
2021), a feature I observe in the data. Although time fixed effects in the regression specification should absorb these trends for both pre- and post-ACA trends, I use billed spending rather than OOP spending as my outcome variable of interest. Note that in my data set, billed spending represents the sum of individual OOP payments and insurer payments to the provider; it does not reflect any price negotiations or other discounts that were provided at the time of service, and therefore does not reflect the listed prices of services.

¹⁰I focus on the diagnoses and screenings of children to highlight the transmission of genetic risk information associated with Type 1 Diabetes Mellitus diagnoses. Similar results hold for the screenings of spouses following a Type 2 Diabetes diagnosis, which transmits health risk information more about lifestyle choices than genetic risks.

¹¹Note that the figure suggests significantly different screening behaviors in the year prior to a family member’s diagnosis. Although these differences are smaller, such pre-event differences could be the result of

Figure 2. Diabetes Diagnoses are Associated with Higher Rates of Diabetes Screenings

(a) Diabetes Screenings (Rate/1,000 Children)



Notes: Figure shows average utilization rates of diabetes screenings for non-diagnosed household members 18 years of age and younger, measured in rates per 1,000 children. Point estimates and 95% confidence intervals are presented. The top (blue) line indicates average rates for households who experience a diabetes diagnosis, and the bottom (gold) line indicates rates for those affected by other chronic diagnoses. The horizontal green dashed line indicates the average utilization rate for all other households in the sample (who do not experience a diagnosis).

To assess the causal effect of multiple diagnoses simultaneously on the utilization of disease-specific preventive care, I use a triple differences approach. This approach disentangles two competing effects: those arising from experiencing any chronic illness (e.g., salience effects) and a disease-specific informational effect. 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:

$$Pr(\text{Screening})_{f,t} = \beta_{DD}(\text{post}_t \times \text{chronic}_f) + \beta_{DDD}(\text{post}_t \times \text{chronic}_f \times \mathbb{1}\{\text{chronic}_f = d\}) + \alpha_f + \tau_t + \varepsilon_{f,t}, \quad (2)$$

where chronic_f is a dummy variable indicating whether *any* chronic diagnosis occurred

households responding to early symptoms of individuals who have not yet received a diagnosis. For example, households may choose to screen all children in a home when one is displaying diabetes-like symptoms, thereby anticipating some form of the health risk information even before the event itself (the diagnosis) takes place.

within the household and $post_t$ indicates all periods including and after the diagnosis. The triple interaction variable includes an additional constraint that the chronic diagnosis $chronic_f$ match the specific diagnosis d (e.g., a diabetes diagnosis when the screening being evaluated is one for diabetes). Hence, β_{DD} identifies the effect of any chronic diagnosis on screening, while the triple interaction β_{DDD} identifies the effect for the specific diagnosis of interest relative to other diagnoses.¹² For example, using this approach I can estimate the impact of a diabetes diagnosis on diabetes screenings as $\beta_{DD} + \beta_{DDD}$, where β_{DD} indicates the impact of experiencing any chronic diagnosis in the household on diabetes screenings and β_{DDD} indicates the specific differential effect of a new diabetes diagnosis occurring in the household.

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 of interest is a screening for a specific service (e.g., diabetes), this approach estimates the effect of a corresponding diagnosis relative to all other diagnoses, for which the screening reveals no information. In this context, the identifying assumption for the triple differences approach is the same as the identifying assumption for the simpler difference-in-differences regressions: that spending differences between diagnosed and undiagnosed households would have evolved similarly over time in the absence of treatment.¹³

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.¹⁴

Finally, to verify my results, I estimate this model for screenings for which health events communicate little useful information, and hence are expected to change behavior little. This

¹²The 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.

¹³When 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 B.6 includes standard difference-in-differences regression results that corroborate the findings reported here.

¹⁴Given 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.

might be because a diagnosis doesn't require a doctor's visit to diagnose (e.g., obesity) or doesn't require preventive screening prior to seeking treatment (e.g., mental health conditions, such as major depressive disorder). Hence, observing a lack of response among these types of preventive services serves to underscore the role that health information, specifically, plays in altering individual behavior. I include "placebo" regressions for the effect of new diabetes diagnoses on obesity diagnoses and the effect of new mental health disorder diagnoses on screenings for depression. Just as observing

Table 3 presents the estimation results from these six 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 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%. Similar to previous work, I 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.

The second panel of Table 3 reports results for placebo regressions including obesity diagnoses and depression screenings. Here, I find neither strong evidence that health events increase disease-specific screenings nor crowd out unrelated ones. This is consistent with the notion that individuals respond by altering their use of preventive care only when the major health event communicates health risk information that necessitates preventive care utilization. Other dimensions of a health event—such as learning about the role of preventive care in medical maintenance overall—do not appear to drive individual behavior changes, at least in the use of preventive services.

I report additional results in Appendix B.6. I find that in addition to selecting screenings based on the health risk information they receive, households are selective in which members they choose to screen. I utilize variation in intrafamilial relationships and corresponding risk to show that households screen those who are most affected by the new health information. 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

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

Notes: Table presents results from six triple-difference regressions highlighting the role of household investments in disease-specific preventive care following adverse health events. Each regression uses as its outcome variable a binary indicator for the screening listed in the first column, and a binary indicator for the event in the second column as its treatment variable (see Equation 2 for the full specification). Regression coefficients for the typical difference-in-difference effect (β_{DD}) indicate the effect of any chronic health event on screenings; the triple differences coefficients (β_{DDD}) indicate the effect of the specific diagnosis on screening choices. Robust standard errors clustered at the household level shown in parentheses. ¹ Due to unavailability/low-use of CPT-4 procedure codes for screenings, these outcomes are measured as new ICD-9-CM/ICD-10-CM diagnosis codes. ² Here, the reference group is all acute major health events. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 3. Effect of Chronic Diagnoses on Take-Up of Disease-Specific Preventive Care

screenings for spouses. Taken together, the observed ways in which major health events affect the use of preventive care are all consistent with a model where households interpret new diagnoses as signals of their own health risk, altering their behaviors accordingly.

3.3 Alternative Explanations for Spending Changes

Although individuals appear highly responsive to new information about their own risk, as discussed in the previous section, additional factors could separately cause or exacerbate observed changes in health spending. These factors include: induced demand responses from changes to the conditional price of medical services (moral hazard effects); salience effects arising from experiencing the trauma of major health events; learning about how to utilize the health care system, including the role of insurers and physician relationship building. In this section, I explore each of these potential competing explanations and show that they are each insufficient to explain my observed results.

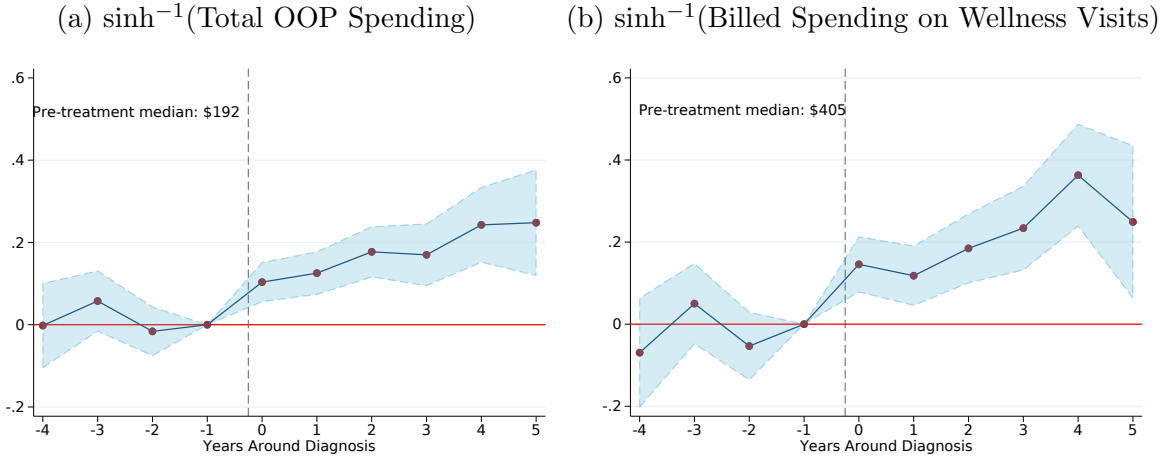
3.3.1 Moral Hazard

A natural response to observing the phenomenon illustrated in Figure 1 is to conclude that the spending increase is driven by moral hazard responses among the non-diagnosed individuals. Here, the intuition is that 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, shift the cost-sharing characteristics of a health plan for the rest of the household, effectively lowering their spot price of future (non-chronic) health care.

Two features of my results suggest that these induced demand responses are unlikely to be the principal driver of my 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 diagnosis 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 the case, either for overall utilization or the use of wellness visits specifically. Second, Figure 3 illustrates that non-diagnosed individuals respond to health shocks even when those shocks do little to change their spot price of medical care. Were moral hazard responses the principal mechanism of response, households in these plans would have much weaker incentives to adjust their choices.¹⁵

¹⁵A corresponding result for the subset plans with nonzero deductibles is included in Appendix B.6. Additional results included in Appendix B.6 show that families who are closer to meeting their deductibles prior to a health event are not more likely to increase their spending than those for whom chronic care costs

Figure 3. Effect of Chronic Diagnoses on Spending: Households Facing Zero Deductible



Notes: These figures show estimated coefficients and 95% confidence intervals for the effect of a new chronic diagnosis on medical spending. This figure uses a limited sample of only households enrolled in health insurance plans with zero deductible at the time of the event. In both panels, the sample includes spending for all household members without major health events. In panel (a), the dependent variable is the inverse hyperbolic sine of total OOP spending; panel (b) estimates the effect on total spending (insurer spending + OOP spending) on wellness visits only. Spending is measured in 2020 USD. Standard errors are clustered at the household level.

3.3.2 Salience Effects

It may also be the case that the intensity of major health events realigns household preferences to prioritize medical care. That is, rather than altering individual *beliefs* about health risks, it is possible that health events alter an household's risk *preferences* by affecting their marginal utility of medical care relative to other forms of consumption. While these salience effects almost certainly play some rule in future household decisions, I find that households alter their spending patterns only in response to certain types of major health events. In particular, I examine household responses to acute hospitalizations, such as heart attacks, and find that household responses to events are estimated to be both smaller and noisier (Appendix Figure 14). However, these hospitalizations make health care at least as salient—if not more so—than chronic diagnoses. This suggests that changes in risk preferences arising from a “health scare” are insufficient to entirely explain changes in behavior.

3.3.3 Health Information

New diagnoses may also alter spending patterns by providing families with more general health information, such as information about the value of medical care, the process of obtaining covered care through an insurer, or how to establish strong provider relationships.

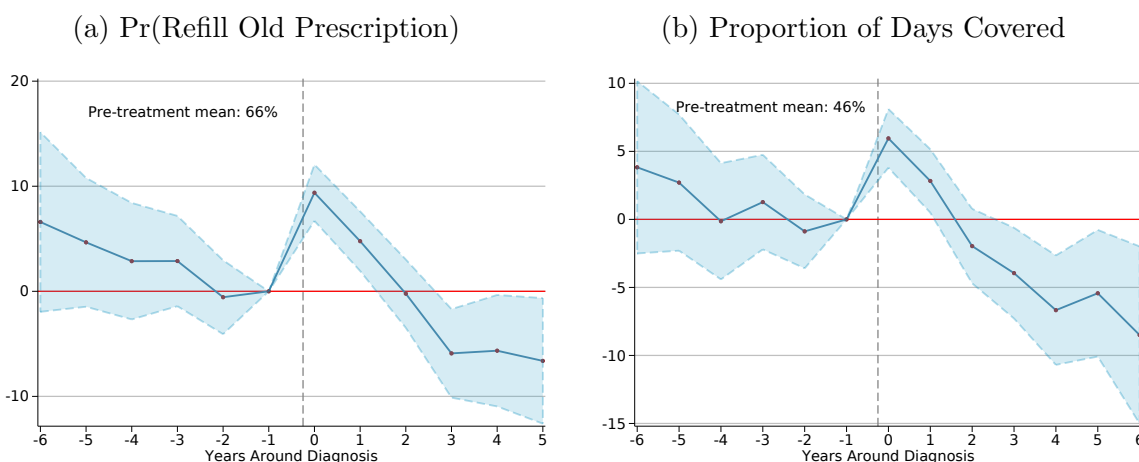
may not meaningfully change family cost-sharing rates.

Generally, learning about health risk and this more systematic learning imply similar responses among affected individuals, making their effects difficult to disentangle.

I focus on a particular case where new diagnoses provide risk information without more systematic information: non-diagnosed household members who were taking medications to prevent cardiovascular disease *prior* to the diagnosis within their family. Cardiovascular preventive drugs, including statins and other cholesterol-lowering drugs, are an extremely common class of medications and are known to be effective in preventing future health problems when used appropriately (O'Connor, 2006).¹⁶ In this analysis, I limit my sample to those who have filled a prescription for these medications at least once per year during their first two years in the sample. I then measure the effects of chronic diagnoses on utilization and adherence among refills of these prescriptions.

This setting provides a unique environment in which to disentangle the effects of general learning about health systems and learning about one's own health risk. Individuals with existing prescriptions already have sufficient knowledge about the health care system to receive this care from their provider and insurer. Hence, while major health events provide them with information about the potential value of adherence to their medication (along with the potential consequences for not doing so), these events are unlikely to provide new knowledge about how to obtain this medication.

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



Notes: These figures show estimated coefficients and 95% confidence intervals for the effect of a new diagnosis on adherence to preventive medications whose prescriptions were first written prior to the major health event. The sample is limited to all non-diagnosed individuals who filled preventive cardiovascular medications at least once per year during their first two years in the sample. In the first panel, the dependent variables is a binary indicator for whether the prescription was refilled at all. The second panel uses the proportion of days covered by any preventive cardiovascular medication as the outcome variable (Choudhry et al., 2009). Standard errors are clustered at the household level.

¹⁶Appendix Table 8 contains a detailed list of the therapeutic classes used in my sample.

I show, however, that new diagnoses alter adherence to these prescriptions. I estimate the effect of a chronic diagnosis on both the likelihood of any use of the medication and general adherence, measured using the proportion of days covered in a year (Choudhry et al., 2009). This measure is standard in the literature on drug 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. One concern in identifying the causal effect of new diagnoses on adherence is that prescription adherence may decay over time in response to barriers such as financial concerns or apathy (Slejko et al., 2014). Importantly, this decay may occur at different rates for different individuals both within and across households, meaning that such downward trends would not be accounted for using only household and year fixed effects. I therefore add a variable controlling for the number of years an individual has been in the sample to Equation 1.

Figure 4 presents the estimated dynamic treatment effect of a new chronic diagnosis on adherence to existing preventive prescriptions. 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 both the likelihood that individuals will fill their prescriptions at all and the proportion of days covered: affected individuals are around ten percentage points more likely to refill their prescription in the year of a major medical event than in the year before, translating to an additional eight percentage point increase in the average proportion of the year for which they are covered by the prescription. The fact that new diagnoses change individual adherence to prescriptions even among a population which has access to and knowledge of specific preventive care illustrates that individuals are learning about more than just how to obtain care. The estimated causal “re-adherence” to prescriptions is consistent with individuals reevaluating the value of their medication given new information about their health risks.

3.4 Quality of Induced Spending Changes

Major health events generate strong spillover effects within a household on both overall utilization patterns and preventive care investments. It is natural, therefore, to ask how these responses are distributed within a larger framework of health spending. Do major health events contribute to more informed decisions about the type of care consumers choose to utilize? Or does the salience associated with health trauma lead to further over-utilization of low-return services? I address these questions by examining household use of services typically deemed as “low-value” by medical professionals and health officials (Chua et al., 2016;

Colla et al., 2015).¹⁷ Low-value services include both services whose cost typically outweighs any benefits to an average patient (e.g., unnecessary surgeries such as arthroscopies) as well as services which are chronically over utilized in ways that dramatically lower their return (e.g., imaging services such as MRI services for chronic migraines). Avoiding the use of these services can result in an overall higher quality of health care through both cost reductions and the avoidance of unnecessary risks.

I find that new chronic diagnoses are associated with a small increase in overall low-value spending of about 5 percent (Appendix B.6, Figure 16). However, these results mask significant heterogeneity across different types of low-value services. Low-value services may differ in their perceived value to an affected household depending on the ways in which health events induce behavior changes. For example, if a chronic diagnosis communicates new risk information to a household, they may find low-value screening services—such as imaging services and preoperative visits—to be more attractive. On the other hand, households that respond to the price effects induced by a major health event may be more likely to seek out high-cost, low-return services such as elective surgeries. To explore these differences in-depth, I separate my sample of low-value services into five categories: pediatric services, including 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, including for lower-back pain; extraneous screening services for adults, including cardiac testing before low-risk surgeries; and adult surgical procedures, such as arthroscopy for knee pain.

<i>Service Category</i>	All Pediatric	Adult Drugs	Adult Imaging	Adult Screening	Adult Surgery
$\text{Post}_t \times \text{Diagnosis}_f$	0.05* (0.017)	-0.00 (0.000)	0.03*** (0.013)	0.10*** (0.014)	-0.10*** (0.012)
Adjusted R^2	0.192	0.143	0.123	0.163	0.230
N	1,538,161	1,538,161	1,538,161	1,538,161	1,538,161

Notes: Table shows estimated difference-in-difference regression coefficients for the effect of a new chronic diagnosis. Outcome variables are the inverse hyperbolic sine of billed spending in each category. See Appendix A.5 for service definitions. Spending is measured in 2020 USD. Standard errors clustered at the household level are reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table 4. Estimated Effects of Chronic Illness on Low-Value Care Utilization, by Category

Table 4 presents results estimating the effect of a new chronic diagnosis in each of the five

¹⁷These 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). Appendix A.5 contains more detail about the specific services included in each measure.

categories using a standard difference-in-differences framework (event study regressions are included in Appendix B.6). New chronic diagnoses shift households spending and utilization into low-value service categories comprised of screening services, pediatric care, and imaging services. The effect sizes range from an increase as large as ten percent for low-value screenings to three percent for imaging services.¹⁸ I find no effect on the misuse of prescription drugs among adults.

These results suggest that households seek out care that they see as useful in preventing or identifying future illness, even if those services are generally understood by health professionals as being low return. 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. This provides new suggestive evidence that the utilization of low-value care may be tied more to risk beliefs rather than ignorance about the actual returns of a service, providing similar evidence to recent work (Finkelstein et al., 2021).

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, in Appendix B.6. In general, I do not find that major health events prompt households to switch their health insurance plans. While new diagnoses in a household are associated with marked differences in observed spending behavior, it is still unclear whether these choices are *ex-post* more optimal for affected households. This motivates a more structural approach to quantify the welfare effects of health information.

4 Empirical Model of Belief Formation

In this section, I estimate the impact of health risk information on consumer choices as well as its implied welfare effects in a structural model of health utilization. I build on a canonical two-stage model of health spending (Cardon and Hendel, 2001). In the first stage, households choose an insurance plan to maximize their *ex-ante* expected utility, based on their available information about the distributions of future shocks. In the second stage, individuals within the household choose their spending and utilization based on realized health shocks and their chosen health plan’s features.

I extend the existing model in two important ways. First, I allow consumers’ types

¹⁸The results also provide preliminary evidence that major health events provide a deterrent from low-value elective surgeries; however, Appendix Table 14 highlights the strong presence of pre-trends in these models, which obfuscates the true causal effect of the diagnosis.

to be adaptive in response to health experiences. In my model, individuals learn about their probability of adverse health events; in addition, health events may alter household risk aversion to capture potential salience effects. Second, I explicitly model the differences between acute and chronic health shocks, as chronic health shocks impose recurring costs on a family, thereby altering conditional OOP prices for non-chronic care and inducing moral hazard effects within a household.

4.1 Model Primitives

Consider a household f comprised of individuals $i \in \mathcal{I}_f$. Individuals belong to one of two types—those without chronic illnesses, and those with at least one chronic condition. I assume state-dependent preferences, so that the utility of receiving medical care differs across these types. Households and individuals are characterized by three main variables: individual beliefs about health risks (p_{it}), household risk aversion (ψ_{ft}), and the distributions of their health shocks (discussed below). New health events—including both new chronic diagnoses and acute hospitalizations—cause both non-diagnosed and diagnosed individuals to update their beliefs about their health risks, as well as potentially altering household risk aversion and OOP prices.

In each period, two types of shocks are realized. Following typical convention, each individual has an acute health realization λ_{it} drawn from an individual-specific distribution $F_{\lambda_{it}}(\cdot)$. Acute health realizations model the uncertain aspect of demand for healthcare, with individuals with higher λ_{it} being sicker and hence demanding greater healthcare consumption.¹⁹ Second, households in each period receive a chronic health shock, m_{ft}^{CH} . For households without a chronic illness in the family, this amounts to the expected cost of a new diagnosis. For households living with chronic conditions, these shocks are the health costs associated with maintaining health for those affected by the conditions.²⁰

4.2 Model Stages

In each period, families first choose their insurance coverage; second, acute and chronic health shocks are realized; finally, individuals choose their yearly health spending. These

¹⁹Rather than simply having families draw their health expenditure m_i following a plan choice (Handel, 2013; Layton, 2017), I explicitly model these health shocks in order to separately identify how spending choices are reflective of beliefs about major health events, as well as to estimate the effects financial distortions caused by health events contribute to moral hazard in spending.

²⁰Many versions of this model incorporate heterogeneity in individual demand elasticities in order to accommodate heterogeneity in moral hazard effects (Einav et al., 2013; Marone and Sabety, 2020). As my model is concerned with disentangling only moral hazard events induced by major health events, I restrict the demand elasticity parameter ω in my model to be homogeneous across individuals and periods.

choices are static, in the sense that both households choose plans and individuals make spending decisions on the basis of the current period's utility and type parameters only, (including their beliefs about health risks). Finally, after each period's choices have been made, individual and household type parameters, including beliefs and risk aversion, are updated in a Bayesian framework. I first present the static choices in reverse order and then discuss how type parameters are updated.

4.2.1 Utilization Choice

After choosing a health plan $j \in \mathcal{J}$ and realizing acute and chronic health shocks $(\lambda_{it}, m_{ft}^{\text{CH}})$, individuals choose their optimal level of spending on non-chronic medical care, m_{it}^* . As is typical for these models, individuals trade off health production and wealth. In my extension of the model, individuals face residual uncertainty as to the likelihood of their own major medical events.²¹ I therefore assume that they choose m_{it} in order to maximize their expected utility over states:

$$m_{it}^* \equiv \operatorname{argmax}_{m_{it}} EU(m_{it}; p_{it}) = p_{it}u_{it,C} + (1 - p_{it})u_{it,H}, \quad (3)$$

where $u_{it,C}$ and $u_{it,H}$ represent individual utilities when diagnosed with a chronic illness and when not diagnosed, respectively. Note that Equation 3 nests the case where an individual has already been diagnosed with a chronic illness, in which case their transition probability is 1. I assume that each individual's utility function is separable in health and wealth for both chronic and healthy individuals:

$$u_{it,H}(m_{it}; \lambda_{it}, m_{ft}^{\text{CH}}) = h_1(m_{it}; \lambda_{it}, m_{ft}^{\text{CH}}) + y_{it}(m_{it}; m_{ft}^{\text{CH}}) + \varepsilon_1(m_{ft}^{\text{CH}}, \lambda_{it}) \quad (4)$$

$$u_{it,C}(m_{it}; \lambda_{it}, m_{ft}^{\text{CH}}) = h_2(m_{it}; \lambda_{it}, m_{ft}^{\text{CH}}) + g(m_{ft}^{\text{CH}}; \lambda_{it}) + y_{it}(m_{it}; m_{ft}^{\text{CH}}) + \varepsilon_2(m_{ft}^{\text{CH}}, \lambda_{it}). \quad (5)$$

The returns to medical spending $h_1(\cdot)$, $h_2(\cdot)$, and $g(\cdot)$ are assumed to be concave, so that within-year health fluctuations λ_{it} alter the optimal level of utilization m_{it}^* . Remaining annual income is denoted by $y_{it}(m_{it}; m_{ft}^{\text{CH}})$. $\varepsilon_1(\cdot)$ and $\varepsilon_2(\cdot)$ are preference shocks to capture unobserved changes in preferences due to major medical events.

I parameterize these utility functions as quadratic loss functions in the difference between medical spending and acute health status, in keeping with past work (Marone and Sabety,

²¹Although the value of chronic care costs are assumed to be made known to a household before they choose their non-chronic spending, the model abstracts away from the specific timing of individual costs within a year. Hence, even within a period, individuals have not learned whether they have a chronic illness, and hence maximize an expected utility across both states of the world. It is not until the end of the period that individuals know their true state and update their beliefs p_{it} .

2020; Einav et al., 2013), but allow for a potentially state-dependent utility function in which health status potentially alters the marginal utility of medical spending.²² Individuals without chronic conditions face the typical utility function:

$$u_{it,H}(m_{it}; \lambda_{it}, m_{ft}^{CH}, j) = (m_{it} - \lambda_{it}) - \frac{1}{2\omega}(m_{it} - \lambda_{it})^2 - c_j(m_{it}). \quad (6)$$

Here, $c_j(m_{it})$ represents the OOP costs associated with spending m_{it} , conditional on the choice of plan j . Hence, individuals choose medical spending to approximately match their acute health realization λ_{it} , accommodating the associated OOP costs of that spending.

On the other hand, individuals in the state of chronic illness face a utility function that depends on both acute and chronic health shocks, with potentially differing preference parameters. Their utility is given by:

$$u_{it,C}(m_{it}; \lambda_{it}, m_{ft}^{CH}, j) = (\alpha_1 m_{it} + \alpha_2 m_{ft}^{CH} - \lambda_{it}) - \frac{1}{2\omega}(\alpha_1 m_{it} + \alpha_2 m_{ft}^{CH} - \lambda_{it})^2 - c_j(m_{it}). \quad (7)$$

In this state, utility is derived from both chronic and non-chronic medical spending, each of which is potentially valued at a different rate than non-chronic medical spending for healthy individuals as indicated by the parameters (α_1, α_2) .

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 (see Appendix C.2 for details). 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:

$$m_{it}^* = \frac{1}{1 + p_{it}(\alpha_1 - 1)} (\lambda_{it} + \omega(1 + p_{it}(\alpha_1 - 1) - c'_j(m_{it}; m_{ft}^{CH})) - p_{it}\alpha_2 m_{ft}^{CH}). \quad (8)$$

The interpretation of Equation 8 elucidates the key insights associated with this state-dependent utility framework with separate chronic care costs. In this expansion of the model, individuals choose to consume less non-chronic health care as chronic care costs increase in value, either by increases in magnitude, marginal utility, or likelihood. As discussed in Bleichrodt and Eeckhoudt (2006), the extent to which households mismeasure p_{it} may artificially alter optimal spending decisions based on both the level of actual risks and the extent of the measurement error. Under the assumptions that households begin with p_{i0}

²²Previous work discuss and provide evidence for state-dependence in the utility of *non-medical* consumption (Finkelstein et al., 2013, 2009); this model introduces suggestive evidence for the state-dependence of non-chronic medical consumption as well.

close to zero, major health events could be associated with large (relative) increases in p_{it} , potentially explaining the dramatic and persistent shifts observed in Section 3.

Equation 8 also highlights the ways that chronic care costs affect spending decisions through prices. The OOP cost function $c_j(m_{it}; m_{ft}^{\text{CH}})$ is assumed to account for the price of chronic care first in the timing of health spending, before any other non-chronic spending. This anticipation of chronic care costs shifts the boundaries between optimal spending solutions by depressing the rate at which discretionary medical spending translates into OOP costs. This is the method by which moral hazard effects arise from major health events.

4.2.2 Plan Choice

In the first stage of the model, households choose an insurance plan to maximize their *ex-ante* expected utilities without knowing their realization of individual health shocks λ_{it} or major health costs m_{ft}^{CH} . This expected utility depends on the distributions of both health shocks as well as a household risk aversion parameter, which depends flexibly on household demographics and is allowed to evolve over time to capture the salience effects associated with health events, as discussed in Section 4.3.1. The household expected utility function for a given plan j is therefore:

$$U_{fjt} = - \sum_{i \in \mathcal{I}_f} \left[\int \int \frac{1}{\psi_{ft}(x_{ft})} \exp\{-\psi_{ft}(x_{ft})u_{it}^*\} dF_{\lambda_i} dG_{m^{\text{CH}}} \right] - c_j(m_{ft}^{\text{CH}}) - \pi_{fj} - \eta \mathbb{1}_{fj,t-1}, \quad (9)$$

where u_{it}^* represents the optimal payoff to individual i in period t given the realization of acute and chronic health states.²³ In addition to each individual's realized OOP costs for non-chronic medical spending, households face OOP costs for chronic care represented by $c_j(m_{ft}^{\text{CH}})$. Households also face plan premiums π_j and a perceived monetary cost η for switching plans ($\mathbb{1}_{fj,t-1}$ is an indicator for whether the family chose plan j in year $t-1$).²⁴

²³One concern with a utilitarian index here is that households may have little incentive to diversify their medical spending across household members. However, the choice of the utility function used in the second (spending) stage of the model makes it optimal for families to allocate care according to each individual's realization of λ_{it} ; hence, this modeling choice does not give rise to families allocating all of their care to a single individual. An alternative approach is to use a CES function for utilities; however, this introduces more nuisance parameters into the estimation framework. Finally, I assume that the von Neumann Morgenstern (vNM) utility index for this decision possesses a constant coefficient of absolute risk aversion, a common choice for these models as it implies no wealth effects.

²⁴I do not observe premiums or contributions in my data and therefore follow the methodology of Layton (2017). In particular, I assume that premiums are equal to the average cost among the employees with dependents enrolled in the plan during the prior year plus a fixed overhead cost, and then assume that employee contributions are 28% of that value (Foundation, 2020). Note that as Layton discusses, identification of the structural parameters in this model do not depend on accurate estimation of premiums, but rather require that the premium differential across firms is correct.

4.2.3 Parameter Updating

After households and individuals have made their plan and spending choices, type parameters evolve in response to health events. Of particular interest is the way that individuals update their beliefs about their unknown transition probability (p_{it}). Additionally, households update their risk aversion parameters (ψ_{ft}) according to an adaptive framework; I discuss this further in Section 4.3.1.

I model individual learning about health risks as a Bayesian updating process in response to health events. In particular, I assume that initial beliefs depend on individual demographics, including age, sex, health risk scores, and the presence of any pre-existing conditions within the household. Individuals' prior beliefs about their true risk probability are assumed to be normally distributed with mean and variance parameters ($\mu_{p,i,0}, \sigma_{p,i,0}^2$). The center of the distribution $\mu_{p,i,0}$ varies with individual demographics and is potentially correlated with other household type parameters.

Major health events provide individuals with signals y_{it} about the underlying distribution of p_{it} , I likewise assume that these signals are normally distributed, so that the mean and variance of an individual's posterior distribution has a closed-form solution in each period. Specifically, if $y_{it} \sim \mathcal{N}(\tilde{\mu}_{it}, \tilde{\sigma}_{it}^2)$, the evolution of the mean and variance parameters can be written as:

$$\sigma_{pi,t+1}^2 = \frac{\tilde{\sigma}_{it}^2 \sigma_{pi0}^2}{\tilde{\sigma}_{it}^2 + s_{it} \sigma_{pi0}^2} \quad (10)$$

$$\mu_{pi,t+1} = \frac{\tilde{\sigma}_{it}^2 \mu_{pit} + \sigma_{pit}^2 \tilde{\mu}_{it}}{\tilde{\sigma}_{it}^2 + \sigma_{pit}^2}, \quad (11)$$

where the variable s_{it} indicates how many health signals an individual has received by the end of period t .

An important potential difficulty when using a Bayesian framework with rare events is the choice of updating frequency. Given the relative rarity with which chronic health shocks occur, updating of probabilities after each period would result in posterior beliefs that are tightly centered around the initial mean, varying little with new information. In such a regime, individuals would have to perceive health shocks as being impossibly likely (e.g., $\tilde{\mu}_{it}$ much greater than 1) in order for health shocks to meaningfully change health beliefs. This is inconsistent with the analysis I have presented previously, which shows that individuals are highly responsive to chronic health shocks; in addition, I find that older individuals have stronger responses to these diagnoses than younger individuals (Appendix B.6). Were individuals continually updating their beliefs in years where adverse events did not occur, I would not observe such large responses or differentially larger responses among

older individuals.

I address this inconsistency in my preferred specification by assuming that households update their beliefs *conditional* on a health event occurring. This reduces the number of uninformative signals individuals process, and hence avoids problems of weight degeneracy, and is consistent with individuals who form beliefs about their health risk once, and then only revisit those beliefs once they have been called into question. Once the individual begins evaluating their health risk beliefs (e.g., after a diagnosis has occurred within the household), they do so in a completely standard way, including updating beliefs in all following years without major health events.

Such an approach is an intuitively appealing way to deal with the issue of Bayesian updating when signals are infrequent. However, my results are robust to alternative specifications, including (i) an adaptive learning framework where individual beliefs change linearly in each period with some dependence $\rho < 1$ on the previous period’s beliefs, and (ii) a more traditional setup where individuals update their beliefs with some probability $p > 0$ in the absence of health events.²⁵ Additional modeling possibilities include the use of quasi-Bayesian modeling where individuals disregard less salient signals, but still update beliefs in each period (Rabin, 2013), or where individuals over-weight “good news” relative to “bad news” (Eil and Rao, 2011).

4.3 Estimation

4.3.1 Parametrization

The unit of observation is a family f comprised of a set of individuals \mathcal{I}_f in year t . Each family faces a choice of plans that varies at the firm-year-state level.²⁶ Households are characterized by their unobserved type variables $\{p_{it}, \lambda_i, \psi_{ft}\}_{i \in \mathcal{I}_f}$. I allow the initial parameters $(p_{i,0}, \lambda_i, \psi_{f,0})$ to be arbitrarily correlated, and link them to observable data by assuming that they are drawn from a multivariate normal distribution which depends on observed demographics:

$$\begin{bmatrix} p_{i,0} \\ \mu_{\lambda,i} \\ \log(\psi_{f,0}) \end{bmatrix} \sim \mathcal{N} \left(\begin{bmatrix} \beta_p \mathbf{X}^p \\ \beta_\lambda \mathbf{X}^\lambda \\ \beta_\psi \mathbf{X}^\psi \end{bmatrix}, \begin{bmatrix} \sigma_p^2 & & \\ \sigma_{p,\lambda} & \sigma_\mu^2 & \\ \sigma_{p,\psi} & \sigma_{\lambda,\psi} & \sigma_\psi^2 \end{bmatrix} \right). \quad (12)$$

²⁵For a more in-depth review of the relative strengths and weaknesses of Bayesian or adaptive learning in structural modeling, see Aguirregabiria and Jeon (2020).

²⁶I ignore plans that have less than five percent of the overall firm-year market share in my data to avoid including executive health plans in employee choice sets.

Covariates \mathbf{X} include age, sex, health risk score, family size, and the presence of pre-existing conditions in a household. In practice, I use individuals' first year of data in \mathbf{X}^p and \mathbf{X}^λ and within-individual averages in \mathbf{X}^ψ .

Individual beliefs evolve in response to signals about their health risks as discussed in section 4.2.3. I assume that these signals y_{it} are normally distributed with variance σ_π^2 (to be estimated) and a mean given by the logit regression:

$$y_{it} = \pi_1 \mathbb{1}\{\text{chronic}\}_{f,-i} + \pi_2 \mathbb{1}\{\text{acute}\}_{f,-i} + \pi_3 \mathbb{1}\{\text{acute}\}_{f,i} + \pi_4 x_{it}, \quad (13)$$

where *chronic* and *acute* indicate the occurrence of chronic or acute health events within a household and x_{it} is a variable for the number of years that have passed since the earliest major health event in the family. Hence, π_1 is the main parameter of interest, identifying the effect of a household chronic diagnosis on individual beliefs. On the other hand the variance of the signals, σ_π^2 , reveals the magnitude of unobserved information affecting individual health risk probabilities.

To parameterize the distribution of acute health shocks, I assume that $F_\lambda(\cdot)$ is a shifted lognormal distribution. Assuming a lognormal distribution for λ is natural, as the distribution of annual health expenditures is highly skewed. The choice of shifting the distribution accommodates the approximately 15% of individuals in my sample who choose zero medical spending in a given year. I therefore assume that an individual's (correct) beliefs about their transient health shocks are given by

$$\ln(\lambda_{it} - \kappa_i) \sim \mathcal{N}(\mu_{\lambda,i}, \sigma_{\lambda,i}^2). \quad (14)$$

When κ_i sufficiently large (and negative), small and negative values of λ may lead to zero spending being the utility-maximizing solution for an individual.²⁷

Acute health shocks at the individual level are therefore summarized by three parameters: $(\mu_{\lambda,i}, \sigma_{\lambda,i}^2, \kappa_i)$. The parameter $\sigma_{\lambda,i}^2$ reflects the precision in an individual's beliefs about their transient health state. Both $\sigma_{\lambda,i}^2$ and κ_i are estimated as a linear projection on individual covariates (see Appendix C.2).

In contrast, I directly use empirical distributions of chronic care costs from my data in household expected utility. I assume that individuals have rational expectations over the distributions of their chronic health care costs, which change they experience major health

²⁷Previous work has allowed the distributions of these shocks to evolve over time (Marone and Sabety, 2020). In my model, which separates acute and chronic health shocks, such variation would amount to shifts in the need for non-chronic health spending, such as variation in an individual's anticipated office-visit spending from year to year. In addition to being of second-order concern to my setting, such variation seems indistinguishable from the random variation in the draws of λ_{it} already present.

events. This is a simplifying assumption employed for tractability, as my model already allows for the identification of rich heterogeneity governing individual expectations about health shocks. However, although there is evidence that consumers do not know the price of health care before they consume it (Lieber, 2017), this is less concerning with chronic care costs, which are typically stable over time and hence more easily predicted by household members. The empirical distributions are similarly assumed to be stable across years, but I use a separate distribution in the year of diagnosis to accommodate potentially higher costs in that year (e.g., for unexpected hospitalizations).

Finally, I allow family risk aversion ψ_{ft} to evolve over time as discussed above. In particular, $\psi_{ft}(x_t)$ evolves linearly according to:

$$\psi_{ft} = \gamma_0 \psi_{f,t-1} + \gamma_1 \{\text{Post}_t \times m_{f0}^{\text{CH}}\} + \gamma_2 \{\text{Post}_t \times c_j(m_{f0}^{\text{CH}})\} + \gamma_3 \{\text{Post}_t \times \text{Hosp}_{f0}\} + \zeta_{ft}, \quad (15)$$

where m_{f0}^{CH} represents the billed spending associated with the diagnostic event, $c_j(m_{f0}^{\text{CH}})$ the OOP spending of the diagnostic event, and Hosp_{f0} indicates whether a hospitalization occurred as part of the diagnosis. I assume that $\zeta_{ft} \sim \mathcal{N}(0, \sigma_\psi^2)$.

I denote the parameters of the model by θ . These parameters include the main parameters of interest $\vec{\pi}$ and $\vec{\psi}$, including the variances σ_π^2 and σ_ψ^2 . Additional parameters included in the estimation are the utility parameters $\alpha_1, \alpha_2, \omega$, and η ; the five vectors of mean shifters, $(\beta_p, \beta_\psi, \beta_\lambda, \beta_{\sigma_\lambda}, \beta_\kappa)$; seven variance and covariance parameters, $(\sigma_p, \sigma_\mu, \sigma_\psi, \sigma_\kappa, \sigma_{p,\psi}, \sigma_{p,\mu}, \sigma_{\psi,\mu})$; and the variance of the idiosyncratic shock term σ_ε^2 , which scales the choice probabilities. I assume that these idiosyncratic shocks follow the typical Type-1 Extreme Value distribution. Based on θ and the data, I am able to simulate values for $p_{it}, \mu_{\lambda,i}, \sigma_{\lambda,i}, \lambda_{it}$, and ψ_{ft} .

I estimate the model via maximum likelihood, following the approach laid out by Revelt and Train (1998) and Train (2009), with the appropriate adaptation for modeling a discrete choice followed by a continuous one (Dubin and McFadden, 1984). For a given household, likelihood functions are constructed as the density of their observed health spending conditional on their observed plan choices. The estimation is done in R version 4.0.3, following some of the best practices laid out in Conlon and Gortmaker (2020). I provide additional estimation details in Appendix D.4.

4.3.2 Identification & Interpretation

My model utilizes multiple sources of variation to separate multiple effects arising from major medical events. In addition to any changes in individual risk beliefs, health events may alter health behaviors by changing the price of non-chronic care, increasing the salience of health consumption, providing experiential learning about how to obtain high-quality health care,

or altering preferences for medical care in other ways. The critical challenge is that changes in risk preferences, salience, or systematic health learning may also increase the willingness to purchase insurance and utilize medical care.

I use a rich set of major health events that vary in their expected costs, both in the year of diagnosis and in following years. This variation in the expected costs needed to maintain health for someone with a chronic condition changes the extent to which a specific chronic condition significantly alters the expected prices for other, non-chronic medical care. This variation, coupled with variation in plan spending characteristics, allows me to separate moral hazard effects from other drivers of behavior.

To separate risk aversion from beliefs, I use variation in insurance plan characteristics and choice sets faced by different households in my data set. These choice sets vary at the firm-state-year level, and typically include plans with a wide range of cost-sharing parameters (Table 2). Under the assumption that risk aversion drives plan choice and not medical spending, and that households with high risk aversion seek to reduce the incidence of high OOP expenditures (Erikstrup et al., 2020), highly risk-averse households will gravitate towards the plans in their choice sets that most limit high expenses (e.g., low-deductible plans). Finally, I use data on the circumstances of major medical events—including the resulting costs and whether a hospitalization occurred—to incorporate the role of salience associated with health trauma in changing household risk aversion.

The principal estimated structural parameters of interest in my model are those governing the evolution of the transition probabilities p_{it} . Changes in these parameters that arise from new chronic diagnoses encompass both a reevaluation of individual health risk beliefs and other informational effects unaccounted for in the model, which may load onto this parameter. These effects include learning about the health care system more generally or forging better relationships with health care providers. Although section 3 suggests that these factors are not the principal mechanisms for responses, they may influence how p_{it} responds to new diagnoses. I therefore interpret changes in p_{it} as resulting from an aggregate informational effect, rather than from moral hazard or salience effects.²⁸

5 Structural Results

Table 5 presents the estimated parameters resulting from maximum likelihood estimation. Column 3 shows the preferred specification described in Section 4, while columns 1 and 2 present simplifications of the model that are useful both in building intuition and validating

²⁸Appendix C.2 discusses an alternative interpretation of p_{it} as a preference weighting across states rather than explicitly health beliefs.

the estimated parameters. Additional parameters not relevant to the welfare effects of health information—including incidental parameters such as switching costs and individual mean-shifting regression coefficients—can be found in Table 15 in Appendix D.4.

I consistently find strong effects on non-diagnosed beliefs associated with household chronic diagnoses. New chronic diagnoses are associated with an average increase in an individual’s belief of a major health event of 33 percentage points, an effect which is far larger than those estimated for acute events for either the individual or their family members, which are only estimated to increase risk beliefs by 5 and 6 percentage points, respectively. These increases are persistent, with little evidence that risk beliefs decrease over time (the estimated time trend coefficient is only 1 percentage point each year). The estimated variance for the unobserved dimension of belief changes is low (1.5 in the preferred specification), suggesting that observed events are dictating most of the changes in risk assessments.

Table 5 also presents parameters illustrating how the effects of new chronic illnesses alter behaviors in other meaningful ways. Major health events—both acute and chronic—are associated with strong salience effects that increase household risk aversion. On average, experiencing a major health event increases the coefficient of household risk aversion by 0.61, a 34.9% increase over the pre-diagnosis average coefficient of 1.75.²⁹ These effects are stronger when the household event entails either a higher amount of total billed spending or a hospitalization, suggesting that households respond differently to the intensity of an event. Panel B reports additional information regarding the distribution of household types and the value of incorporating the full richness of the model in rationalizing observed plan choices and spending. In particular, I estimate a high degree of variance in individual health risk beliefs (prior to any health event). These beliefs are weakly positively correlated with acute health status and negatively correlated with household risk aversion. These facts suggest that variation in individuals’ estimated beliefs reflects variation in individual health status, as expected. Finally, in the full version of the model, the variance of the idiosyncratic error term is small, suggesting that most of the observed variation in consumer behavior can be explained by heterogeneity in individual types, responses to major health events, or both.

Models 1 and 2 of Table 5 illustrate simplifications of the model that help validate the estimated parameters and build intuition. In Model 1, I estimate a version of the model

²⁹To put these numbers into context, I follow the results of Cohen and Einav (2007) and consider the amount \$X that would make the average household in my sample indifferent between a sure payoff of \$0 and an equal-odds gamble between winning \$100 and losing \$X. Prior to a diagnosis, the average value of \$X is roughly \$85.08; after diagnosis, this value changes to \$80.85. These results are comparable with previous estimates of household risk aversion for health insurance (Einav et al., 2013; Marone and Sabety, 2020)—however, as mentioned in Einav et al. (2013), the coefficients from models incorporating both health and financial risk do not compare to those of models with pure financial risk (Cohen and Einav, 2007; Handel, 2013).

		Model 1		Model 2		Model 3	
		Estimate	Std. Err.	Estimate	Std. Err.	Estimate	Std. Err.
Panel A: Dynamic Parameters							
<i>Belief Evolution</i>							
π_1	Family Chronic Event	0.69	(0.002)	0.17	(0.002)	0.33	(0.002)
π_2	Own Acute Event	0.07	(0.002)	0.02	(0.001)	0.05	(0.002)
π_3	Family Acute Event	0.09	(0.002)	0.03	(0.001)	0.06	(0.002)
π_4	Years since Chronic Event	-0.01	(0.000)	0.002	(0.000)	0.01	(0.000)
σ_π	Error Variance	10.29	(0.000)	0.12	(0.005)	1.52	(0.018)
<i>Risk Aversion Evolution</i>							
ψ_0	Persistence, Year $t - 1$	—	—	—	—	0.95	(0.025)
ψ_1	Major Health Event (MHE)	—	—	—	—	0.61	(0.015)
ψ_2	MHE \times Total Cost (Year 0)	—	—	—	—	0.19	(0.020)
ψ_3	MHE \times OOP (Year 0)	—	—	—	—	-0.88	(0.024)
ψ_4	MHE \times Hospitalization	—	—	—	—	1.51	(0.033)
σ_ψ	Error Variance	—	—	—	—	0.01	(0.016)
Panel B: Heterogeneity in Types							
σ_ε^2	Idiosyncratic Shock	5.92	(1.006)	6.24	(0.109)	3.56	(0.085)
σ_p^2	Initial Beliefs	16.59	(0.410)	24.43	(0.003)	14.51	(0.001)
σ_ψ^2	Initial Risk Aversion	15.22	(0.289)	5.55	(0.005)	2.57	(0.005)
σ_λ^2	Acute Shocks	—	—	0.58	(0.004)	2.03	(0.001)
$\rho_{p,\psi}$		-0.87	(0.360)	-0.43	(0.002)	-0.54	(0.002)
$\rho_{p,\lambda}$		—	—	-0.91	(0.006)	0.38	(0.002)
$\rho_{\psi,\lambda}$		—	—	0.12	(0.002)	0.09	(0.002)
Beliefs Evolve		Yes		Yes		Yes	
Heterogeneity in Acute Shocks				Yes		Yes	
Risk Aversion Evolves						Yes	

Notes: This table presents estimates for selected parameters of the structural model of health choice; Table 15 presents estimates for the remaining parameters. Belief evolution parameters $\vec{\pi}$ are reported as marginal effects. Standard errors are derived from the analytical Hessian of the likelihood function. Column 3 presents my primary estimates used in later calculations. All models are estimated on an unbalanced panel of 179,044 households over eight years. Preference coefficients are relative to thousands of dollars.

Table 5. Estimated Structural Parameters of Interest

with no heterogeneity in acute health shocks or changes in household risk aversion. That is, $\mu_{\lambda,i}$, $\sigma_{\lambda,i}$, and $\kappa_{\lambda,i}$ are not allowed to vary based on individual covariates, and ψ_{ft} is fixed over time. A key difference between Model 1 and my preferred specification is that the estimated impact of chronic health shocks on risk belief distributions is much higher when I do not accommodate heterogeneity either in period-level health shocks or salience effects. This result is intuitive, as the absence of this heterogeneity leads to the inaccurate “loading” of belief changes onto specific events.³⁰ This loading is observed on a comparable scale for coefficients for acute major health events as well; however, note that these effects are associated with higher overall variance in belief evolution, presumably because the simplified model attempts to explain multiple sources of variation through a single channel.

Column 2 adds variation in acute health status to the model while continuing to hold household risk aversion constant over time. Accounting for this heterogeneity explains a substantial portion of the belief evolution pattern suggested by the most simplified model, with the estimated effect of all major health events reduced by about 2/3 and the estimated size of belief shocks (σ_{π}) reduced drastically. Similarly, including acute health shocks in each period reduces the estimated variation in initial coefficients of risk aversion and the correlation between risk aversion and beliefs, suggesting that including that accounting for variation across health states is important in estimating both health learning and salience effects.

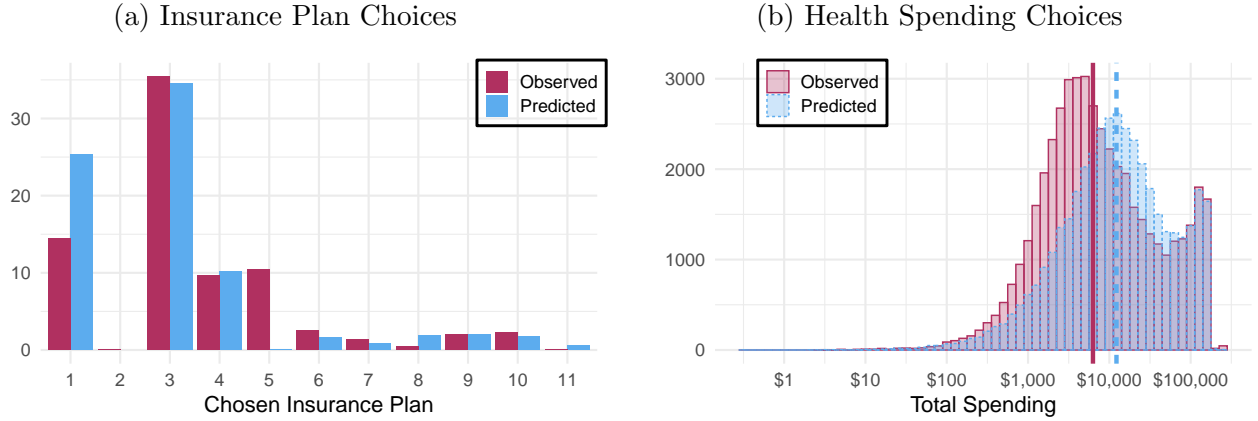
5.1 Model Fit

I evaluate the fit of my estimated model at both the plan choice and spending stages. To evaluate plan choices, I compare plan choices for households observed in the data with those predicted by the model in Figure. Predicted choice probabilities are influenced by premiums, inertia, and household expectations of their acute and chronic health shocks, valued based on their level of risk aversion. At the level of household spending, I compare observed household spending distributions to those predicted by the model. As spending decisions are made after the realization of two random variables (acute and chronic health shocks), I base the model predictions off of a single draw of these underlying variables. I pool all individuals within a firm across years.

Figure 5 presents the results. The first panel shows the observed and predicted market shares for enrollment in plans offered in the largest firm in my sample. Overall, predicted shares are closely matched. The panel on the right presents observed and estimated spending conditional on a plan choice. Here, the model predicts slightly higher levels of billed

³⁰This is exacerbated by the fact that acute health states and chronic diagnoses are correlated, as presented in Panel B of Table 5.

Figure 5. Predicted and Observed Insurance Plan and Health Care Spending Choices



Notes: Figures show overall match between estimated model predictions and observed household choices, at both the plan choice (left) and spending (right) stages of the model. In the first panel, market shares for each insurance plan offered to employees of the single largest firm are shown (see Appendix D.4 for other firms). All years are pooled, so each observation is a household-year. The overall match rate is 82.2%. The second panel plots distributions of predicted and observed household health care spending, conditional on predicted/observed spending greater than zero (the observed rate of zero spending is 16.6% and the predicted rate is 13.2%). All years are pooled, so an observation is a household-year. Vertical lines represent the mean of the respective distribution.

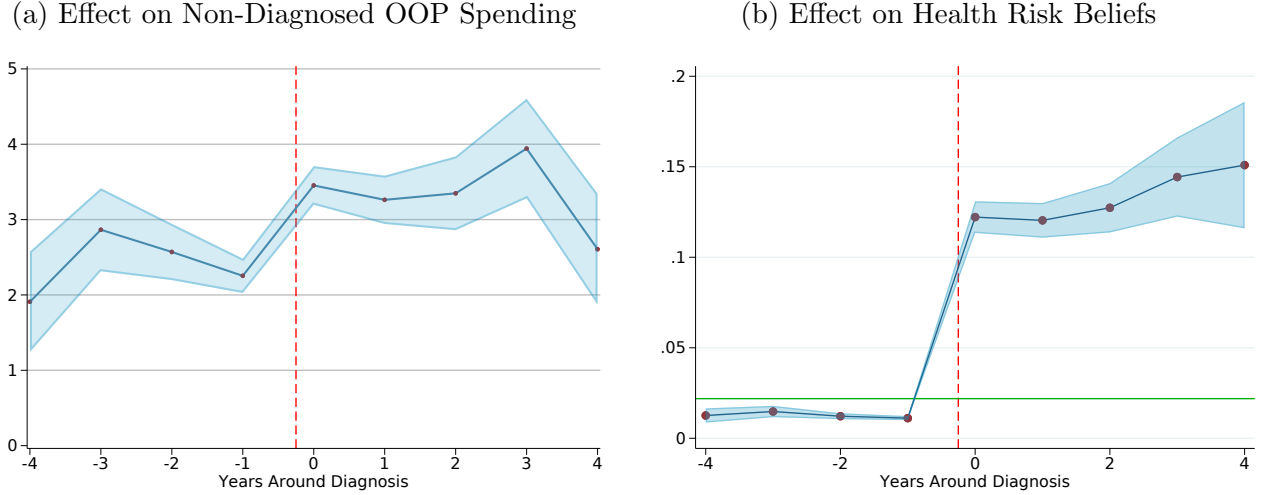
spending than are typically observed, with a difference of about \$1,000 between the means of the two distributions. The model appropriately predicts the extensive margin of spending, appropriately capturing the fraction of individuals who choose zero medical spending in a given year.

5.2 Spending Response to Major Health Events

Figure 6 illustrates the model’s predictions surrounding behavior following new chronic diagnoses in a household as recentered time series graphs. Similar to the results in Section 3, I examine how these diagnoses alter the spending patterns of other household members in the panel (a). I also present estimates for how diagnoses affect estimates for individuals’ underlying transition probabilities p_{it} in panel (b). I find that household diagnoses are associated with about a 20% increase in OOP spending—this is slightly higher than the 10% estimated changes reported in Figure 1, although the differences are not statistically significant.

Importantly, I predict large accompanying changes in individual health risk beliefs following a new chronic diagnosis in the family. The horizontal green line in the Panel (b) of Figure 6 depicts the pooled average risk of diagnosis within my sample, which is roughly 2.5%. Prior to health events, individuals tend to underweight their health risks by about 58%; however, following a diagnosis, individuals move to *over-weighting* their risks by over

Figure 6. Model Predictions: Non-Diagnosed Spending and Beliefs Around a New Diagnosis



Notes: Figures show recentered time series for model predictions of spending and beliefs for non-diagnosed household members who have experienced a diagnosis with a new chronic illness in the household. The first panel illustrates percentage changes in the inverse hyperbolic sine of OOP spending, measured in 2020 USD. The second panel illustrates estimated changes in predicted beliefs, averaged over draws from individual posterior distributions. The green horizontal line in Panel (b) illustrates the average in-sample rate of diagnosis with a new chronic condition.

six times the true in-sample rates of diagnosis. Instead, these households make choices as though they perceived their risk of a chronic diagnosis to be greater than one in ten. This provides suggestive evidence that individuals in affected households may over-respond to these events. I explore the welfare implications of these facts in the following section.

6 Welfare & Counterfactual Simulations

Based on the estimated model parameters, I am able to construct a measure of each household's willingness to pay for information associated with their own health risks. I use this measure to provide a benchmark for the value associated with this information, with particular focus on whether major health events meaningfully alter individual expected utility and social surplus.

6.1 Welfare Effects of Information

Households who receive health information alter their plan choice and medical spending decisions, thereby altering their *ex-ante* expected payoffs from care. I estimate the value of this information by comparing household certainty equivalents for two regimes: one in which the health information is revealed as observed, and a benchmark regime in which no

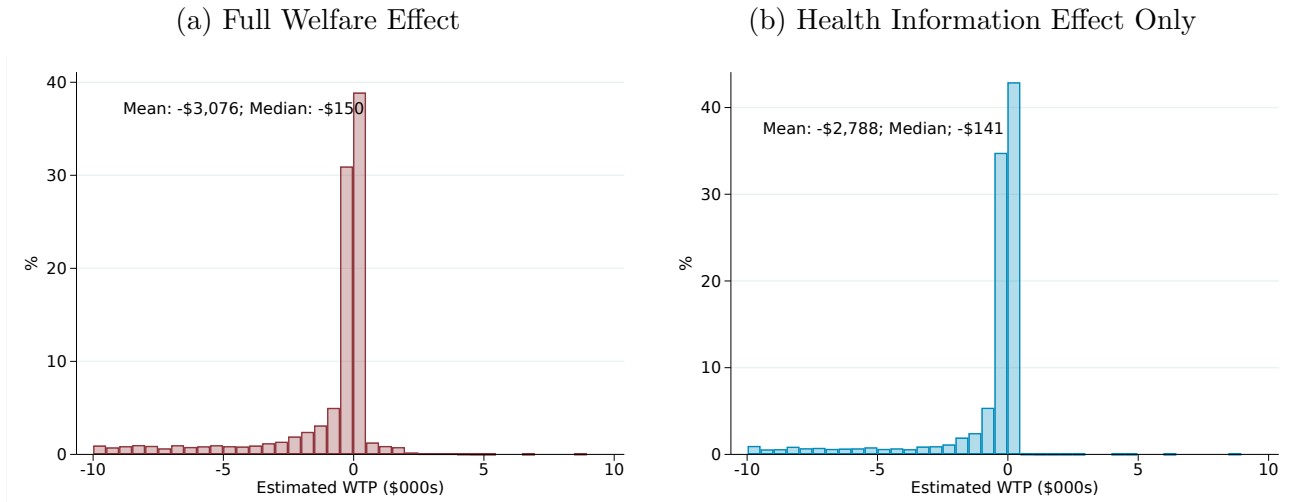
information is transmitted. Specifically, in the benchmark state of the world, households experience changes to their health states without corresponding changes to their beliefs, risk aversion, or the conditional costs of non-chronic care. I perform this analysis only on non-diagnosed family members within an affected household to examine only changes in welfare associated with information, rather than true changes in underlying health status.

A household's willingness to pay for health information is equal to the difference in certainty equivalents across these two regimes. Certainty equivalents are given by

$$CE_{fjt} = -\psi_{ft}^{-1} \log(-U_{fjt}), \quad (16)$$

where U_{fjt} is the total *ex-ante* expected utility family f expects when enrolling in plan j at time t , as defined in equation 9. I assume that conditional on the estimated parameters, households are fully rational and enroll in the plan that gives the highest expected utility at the time of choice.³¹ Throughout, I report differences between CE_{fjt} across the benchmark state of the world and regimes where information is partially or fully revealed; hence, reported values are “marginal” willingness to pay measures.

Figure 7. Variation in Welfare Effects Associated with Health Events and Health Information



Notes: Figures show estimated changes in household willingness to pay associated with major health events. The panel on the left shows differences in household certainty equivalents in the case of a full response to a new diagnosis, including adjustments to risk aversion and moral hazard effects; the panel on the right shows only differences arising from adjustments to household risk assessments. Welfare effects are calculated in the year of the diagnosis relative to a benchmark in which no information is transmitted.

³¹The model allows for rich heterogeneity in the prediction of health states as well as rationalizations for common choice mistakes, including switching costs. Hence, such an assumption is reasonable. Similarly, I assume that the idiosyncratic shock parameter is not relevant for the context of estimating welfare gains from health information.

Figure 7 presents the main welfare results in the year of the new chronic diagnosis.³² Household members who are exposed to a new chronic diagnosis experience a welfare penalty that averages \$3,076 per household per year. However, there is substantial heterogeneity in these effects, including 28% of treated families who have a higher resulting expected utility following the realization of health information.

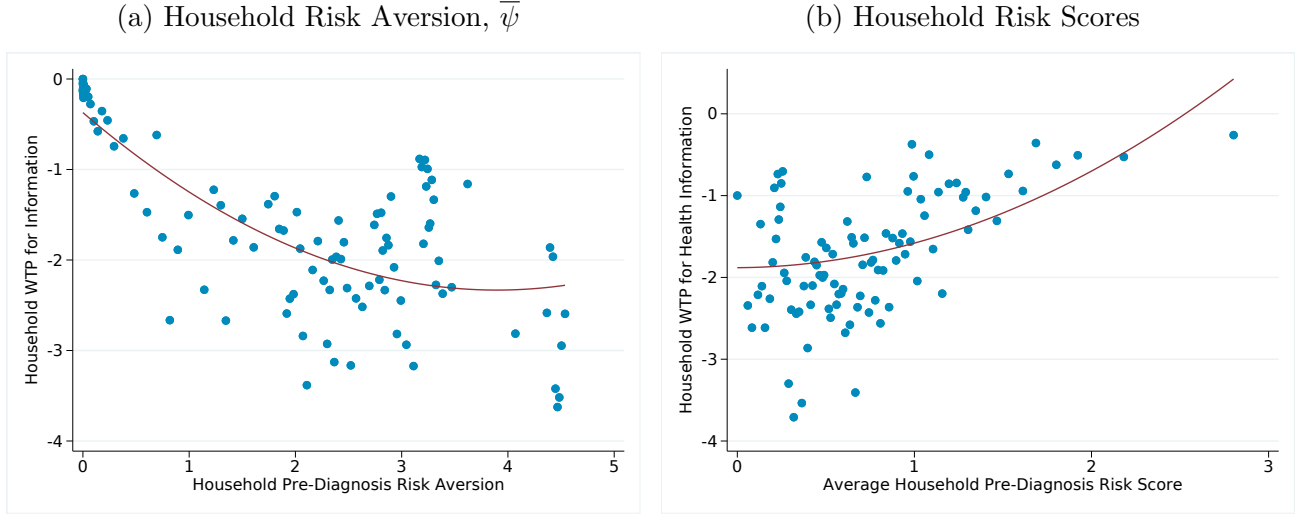
The right panel of Figure 7 shows the distribution of welfare effects associated solely with receiving new health information. A novel feature of my structural model is the ability to separate changes to household welfare that arise from dimensions of a health event other than the realization of health information. I recalculate welfare changes associated with *only* changes to household beliefs by holding constant changes to both household risk aversion and any moral hazard effects that arise from changes to spot prices. My analysis reveals that these dimensions contribute little to overall changes in household welfare, with 90% of welfare changes being explicitly attributable to changes in household beliefs. The average household experiences a welfare penalty of \$2,788 associated with changes to how they evaluate their risk of developing a chronic condition.

Although at first glance associating new information with a welfare penalty seems counter-intuitive, my results are consistent with a story of household over-responsiveness to information. The observed choice data which informed the estimated model parameters suggests that new chronic diagnosis spur large swings in household members' assessments of their health risks; however, these welfare calculations make clear that in many cases, households would be better off if they had acted as though they had not received the information. This is precisely because of the magnitude of the shifts in household beliefs, as I illustrate in the following section.

Importantly, the returns to health information vary with key household characteristics, including household risk levels and estimated risk aversion. Figure 8 presents these results. Households who are less averse to negative outcomes prior to the diagnosis experience lower welfare penalties, on average, than those with higher risk aversion. Differences in this parameter are intuitively meaningful: households with greater risk aversion experience greater "translation" of new health information into changes in insurance plan choices and, subsequently, health spending. Hence, households with lower levels of risk aversion tend to respond less to new information, presumably contributing to the lower estimated welfare penalties associated with the event. Similarly, households with high expected health risks prior to a new diagnosis experience lower welfare penalties. This, too, is related to overall muted responses to health information. However, this low level of responsiveness is attributable not

³²These welfare effects are relatively stable in the first few years following the diagnosis. For details on this, see Appendix D.4.

Figure 8. Heterogeneity in the Value of Information based on Household Characteristics



Notes: Figures show binscatters depicting the association between the estimated welfare effects of receiving health risk information and pre-diagnosis household health characteristics. These include (a) average household risk aversion and (b) average household risk scores (calculated using the Johns Hopkins ACG System). Welfare effects are calculated in the year of the diagnosis relative to a benchmark in which no information is transmitted; see Figure 7 for details. Binscatters are constructed using 100 bins and a quadratic fit line.

to low variation in expected utility but an already high level of expected spending, meaning new health events change outcomes (in percentage terms) less.

6.2 Evaluating Household Over-Responsiveness to Information

The results above imply that while households respond meaningfully to new health information, they may not be doing so in ways that are welfare improving. Given these estimated welfare penalties, in this section I assess the extent to which consumers' over-responsiveness to health information dampens potential welfare gains. The model predicts large swings in consumer beliefs when exposed to chronic diagnoses in a household. I therefore first assess the extent to which limiting the magnitude of these changes affects estimated welfare differences. I then turn to practical policy questions surrounding when information revelation is optimal, and whether targeted revelation can improve social outcomes.

6.2.1 Bounding Belief Updating

I first consider how limiting household responsiveness to adverse health events alters estimated welfare gains or penalties from new health risk information. Here, I present estimated effects from imposing arbitrary upper bounds on an individual's beliefs about their own health risks; that is, imposing that any predicted value p_{it} in the model be no greater

than some threshold \bar{p} . This exercise illustrates that if consumers' responses more closely matched their true expected risk (conditional on the household member's diagnosis), health information would be associated with welfare gains rather than losses.³³

Upper bound, \bar{p} (%)	Willingness to Pay for Health Information		% with Welfare Gains
	Mean	Median	
—	−\$2,788	−\$141	00.2
10	\$2,439	\$207	78.9
5	\$2,472	\$223	80.3
2.5	\$2,484	\$230	81.5
1	\$2,492	\$234	83.1
0.5	\$2,495	\$234	84.4
\hat{p}_{it}	\$2,385	\$219	83.9

Notes: Welfare effects are calculated for the year of the diagnosis, relative to a benchmark state where no information is transmitted. Moral hazard and risk aversion do not change across states.

Table 6. Value of Health Information After Bounding Consumer Responsiveness

Table 6 presents the results. In general, even imposing a bound on consumers' beliefs such that no individual rates their *ex-post* risk of developing a chronic condition higher than 10% increases the percentage of households who gain from health information from 0.2% to 78.9%. Once this bound is imposed, households would be willing to pay an average (median) of \$2,439 (\$207) to have this health information revealed to them, rather than experiencing a welfare penalty in the unrestricted context. Further restrictions on this bound improve welfare estimates and the percentage of households with positive welfare changes, but only slightly. This suggests that welfare losses are arising principally from individuals responding to diagnoses with new beliefs well above 10%. To the extent that these chronic conditions remain rare and genetic information does not inform risk probabilities above 10%, welfare gains from health information are hence being eliminated as individuals overweight their *ex-post* probabilities of illness.

One concern with this approach is that individuals within households may have private information compelling them to have these large changes in their beliefs. Such private information, including about underlying health status, could rationalize observed welfare penalties. The extent to which private information could inform patients of health risks of developing a chronic condition higher than odds of one in ten seem unlikely. However, to address this concern, I estimate individual-specific health risks \hat{p} based on demographics

³³I present alternative simulations expressing the same sentiment in Appendix D.4; these include imposing bounds on the model parameter π_1 controlling the impact of chronic events on individual beliefs.

including age, sex, and relationship with diagnosed household members. Although these predicted health risk probabilities do not capture the full range of private information, they address individual differences in potential responsiveness to new information.

I estimate predicted health risk probabilities on a validation sample constructed from all Marketscan households not in my main sample who experience at least one chronic event during their observed period. Additional details about this estimation and summary statistics for the resulting probabilities are provided in Appendix D.4. The predicted probabilities are small, averaging about 2%.

The bottom row of Table 6 uses these predicted probabilities as the upper bound for the predicted changes to individual beliefs arising from the model (note that now these bounds are individual-specific). Even accommodating individual-specific deviations in information bounds, I continue to find that restraining the extent to which individuals respond to health information corresponds to higher overall valuations of health information. The estimated effect sizes are similar to arbitrary uniform bounds, with an average welfare gain of \$2,385 per household-year and 83.9% of households experiencing gains from health information.

6.2.2 Targeting Information to Maximize Gains

In addition to concerns about individual over-responsiveness to health information, policy guiding the revelation of health information must also balance the potentially heterogeneous returns from such revelation. In the face of such variation, full information revelation may not be socially optimal. This includes cases where a full screening regime is not financially feasible, where the information itself may result in consumers declining actuarially fair insurance (Posey and Thistle, 2021), or where there is a disconnect between privately and socially optimal information revelation (Oster et al., 2013). In these cases, the ability to target policies that reveal health risk information may improve the social returns as well as the fraction of households who benefit from these programs.

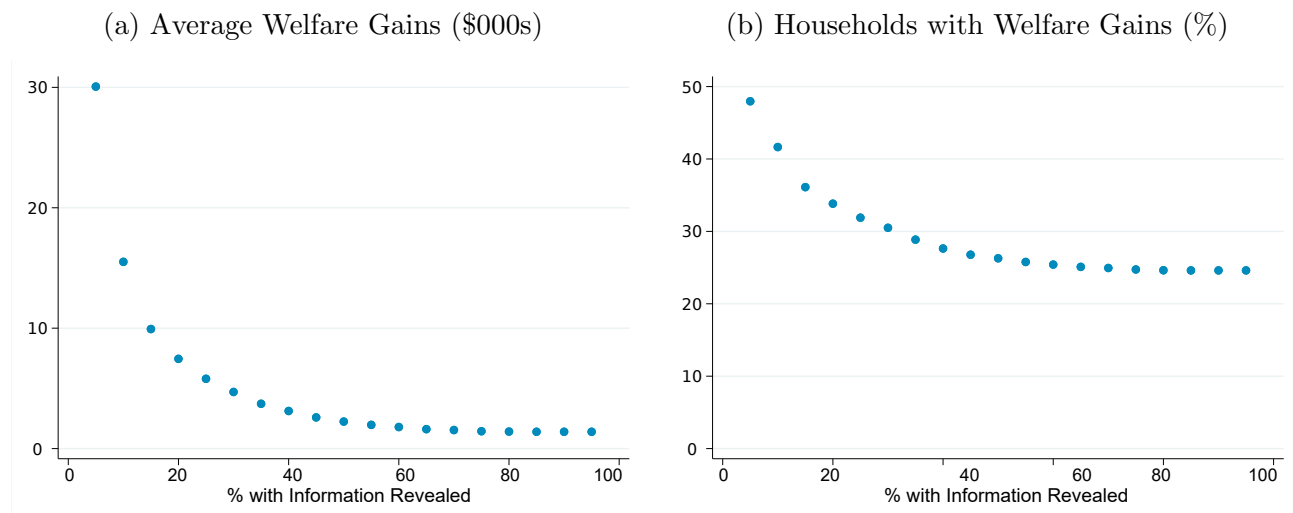
I estimated strong heterogeneous returns to health information as presented in Figure 8. Based on these results, I consider the effects of targeting information revelation based on observable characteristics, such as individual risk measured by risk scores.³⁴ I consider a scenario in which individuals can receive a one-time update to information about their health risks, modeled as changes to their probability of adverse health events p_{it} . When individuals receive this information, this probability is adjusted to be equivalent to their predicted risk probability \hat{p}_{it} defined above. I assume that following this information, individual beliefs

³⁴My model predicts variation in the value of health information based on less-easily observable characteristics, such as household risk aversion. Although perhaps more costly to observe, information targeting along this dimension may also prove beneficial. I present these results in Appendix D.4.

are constant at their predicted risk level, with no residual uncertainty or updating across periods.³⁵ I further assume that this information does not entail any salience or moral hazard effects.

This scenario therefore mirrors a hypothetical transmission of health information that informs consumers of their health risks as perfectly as population-data allows.³⁶ I present results illustrating the individual and social value of such a policy based on a sample of 10,000 households in my sample which do not experience major health events. These individuals may still have erroneous beliefs about their health risks and may benefit from new health information. Further, estimating the effects of this policy on this group provides a validating sample for the results presented earlier documenting the value of information transmitted in a more quasi-random setting.

Figure 9. Changes in Welfare Gains From Targeted Revelation of Information



Notes: Figures show estimated welfare gains associated with revelation of health information. Individuals are organized based on their average risk scores, from highest to lowest. Each point in both panels represents a different scenario in which the individuals with in the highest x%-percentile of risk score are given information about their predicted health risks, \hat{p} , as described in the text. Returns to health information are presented as (a) average expected welfare changes, measured as willingness to pay in thousands of dollars, and (b) the percentage of households with positive welfare gains.

Figure 9 presents the results. Across all the households in my sample, the average household would be willing to pay approximately \$1,500 per year for information about

³⁵In reality, individuals are more likely to behave as though this information revelation were a single non-definitive signal, although perhaps one to which they ascribe a large weight relative to the others. This would imply that individuals do not update their beliefs fully to the revealed target \hat{p}_{it} in the year of the signal nor hold their beliefs constant in the years after the signal. However, for the purposes of this exercise, such fluctuations serve only to obfuscate the potential benefits of targeting information revelation relative to universal revelation. I therefore use the simplest version of the model that highlights these differences.

³⁶Note that \hat{p}_{it} does not represent an individual's *true* risk given the lack of private information, including underlying health states.

their specific health risks (the right-most point in Panel (a)); however, only about a quarter of households would be willing to pay anything for this information (the right-most point in Panel (b)). This highlights the central tension behind the revelation of health information: even revealing risks that are true on average for an individual can result in worse *ex-ante* welfare. This is, in part, due to the fact that households who increase their beliefs about the risk of chronic diagnosis value non-chronic health care less.

Both panels of the figure present alternative scenarios where the information is not revealed to every household indiscriminately, but only to individuals with the highest estimated levels of risk. Each point represents a scenario in which only individuals with the highest $x\%$ risk scores receive health information. These alternative scenarios highlight that targeting health information along observable demographics may help improve the returns from a policy increasing access to health information. Even under the assumption that this information can be revealed without over-responsiveness, demographics can be used to predict which individuals and households are most likely to benefit from the information.

7 Conclusion

This paper assesses the extent to which information about one’s health risks alters individual and household decision-making in health care. I demonstrate that households who receive new information about health risks from a new diagnosis in the household increase their overall levels of spending, including investments in both preventive and low-value services. These changes in behavior are best explained by individual household members reassessing their risks, rather than responding to financial incentives or salience effects. However, these reassessments do not meaningfully improve the quality of health care choices. While access to new health information changes behavior in meaningful ways, it does not necessarily do so in welfare-improving ones.

To explore this further, I use a structural approach to quantify a household’s willingness to pay for health information, isolating the specific effects of new health information from other mechanisms. The model implies low realized returns to health information, most likely due to individual misinterpretation of their health risks following the health event. Bounding the extent to which individuals increase their beliefs about risks post-diagnosis substantially improves realized welfare. Finally, my analysis illustrates that information revelation is privately most optimal for individuals with high *ex-ante* risk and those with low risk aversion.

The analysis I present could be extended in several meaningful ways. First, future work could relax the assumption that individuals have no control over their chronic care health

costs. This would be particularly interesting in non-ESI covered populations, such as those covered by public insurance programs or without any coverage, for whom chronic diagnoses may impose large financial burdens (Hadley, 2007). Another important consideration left out of the model is how liquidity constraints change *ex-post* spending adjustments as health risks change (Gross et al., 2020). 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).

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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A Data Cleaning

This appendix provides more detail on how the sample is constructed for analysis. I discuss the imputing of plan characteristics, identification of major health events, and identification of costs associated with chronic illnesses in detail in each of the sections below.

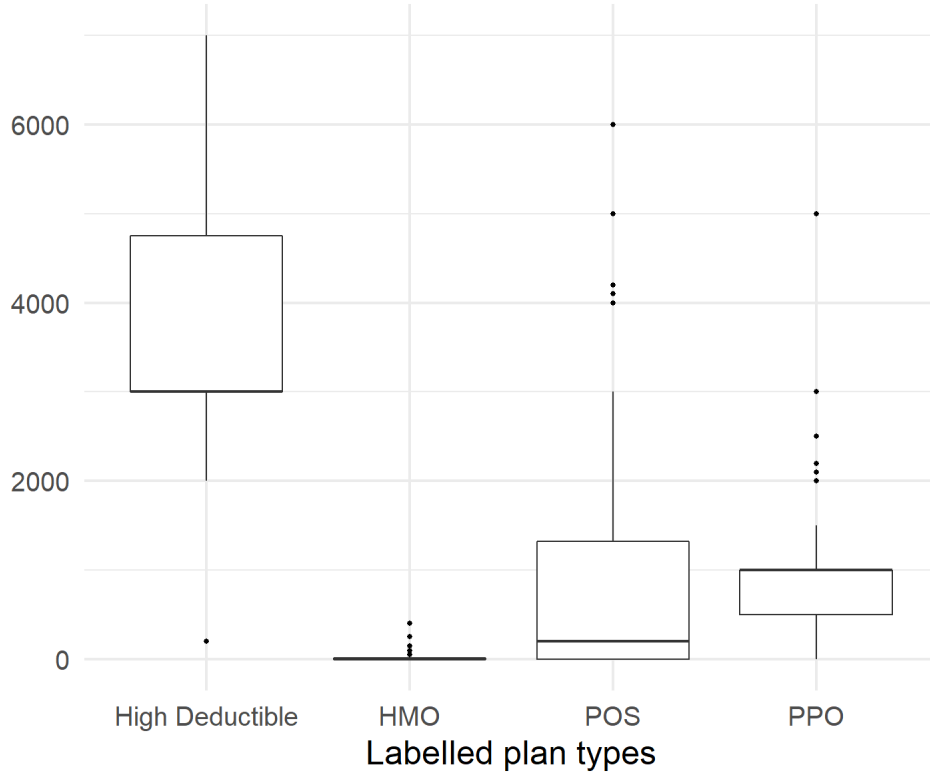
A.1 Identifying Plan Characteristics

I follow the methodology of Zhang et al. (2018) in inferring individual and household deductibles from the empirical distribution of claims. In particular, given the claims for an individual plan-year, I follow these steps:

1. Remove claims that are out-of-network, as well as claims with negative values in any of the total paid, plan paid, deductible, and OOP fields.
2. Limit attention to families that had at least 4 consecutive zero-deductible claims after the last positive deductible claim (to ensure that the deductible has really been reached).
3. Calculate each family’s total deductible contribution over the year.
4. Next, estimate the mode and 95th percentile of the deductible within each plan-year.

Figure 10 illustrates the match quality of these assignments by comparing the distribution of imputed plan family deductibles across listed plan types (Rabideau et al., 2021).

Figure 10. Imputed Family Deductibles by Listed Plan Type

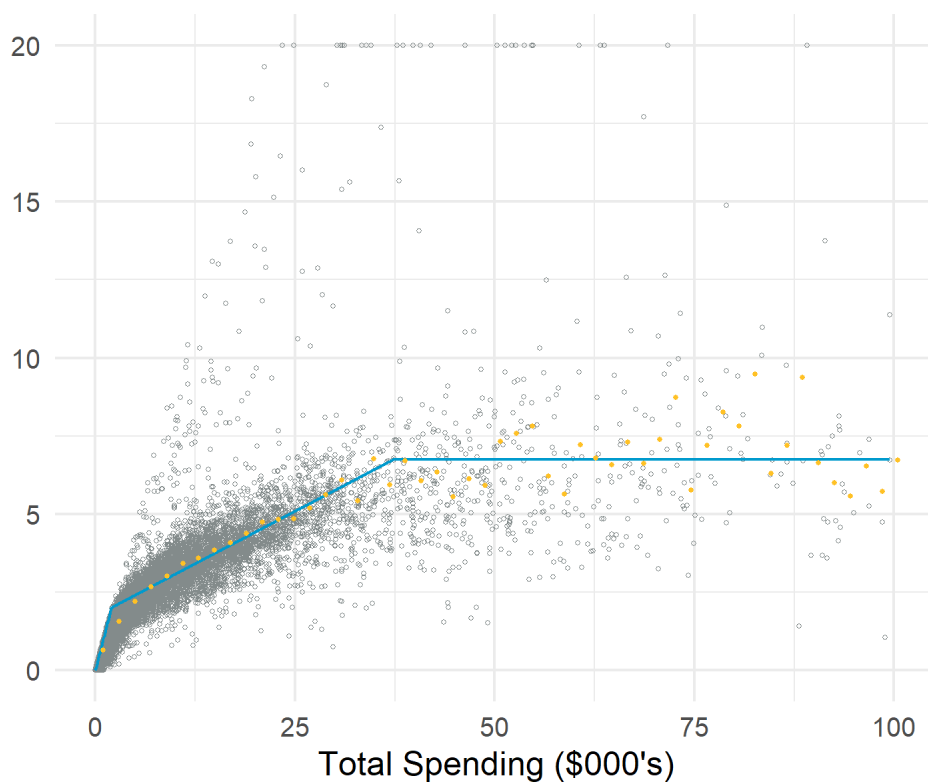


Notes: Box and whisker plot summarizing imputed household deductibles for each listed plan type. Each observation is a plan-year. The box in each boxplot extends from the first quartile to the third quartile of all family deductibles, with a line in the middle for the median. Whiskers extend to 1.5 times the interquartile range (the length of the box) if applicable. All plan years with deductibles outside of the whiskers are shown as outlier points.

Once deductibles are estimated, average coinsurance rates and out-of-pocket maxima are estimated using the methodology of Marone and Sabety (2020). These cost-sharing parameters are those which minimizes the sum of squared residuals between predicted and observed out-of-pocket cost, where predicted out-of-pocket costs utilizes the estimated deductible and assumed coinsurance and OOP maximum and observed spending comes directly from the claims data. This estimation is done separately for each plan-year.

Figure 11 illustrates the estimated function used in calculating out-of-pocket costs for a given amount of medical spending in a particular insurance plan and year, compared with the realized distribution of total and out-of-pocket spending for all households enrolled in that plan during the year. Each gray dot represents a household, and gold dots are a binscatter plot of all households, using 100 bins. The estimated features of this plan are a family deductible of \$2,000, a coinsurance rate of 13.4%, and a family out-of-pocket maximum of \$6,750.41.

Figure 11. Inferred Characteristics for a Sample Insurance Plan



Notes: Data shown for a single plan year. Each gray dot corresponds to a single household's observed total and out-of-pocket spending. Gold dots show averages within 50 bins. Blue line illustrates the estimated piece-wise linear function translating observed billed spending into out-of-pocket spending, determined by a plan deductible, coinsurance rate, and out-of-pocket maximum.

A.2 Identifying Major Health Events

I assign major health events using a set of chronic and acute HCCs, as discussed in Section 2 of the main text. The following table identifies each major health event as well as its corresponding status (acute/chronic) and accompanying diagnosis codes. Prior to October 2015, MarketScan claims data relied on ICD-9-CM diagnosis codes, transitioning to ICD-10-CM diagnosis codes thereafter.

Need to add the following HCCs for acute events:

- 3, 4, 38, 45, 55, 127, 131, 132, 145, 146, 149, 156

Service Type	Chronic?	Diagnosis Codes (ICD-9-CM)	Diagnosis Codes (ICD-10-CM)
Adrenal/Pituitary Disorders	Yes	0363, 2510, 25200, 25201, 25202, 25208, 2521, 2528, 2529, 2530, 2531, 2532, 2533, 2534, 2535, 2536, 2537, 2538, 2539, 2540, 2541, 2548, 2549, 2550, 25510, 25511, 25512, 25513, 25514, 2552, 2553, 25541, 25542, 2555, 2556, 2558, 2559, 25801, 25802, 25803, 2581, 2588, 2589, 5881, 58881	A391, E035, E15, E200, E208, E209, E210, E211, E212, E213, E214, E215, E220, E221, E222, E228, E229, E230, E231, E232, E233, E236, E237, E240, E241, E242, E243, E244, E248, E249, E250, E258, E259, E2601, E2602, E2609, E261, E2681, E2689, E269, E270, E271, E272, E273, E2740, E2749, E275, E278, E279, E310, E311, E3120, E3121, E3122, E3123, E318, E319, E320, E321, E328, E329, E344, E892, E893, E896, N251, N2581
Asthma	Yes	49300, 49301, 49302, 49310, 49311, 49312, 49381, 49382, 49390, 49391, 49392	J4520, J4521, J4522, J4530, J4531, J4532, J4540, J4541, J4542, J4550, J4551, J4552, J45901, J45902, J45909, J45990, J45991, J45998
Brain Infections	No	00321, 0065, 01300, 01301, 01302, 01303, 01304, 01305, 01306, 01310, 01311, 01312, 01313, 01314, 01315, 01316, 01320, 01321, 01322, 01323, 01324, 01325, 01326, 01330, 01331, 01332, 01333, 01334, 01335, 01336, 01340, 01341, 01342, 01343, 01344, 01345, 01346, 01350, 01351, 01352, 01353, 01354, 01355, 01356, 01360, 01361, 01362, 01363, 01364, 01365, 01366, 01380, 01381, 01382, 01383, 01384, 01385, 01386, 01390, 01391, 01392, 01393, 01394, 01395, 01396, 0360, 0361, 037, 04500, 04501, 04502, 04503, 04510, 04511, 04512, 04513, 04520, 04521, 04522, 04523, 04590, 04591, 04592, 04593, 0498, 0499, 0520, 0543, 0550, 05601, 05821, 05829, 0620, 0621, 0622, 0623, 0624, 0625, 0628, 0629, 0630, 0631, 0632, 0638, 0639, 064, 0662, 06640, 06641, 06642, 06649, 071, 0722, 09040, 09041, 09042, 09049, 09181, 0940, 0941, 0942, 0943, 09481, 09482, 09483, 09484, 09485, 09486, 09489, 0949, 09882, 10081, 11283, 1142, 11501, 11511, 11591, 3200, 3201, 3202, 3203, 3207, 32081, 32082, 32089, 3209, 3211, 3213, 3214, 3218, 32301, 3231, 3232, 32341, 32351, 32361, 32362, 32381, 3239, 3240, 3241, 3249, 325	A0101, A0221, A066, A170, A171, A1781, A1782, A1783, A1789, A179, A203, A2781, A3211, A3212, A34, A35, A390, A3981, A4281, A4282, A5040, A5041, A5042, A5043, A5044, A5045, A5049, A5141, A5210, A5211, A5212, A5213, A5214, A5215, A5216, A5217, A5219, A522, A523, A5481, A5482, A6921, A800, A801, A802, A8030, A8039, A804, A809, A820, A821, A829, A830, A831, A832, A833, A834, A835, A836, A838, A839, A840, A841, A848, A849, A850, A851, A852, A858, A86, A888, A89, A922, A9230, A9231, A9232, A9239, B004, B0111, B020, B050, B0601, B1001, B1009, B262, B375, B384, B4081, B4281, B431, B5741, B5742, B6011, G000, G001, G002, G003, G008, G009, G01, G02, G0400, G0401, G0402, G042, G0430, G0431, G0432, G0439, G0481, G0490, G053, G060, G061, G062, G07, G08
Breast and Prostate Cancer	Yes	1740, 1741, 1742, 1743, 1744, 1745, 1746, 1748, 1749, 1750, 1759, 179, 1800, 1801, 1808, 1809, 1820, 1821, 1828, 1840, 1841, 1842, 1843, 1844, 1848, 1849, 185, 1880, 1881, 1882, 1883, 1884, 1885, 1886, 1887, 1888, 1889, 1892, 1893, 1894, 1898, 1899, 1900, 1901, 1902, 1903, 1904, 1905, 1906, 1907, 1908, 1909, 1950, 1951, 1952, 1953, 1954, 1955, 1958, 1992, 20100, 20101, 20102, 20103, 20104, 20105, 20106, 20107, 20108, 20110, 20111, 20112, 20113, 20114,	C4A0, C4A10, C4A11, C4A12, C4A20, C4A21, C4A22, C4A30, C4A31, C4A39, C4A4, C4A51, C4A52, C4A59, C4A60, C4A61, C4A62, C4A70, C4A71, C4A72, C4A8, C4A9, C50011, C50012, C50019, C50021, C50022, C50029, C50111, C50112, C50119, C50121, C50122, C50129, C50211, C50212, C50219, C50221, C50222, C50229, C50311, C50312, C50319, C50321, C50322, C50329, C50411, C50412, C50419, C50421, C50422, C50429, C50511, C50512, C50519,

Service Type	Chronic?	Diagnosis Codes (ICD-9-CM)	Diagnosis Codes (ICD-10-CM)
		20115, 20116, 20117, 20118, 20120, 20121, 20122, 20123, 20124, 20125, 20126, 20127, 20128, 20140, 20141, 20142, 20143, 20144, 20145, 20146, 20147, 20148, 20150, 20151, 20152, 20153, 20154, 20155, 20156, 20157, 20158, 20160, 20161, 20162, 20163, 20164, 20165, 20166, 20167, 20168, 20170, 20171, 20172, 20173, 20174, 20175, 20176, 20177, 20178, 20190, 20191, 20192, 20193, 20194, 20195, 20196, 20197, 20198, 20900, 20901, 20902, 20903, 20910, 20911, 20912, 20913, 20914, 20915, 20916, 20917, 20920, 20921, 20922, 20923, 20924, 20925, 20926, 20927, 20929, 20930, 20931, 20932, 20933, 20934, 20935, 20936, 2250, 2251, 2252, 2253, 2254, 2258, 2259, 2273, 2274, 22802, 2370, 2371, 2373, 2375, 2376, 2379, 2396, 7595, 7596	C50521, C50522, C50529, C50611, C50612, C50619, C50621, C50622, C50629, C50811, C50812, C50819, C50821, C50822, C50829, C50911, C50912, C50919, C50921, C50922, C50929, C510, C511, C512, C518, C519, C52, C530, C531, C538, C539, C540, C541, C542, C543, C548, C549, C55, C577, C578, C579, C61, C661, C662, C669, C670, C671, C672, C673, C674, C675, C676, C677, C678, C679, C680, C681, C688, C689, C6900, C6901, C6902, C6910, C6911, C6912, C6920, C6921, C6922, C6930, C6931, C6932, C6940, C6941, C6942, C6950, C6951, C6952, C6960, C6961, C6962, C6980, C6981, C6982, C6990, C6991, C6992, C760, C761, C762, C763, C7640, C7641, C7642, C7650, C7651, C7652, C768, C7A00, C7A010, C7A011, C7A012, C7A019, C7A020, C7A021, C7A022, C7A023, C7A024, C7A025, C7A026, C7A029, C7A090, C7A091, C7A092, C7A093, C7A094, C7A095, C7A096, C7A098, C7A1, C7A8, C802, C8100, C8101, C8102, C8103, C8104, C8105, C8106, C8107, C8108, C8109, C8110, C8111, C8112, C8113, C8114, C8115, C8116, C8117, C8118, C8119, C8120, C8121, C8122, C8123, C8124, C8125, C8126, C8127, C8128, C8129, C8130, C8131, C8132, C8133, C8134, C8135, C8136, C8137, C8138, C8139, C8140, C8141, C8142, C8143, C8144, C8145, C8146, C8147, C8148, C8149, C8170, C8171, C8172, C8173, C8174, C8175, C8176, C8177, C8178, C8179, C8190, C8191, C8192, C8193, C8194, C8195, C8196, C8197, C8198, C8199, D1802, D320, D321, D329, D330, D331, D332, D333, D334, D337, D339, D352, D353, D354, D420, D421, D429, D430, D431, D432, D433, D434, D438, D439, D443, D444, D445, D446, D447, D496, Q851, Q858, Q859
Cardio-Respiratory Failure	No	42741, 42742, 4275, 514, 5184, 51881, 51882, 51883, 51884, 769, 7703, 7704, 7705, 7707, 77084, 77985, 78550, 78551, 7980, 7981, 7982, 7989, 9584	I462, I468, I469, I4901, I4902, J182, J80, J810, J811, J9600, J9601, J9602, J9610, J9611, J9612, J9620, J9621, J9622, J9690, J9691, J9692, P220, P260, P261, P268, P269, P270, P271, P278, P279, P280, P2810, P2811, P2819, P285, P2981, R570, R579, T794XXA
Chronic Hepatitis	Yes	07022, 07023, 07032, 07033, 07044, 07054, 57140, 57141, 57142, 57149	B180, B181, B182, B188, B189, K730, K731, K732, K738, K739, K754
Chronic Skin Ulcer	Yes	4540, 4542, 45911, 45913, 45931, 45933, 68601, 70710, 70711, 70712, 70713, 70714, 70715, 70719, 7078, 7079	I83001, I83002, I83003, I83004, I83005, I83008, I83009, I83011, I83012, I83013, I83014, I83015, I83018, I83019, I83021, I83022, I83023, I83024, I83025, I83028, I83029, I83201, I83202, I83203, I83204,

Service Type	Chronic?	Diagnosis Codes (ICD-9-CM)	Diagnosis Codes (ICD-10-CM)
			I83205, I83208, I83209, I83211, I83212, I83213, I83214, I83215, I83218, I83219, I83221, I83222, I83223, I83224, I83225, I83228, I83229, I87011, I87012, I87013, I87019, I87031, I87032, I87033, I87039, I87311, I87312, I87313, I87319, I87331, I87332, I87333, I87339, L88, L97101, L97102, L97103, L97104, L97109, L97111, L97112, L97113, L97114, L97119, L97121, L97122, L97123, L97124, L97129, L97201, L97202, L97203, L97204, L97209, L97211, L97212, L97213, L97214, L97219, L97221, L97222, L97223, L97224, L97229, L97301, L97302, L97303, L97304, L97309, L97311, L97312, L97313, L97314, L97319, L97321, L97322, L97323, L97324, L97329, L97401, L97402, L97403, L97404, L97409, L97411, L97412, L97413, L97414, L97419, L97421, L97422, L97423, L97424, L97429, L97501, L97502, L97503, L97504, L97509, L97511, L97512, L97513, L97514, L97519, L97521, L97522, L97523, L97524, L97529, L97801, L97802, L97803, L97804, L97809, L97811, L97812, L97813, L97814, L97819, L97821, L97822, L97823, L97824, L97829, L97901, L97902, L97903, L97904, L97909, L97911, L97912, L97913, L97914, L97919, L97921, L97922, L97923, L97924, L97929, L98411, L98412, L98413, L98414, L98419, L98421, L98422, L98423, L98424, L98429, L98491, L98492, L98493, L98494, L98499, I70231, I70232, I70233, I70234, I70235, I70238, I70239, I70241, I70242, I70243, I70244, I70245, I70248, I70249, I7025, I70331, I70332, I70333, I70334, I70335, I70338, I70339, I70341, I70342, I70343, I70344, I70345, I70348, I70349, I7035, I70431, I70432, I70433, I70434, I70435, I70438, I70439, I70441, I70442, I70443, I70444, I70445, I70448, I70449, I7045, I70531, I70532, I70533, I70534, I70535, I70538, I70539, I70541, I70542, I70543, I70544, I70545, I70548, I70549, I7055, I70631, I70632, I70633, I70634, I70635, I70638, I70639, I70641, I70642, I70643, I70644, I70645, I70648, I70649, I7065, I70731, I70732, I70733, I70734, I70735, I70738, I70739, I70741, I70742, I70743, I70744, I70745, I70748, I70749, I7075
Congestive Heart Failure	Yes	39891, 40201, 40211, 40291, 40401, 40403, 40411, 40413, 40491, 40493, 4150, 4160, 4161, 4168, 4169, 4170, 4171, 4178, 4179, 4250, 42511, 42518, 4252, 4253, 4254, 4255, 4257, 4258, 4259, 4280, 4281, 42820, 42821, 42822, 42823, 42830, 42831, 42832, 42833,	A3681, B3324, I0981, I110, I130, I132, I2601, I2602, I2609, I270, I271, I272, I2781, I2789, I279, I280, I281, I288, I289, I420, I421, I422, I423, I424, I425, I426, I427, I428, I429, I43, I501, I5020, I5021, I5022, I5023, I5030,

Service Type	Chronic?	Diagnosis Codes (ICD-9-CM)	Diagnosis Codes (ICD-10-CM)
		42840, 42841, 42842, 42843, 4289, 4290, 4291	I5031, I5032, I5033, I5040, I5041, I5042, I5043, I509, I514, I515
Diabetes w/ Complications	Yes	24940, 24941, 24950, 24951, 24960, 24961, 24970, 24971, 24980, 24981, 24990, 24991, 25040, 25041, 25042, 25043, 25050, 25051, 25052, 25053, 25060, 25061, 25062, 25063, 25070, 25071, 25072, 25073, 25080, 25081, 25082, 25083, 25090, 25091, 25092, 25093, 3572, 36201, 36202, 36203, 36204, 36205, 36206, 36207, 36641	E0821, E0822, E0829, E08311, E08319, E08321, E08329, E08331, E08339, E08341, E08349, E08351, E08359, E0836, E0839, E0840, E0841, E0842, E0843, E0844, E0849, E0851, E0852, E0859, E08610, E08618, E08620, E08621, E08622, E08628, E08630, E08638, E08649, E0865, E0869, E088, E0921, E0922, E0929, E09311, E09319, E09321, E09329, E09331, E09339, E09341, E09349, E09351, E09359, E0936, E0939, E0940, E0941, E0942, E0943, E0944, E0949, E0951, E0952, E0959, E09610, E09618, E09620, E09621, E09622, E09628, E09630, E09638, E09649, E0965, E0969, E098, E1021, E1022, E1029, E10311, E10319, E10321, E10329, E10331, E10339, E10341, E10349, E10351, E10359, E1036, E1039, E1040, E1041, E1042, E1043, E1044, E1049, E1051, E1052, E1059, E10610, E10618, E10620, E10621, E10622, E10628, E10630, E10638, E10649, E1065, E1069, E108, E1121, E1122, E1129, E11311, E11319, E11321, E11329, E11331, E11339, E11341, E11349, E11351, E11359, E1136, E1139, E1140, E1141, E1142, E1143, E1144, E1149, E1151, E1152, E1159, E11610, E11618, E11620, E11621, E11622, E11628, E11630, E11638, E11649, E1165, E1169, E118, E1321, E1322, E1329, E13311, E13319, E13321, E13329, E13331, E13339, E13341, E13349, E13351, E13359, E1336, E1339, E1340, E1341, E1342, E1343, E1344, E1349, E1351, E1352, E1359, E13610, E13618, E13620, E13621, E13622, E13628, E13630, E13638, E13649, E1365, E1369, E138
Diabetes w/o Complications	Yes	24900, 24901, 25000, 25001, 25002, 25003, V5867	E089, E099, E109, E119, E139, Z794
Fibrosis of Lung	Yes	135, 4950, 4951, 4952, 4953, 4954, 4955, 4956, 4957, 4958, 4959, 500, 501, 502, 503, 504, 505, 5060, 5061, 5062, 5063, 5064, 5069, 5080, 5081, 515, 5160, 5161, 5162, 51630, 51631, 51632, 51633, 51634, 51635, 51636, 51637, 5164, 5165, 51661, 51662, 51663, 51664, 51669, 5168, 5169, 5171, 5172, 5178, 5183, 5186	B4481, D860, D862, J60, J61, J620, J628, J630, J631, J632, J633, J634, J635, J636, J64, J65, J660, J661, J662, J668, J670, J671, J672, J673, J674, J675, J676, J677, J678, J679, J680, J681, J682, J683, J684, J688, J689, J700, J701, J82, J8401, J8402, J8403, J8409, J8410, J84111, J84112, J84113, J84114, J84115, J84116, J84117, J8417, J842, J8481, J8482, J8483, J84841, J84842, J84843, J84848, J8489, J849, J99, M3213, M3301, M3311, M3321, M3391, M3481, M3502
Heart Arrhythmias	Yes	4260, 4270, 4271, 4272, 42731, 42732, 42781	I442, I470, I471, I472, I479, I480, I481, I482, I483, I484, I4891, I4892, I492, I495

Service Type	Chronic?	Diagnosis Codes (ICD-9-CM)	Diagnosis Codes (ICD-10-CM)
Inflammatory Bowel Disease	Yes	5550, 5551, 5552, 5559, 5560, 5561, 5562, 5563, 5564, 5565, 5566, 5568, 5569	K5000, K50011, K50013, K50014, K50018, K50019, K5010, K50111, K50113, K50114, K50118, K50119, K5080, K50811, K50813, K50814, K50818, K50819, K5090, K50911, K50913, K50914, K50918, K50919, K5100, K51011, K51013, K51014, K51018, K51019, K5120, K51211, K51213, K51214, K51218, K51219, K5130, K51311, K51313, K51314, K51318, K51319, K5140, K51411, K51413, K51414, K51418, K51419, K5150, K51511, K51513, K51514, K51518, K51519, K5180, K51811, K51813, K51814, K51818, K51819, K5190, K51911, K51913, K51914, K51918, K51919, K50012, K50112, K50812, K50912, K51012, K51212, K51312, K51412, K51512, K51812, K51912
Lupus	Yes	0993, 4465, 7100, 7102, 7105, 7108, 7109, 71110, 71111, 71112, 71113, 71114, 71115, 71116, 71117, 71118, 71119, 7144, 71489, 7149, 725	M0230, M02311, M02312, M02319, M02321, M02322, M02329, M02331, M02332, M02339, M02341, M02342, M02349, M02351, M02352, M02359, M02361, M02362, M02369, M02371, M02372, M02379, M0238, M0239, M064, M1200, M12011, M12012, M12019, M12021, M12022, M12029, M12031, M12032, M12039, M12041, M12042, M12049, M12051, M12052, M12059, M12061, M12062, M12069, M12071, M12072, M12079, M1208, M1209, M315, M316, M320, M3210, M3211, M3212, M3213, M3214, M3215, M3219, M328, M329, M3500, M3501, M3502, M3503, M3504, M3509, M351, M353, M355, M358, M359, M368
Major Depressive and Bipolar Disorders	Yes	29600, 29601, 29602, 29603, 29604, 29605, 29606, 29610, 29611, 29612, 29613, 29614, 29615, 29616, 29620, 29621, 29622, 29623, 29624, 29625, 29626, 29630, 29631, 29632, 29633, 29634, 29635, 29636, 29640, 29641, 29642, 29643, 29644, 29645, 29646, 29650, 29651, 29652, 29653, 29654, 29655, 29656, 29660, 29661, 29662, 29663, 29664, 29665, 29666, 2967, 29680, 29681, 29682, 29689, 29690, 29699, E9500, E9501, E9502, E9503, E9504, E9505, E9506, E9507, E9508, E9509, E9510, E9511, E9518, E9520, E9521, E9528, E9529, E9530, E9531, E9538, E9539, E954, E9550, E9551, E9552, E9553, E9554, E9555, E9556, E9557, E9559, E956, E9570, E9571, E9572, E9579, E9580, E9581, E9582, E9583, E9584, E9585, E9586, E9587, E9588, E9589, E959	F3010, F3011, F3012, F3013, F302, F303, F304, F308, F309, F310, F3110, F3111, F3112, F3113, F312, F3130, F3131, F3132, F314, F315, F3160, F3161, F3162, F3163, F3164, F3170, F3171, F3172, F3173, F3174, F3175, F3176, F3177, F3178, F3181, F3189, F319, F322, F323, F332, F333, T1491, T360X2A, T360X2S, T361X2A, T361X2S, T362X2A, T362X2S, T363X2A, T363X2S, T364X2A, T364X2S, T365X2A, T365X2S, T366X2A, T366X2S, T367X2A, T367X2S, T368X2A, T368X2S, T3692XA, T3692XS, T370X2A, T370X2S, T371X2A, T371X2S, T372X2A, T372X2S, T373X2A, T373X2S, T374X2A, T374X2S, T375X2A, T375X2S, T378X2A, T378X2S, T3792XA, T3792XS, T380X2A, T380X2S, T381X2A, T381X2S, T382X2A, T382X2S, T383X2A, T383X2S, T384X2A, T384X2S, T385X2A, T385X2S, T386X2A, T386X2S, T387X2A, T387X2S, T38802A, T38802S, T38812A, T38812S, T38892A, T38892S, T38902A, T38902S, T38992A, T38992S, T39012A, T39012S,

Service Type	Chronic?	Diagnosis Codes (ICD-9-CM)	Diagnosis Codes (ICD-10-CM)
			T39092A, T39092S, T391X2A, T391X2S, T392X2A, T392X2S, T39312A, T39312S, T39392A, T39392S, T394X2A, T394X2S, T398X2A, T398X2S, T3992XA, T3992XS, T400X2A, T400X2S, T401X2A, T401X2S, T402X2A, T402X2S, T403X2A, T403X2S, T404X2A, T404X2S, T405X2A, T405X2S, T40602A, T40602S, T40692A, T40692S, T407X2A, T407X2S, T408X2A, T408X2S, T40902A, T40902S, T40992A, T40992S, T410X2A, T410X2S, T411X2A, T411X2S, T41202A, T41202S, T41292A, T41292S, T413X2A, T413X2S, T4142XA, T4142XS, T415X2A, T415X2S, T420X2A, T420X2S, T421X2A, T421X2S, T422X2A, T422X2S, T423X2A, T423X2S, T424X2A, T424X2S, T425X2A, T425X2S, T426X2A, T426X2S, T4272XA, T4272XS, T428X2A, T428X2S, T43012A, T43012S, T43022A, T43022S, T431X2A, T431X2S, T43202A, T43202S, T43212A, T43212S, T43222A, T43222S, T43292A, T43292S, T433X2A, T433X2S, T434X2A, T434X2S, T43502A, T43502S, T43592A, T43592S, T43602A, T43602S, T43612A, T43612S, T43622A, T43622S, T43632A, T43632S, T43692A, T43692S, T438X2A, T438X2S, T4392XA, T4392XS, T440X2A, T440X2S, T441X2A, T441X2S, T442X2A, T442X2S, T443X2A, T443X2S, T444X2A, T444X2S, T445X2A, T445X2S, T446X2A, T446X2S, T447X2A, T447X2S, T448X2A, T448X2S, T44902A, T44902S, T44992A, T44992S, T450X2A, T450X2S, T451X2A, T451X2S, T452X2A, T452X2S, T453X2A, T453X2S, T454X2A, T454X2S, T45512A, T45512S, T45522A, T45522S, T45602A, T45602S, T45612A, T45612S, T45622A, T45622S, T45692A, T45692S, T457X2A, T457X2S, T458X2A, T458X2S, T4592XA, T4592XS, T460X2A, T460X2S, T461X2A, T461X2S, T462X2A, T462X2S, T463X2A, T463X2S, T464X2A, T464X2S, T465X2A, T465X2S, T466X2A, T466X2S, T467X2A, T467X2S, T468X2A, T468X2S, T46902A, T46902S, T46992A, T46992S, T470X2A, T470X2S, T471X2A, T471X2S, T472X2A, T472X2S, T473X2A, T473X2S, T474X2A, T474X2S, T475X2A, T475X2S, T476X2A, T476X2S, T477X2A, T477X2S, T478X2A, T478X2S, T4792XA, T4792XS, T480X2A, T480X2S, T481X2A, T481X2S, T48202A, T48202S, T48292A, T48292S, T483X2A, T483X2S, T484X2A, T484X2S, T485X2A, T485X2S, T486X2A, T486X2S, T48902A, T48902S, T48992A, T48992S,

Service Type	Chronic?	Diagnosis Codes (ICD-9-CM)	Diagnosis Codes (ICD-10-CM)
			T490X2A, T490X2S, T491X2A, T491X2S, T492X2A, T492X2S, T493X2A, T493X2S, T494X2A, T494X2S, T495X2A, T495X2S, T496X2A, T496X2S, T497X2A, T497X2S, T498X2A, T498X2S, T4992XA, T4992XS, T500X2A, T500X2S, T501X2A, T501X2S, T502X2A, T502X2S, T503X2A, T503X2S, T504X2A, T504X2S, T505X2A, T505X2S, T506X2A, T506X2S, T507X2A, T507X2S, T508X2A, T508X2S, T50902A, T50902S, T50992A, T50992S, T50A12A, T50A12S, T50A22A, T50A22S, T50A92A, T50A92S, T50B12A, T50B12S, T50B92A, T50B92S, T50Z12A, T50Z12S, T50Z92A, T50Z92S, T510X2A, T510X2S, T511X2A, T511X2S, T512X2A, T512X2S, T513X2A, T513X2S, T518X2A, T518X2S, T5192XA, T5192XS, T520X2A, T520X2S, T521X2A, T521X2S, T522X2A, T522X2S, T523X2A, T523X2S, T524X2A, T524X2S, T528X2A, T528X2S, T5292XA, T5292XS, T530X2A, T530X2S, T531X2A, T531X2S, T532X2A, T532X2S, T533X2A, T533X2S, T534X2A, T534X2S, T535X2A, T535X2S, T536X2A, T536X2S, T537X2A, T537X2S, T5392XA, T5392XS, T540X2A, T540X2S, T541X2A, T541X2S, T542X2A, T542X2S, T543X2A, T543X2S, T5492XA, T5492XS, T550X2A, T550X2S, T551X2A, T551X2S, T560X2A, T560X2S, T561X2A, T561X2S, T562X2A, T562X2S, T563X2A, T563X2S, T564X2A, T564X2S, T565X2A, T565X2S, T566X2A, T566X2S, T567X2A, T567X2S, T56812A, T56812S, T56892A, T56892S, T5692XA, T5692XS, T570X2A, T570X2S, T571X2A, T571X2S, T572X2A, T572X2S, T573X2A, T573X2S, T578X2A, T578X2S, T5792XA, T5792XS, T5802XA, T5802XS, T5812XA, T5812XS, T582X2A, T582X2S, T588X2A, T588X2S, T5892XA, T5892XS, T590X2A, T590X2S, T591X2A, T591X2S, T592X2A, T592X2S, T593X2A, T593X2S, T594X2A, T594X2S, T595X2A, T595X2S, T596X2A, T596X2S
Multiple Sclerosis	Yes	340, 3410, 3411	G35, G360, G370, G375
Personality Disorder	Yes	30012, 30013, 30014, 30015, 3006, 3010, 30110, 30111, 30112, 30113, 30120, 30121, 30122, 3013, 3014, 30150, 30151, 30159, 3016, 3017, 30181, 30182, 30183, 30184, 30189, 3019	F21, F440, F441, F4481, F481, F600, F601, F602, F603, F604, F605, F606, F607, F6081, F6089, F609
Rheumatoid Arthritis	Yes	1361, 4460, 4461, 44620, 44621, 44629, 4463, 4464, 4466, 4467, 6960, 7101, 7103, 7104, 71120, 71121, 71122, 71123, 71124, 71125, 71126, 71127, 71128, 71129, 7140,	L4050, L4051, L4052, L4053, L4054, L4059, M0500, M05011, M05012, M05019, M05021, M05022, M05029, M05031, M05032, M05039, M05041, M05042, M05049,

Service Type	Chronic?	Diagnosis Codes (ICD-9-CM)	Diagnosis Codes (ICD-10-CM)
		7141, 7142, 71430, 71431, 71432, 71433, 71481, 7200	M05051, M05052, M05059, M05061, M05062, M05069, M05071, M05072, M05079, M0509, M0510, M05111, M05112, M05119, M05121, M05122, M05129, M05131, M05132, M05139, M05141, M05142, M05149, M05151, M05152, M05159, M05161, M05162, M05169, M05171, M05172, M05179, M0519, M0520, M05211, M05212, M05219, M05221, M05222, M05229, M05231, M05232, M05239, M05241, M05242, M05249, M05251, M05252, M05259, M05261, M05262, M05269, M05271, M05272, M05279, M0529, M0530, M05311, M05312, M05319, M05321, M05322, M05329, M05331, M05332, M05339, M05341, M05342, M05349, M05351, M05352, M05359, M05361, M05362, M05369, M05371, M05372, M05379, M0539, M0540, M05411, M05412, M05419, M05421, M05422, M05429, M05431, M05432, M05439, M05441, M05442, M05449, M05451, M05452, M05459, M05461, M05462, M05469, M05471, M05472, M05479, M0549, M0550, M05511, M05512, M05519, M05521, M05522, M05529, M05531, M05532, M05539, M05541, M05542, M05549, M05551, M05552, M05559, M05561, M05562, M05569, M05571, M05572, M05579, M0559, M0560, M05611, M05612, M05619, M05621, M05622, M05629, M05631, M05632, M05639, M05641, M05642, M05649, M05651, M05652, M05659, M05661, M05662, M05669, M05671, M05672, M05679, M0569, M0570, M05711, M05712, M05719, M05721, M05722, M05729, M05731, M05732, M05739, M05741, M05742, M05749, M05751, M05752, M05759, M05761, M05762, M05769, M05771, M05772, M05779, M0579, M0580, M05811, M05812, M05819, M05821, M05822, M05829, M05831, M05832, M05839, M05841, M05842, M05849, M05851, M05852, M05859, M05861, M05862, M05869, M05871, M05872, M05879, M0589, M059, M0600, M06011, M06012, M06019, M06021, M06022, M06029, M06031, M06032, M06039, M06041, M06042, M06049, M06051, M06052, M06059, M06061, M06062, M06069, M06071, M06072, M06079, M0608, M0609, M061, M0620, M06211, M06212, M06219, M06221, M06222, M06229, M06231, M06232, M06239, M06241,

Service Type	Chronic?	Diagnosis Codes (ICD-9-CM)	Diagnosis Codes (ICD-10-CM)
			M06242, M06249, M06251, M06252, M06259, M06261, M06262, M06269, M06271, M06272, M06279, M0628, M0629, M0630, M06311, M06312, M06319, M06321, M06322, M06329, M06331, M06332, M06339, M06341, M06342, M06349, M06351, M06352, M06359, M06361, M06362, M06369, M06371, M06372, M06379, M0638, M0639, M0680, M06811, M06812, M06819, M06821, M06822, M06829, M06831, M06832, M06839, M06841, M06842, M06849, M06851, M06852, M06859, M06861, M06862, M06869, M06871, M06872, M06879, M0688, M0689, M069, M0800, M08011, M08012, M08019, M08021, M08022, M08029, M08031, M08032, M08039, M08041, M08042, M08049, M08051, M08052, M08059, M08061, M08062, M08069, M08071, M08072, M08079, M0808, M0809, M081, M0820, M08211, M08212, M08219, M08221, M08222, M08229, M08231, M08232, M08239, M08241, M08242, M08249, M08251, M08252, M08259, M08261, M08262, M08269, M08271, M08272, M08279, M0828, M0829, M083, M0840, M08411, M08412, M08419, M08421, M08422, M08429, M08431, M08432, M08439, M08441, M08442, M08449, M08451, M08452, M08459, M08461, M08462, M08469, M08471, M08472, M08479, M0848, M0880, M08811, M08812, M08819, M08821, M08822, M08829, M08831, M08832, M08839, M08841, M08842, M08849, M08851, M08852, M08859, M08861, M08862, M08869, M08871, M08872, M08879, M0888, M0889, M0890, M08911, M08912, M08919, M08921, M08922, M08929, M08931, M08932, M08939, M08941, M08942, M08949, M08951, M08952, M08959, M08961, M08962, M08969, M08971, M08972, M08979, M0898, M0899, M300, M301, M302, M303, M308, M310, M311, M312, M3130, M3131, M314, M317, M3300, M3301, M3302, M3309, M3310, M3311, M3312, M3319, M3320, M3321, M3322, M3329, M3390, M3391, M3392, M3399, M340, M341, M342, M3481, M3482, M3483, M3489, M349, M352, M360, M450, M451, M452, M453, M454, M455, M456, M457, M458, M459, M488X1, M488X2, M488X3, M488X4, M488X5, M488X6, M488X7, M488X8, M488X9

Service Type	Chronic?	Diagnosis Codes (ICD-9-CM)	Diagnosis Codes (ICD-10-CM)
Seizures	Yes	1361, 4460, 4461, 44620, 44621, 44629, 4463, 4464, 4466, 4467, 6960, 7101, 7103, 7104, 71120, 71121, 71122, 71123, 71124, 71125, 71126, 71127, 71128, 71129, 7140, 7141, 7142, 71430, 71431, 71432, 71433, 71481, 7200	G40001, G40009, G40011, G40019, G40101, G40109, G40111, G40119, G40201, G40209, G40211, G40219, G40301, G40309, G40311, G40319, G40401, G40409, G40411, G40419, G40501, G40509, G40801, G40802, G40803, G40804, G40811, G40812, G40813, G40814, G40821, G40822, G40823, G40824, G4089, G40901, G40909, G40911, G40919, G40A01, G40A09, G40A11, G40A19, G40B01, G40B09, G40B11, G40B19, P90, R5600, R5601, R561, R569
Sepsis and Shock	No	0031, 0202, 0223, 0362, 0380, 03810, 03811, 03812, 03819, 0382, 0383, 03840, 03841, 03842, 03843, 03844, 03849, 0388, 0389, 04082, 0545, 77181, 78552, 78559, 99590, 99591, 99592, 99593, 99594	A021, A207, A227, A267, A327, A392, A393, A394, A400, A401, A403, A408, A409, A4101, A4102, A411, A412, A413, A414, A4150, A4151, A4152, A4153, A4159, A4181, A4189, A419, A427, A483, A5486, B007, B377, P360, P3610, P3619, P362, P3630, P3639, P364, P365, P368, P369, R571, R578, R6510, R6511, R6520, R6521
Thyroid Cancer	Yes	1720, 1721, 1722, 1723, 1724, 1725, 1726, 1727, 1728, 1729, 1860, 1869, 1871, 1872, 1873, 1874, 1875, 1876, 1877, 1878, 1879, 193, 1941, 1945, 1946, 1948, 1949, 1991, 23770, 23771, 23772, 23773, 23779, 2592	C430, C4310, C4311, C4312, C4320, C4321, C4322, C4330, C4331, C4339, C434, C4351, C4352, C4359, C4360, C4361, C4362, C4370, C4371, C4372, C438, C439, C600, C601, C602, C608, C609, C6200, C6201, C6202, C6210, C6211, C6212, C6290, C6291, C6292, C6300, C6301, C6302, C6310, C6311, C6312, C632, C637, C638, C639, C73, C750, C754, C755, C758, C759, C801, D030, D0310, D0311, D0312, D0320, D0321, D0322, D0330, D0339, D034, D0351, D0352, D0359, D0360, D0361, D0362, D0370, D0371, D0372, D038, D039, E340, Q8500, Q8501, Q8502, Q8503, Q8509
Unstable Angina	No	41000, 41002, 41010, 41012, 41020, 41022, 41030, 41032, 41040, 41042, 41050, 41052, 41060, 41062, 41070, 41072, 41080, 41082, 41090, 41092, 4110, 4111, 41181, 41189	I200, I230, I231, I232, I233, I236, I237, I238, I240, I241, I248, I249, I25110, I25700, I25710, I25720, I25730, I25750, I25760, I25790
Viral Meningitis	No	0470, 0471, 0478, 0479, 048, 0490, 0491, 0530, 05472, 0721, 3212, 3220, 3221, 3222, 3229	A870, A871, A872, A878, A879, A880, B003, B010, B021, B051, B0602, B261, B2702, B2712, B2782, B2792, D8681, G030, G031, G032, G038, G039

When assigning HCCs, I exclude diagnoses associated with the following place of service and procedure codes, due to their high potential for false positive diagnoses:

Excluded ranges		
Place of Service Codes		
	12	Private residence home
	31	Skilled nursing facility
	32	Nursing home
	33	Custodial care
	34	Hospice
	41	Ambulance – land
	42	Ambulance – other
	65	Renal dialysis
	81	Independent lab
	99	Unknown
Procedure Codes		
	36415-36416	Drawing blood
	70000-76999	X-ray and ultrasound
	78000-78999	Imaging
	80000-87999	Lab tests
	88000-88099	Autopsy
	88104-88299	Cytopathology
	88300-88399	Surgical Pathology
	88720-88741	In Vivo
	92551-92569	Hearing tests
	93000-93350	ECG and ultrasound
	99000-99001	Specimen handling
	A0021-A0999	Ambulance
	A4206-A999	Medical and surgical supplies
	B4304-B999	Enteral Supplies
	G0001	Drawing blood
	E0100-E9999	Durable medical equipment
	K0001-K9999	Wheelchairs and accessories
	L0100-L4599	Orthotics
	L5000-L9900	Prosthetics
	P2028-P9999	Pathology and Lab
	R0070-R0076	Radiology

Table 7 identifies additional demographic information, as well as illustrating the balance in my sample across households with and without a chronic condition in the family. The table also shows the frequency of the various chronic conditions utilized in my sample. Households

with chronic conditions are not markedly different in terms of age or sex composition or family size, but do incur significantly higher medical costs in a year. They are not, however, more likely to switch insurance plans from year to year. There is wide variation in the onset of chronic illnesses, the three most common illnesses are asthma, major depressive disorder, and diabetes.

	Full Sample	Households with chronic conditions
Demographics & Utilization		
Enrollee age	30.87 (0.008)	29.61 (0.046)
% female enrollees	50.17 (0.000)	50.46 (0.001)
Mean [median] total spending	\$2,504.41 [\$679.75] (4.510)	\$3,378.17 [\$957.52] (23.752)
Mean [median] OOP spending	\$443.07 [\$109.66] (0.525)	\$531.93 [\$151.18] (3.153)
% switching plans ever	—	
Incidence of chronic illness (per 1,000 individuals)		
Adrenal & pituitary disorders	0.22	7.35
Asthma	2.93	96.08
Breast/prostate cancer	0.35	11.58
Chronic hepatitis	0.10	3.23
Chronic skin condition	0.23	7.46
Congestive heart failure	0.14	4.52
Diabetes with complications	0.39	12.72
Diabetes without complications	1.18	38.57
Fibrosis of lung	0.46	15.10
Heart arrhythmias	0.00	0.00
Inflammatory bowel disease	0.14	4.65
Lupus	0.16	5.20
Major depressive/bipolar disorder	1.62	52.76
Multiple sclerosis	1.10	36.17
Personality disorder	0.09	2.81
Rheumatoid arthritis	0.17	5.70
Seizures	0.30	9.82
Thyroid cancer	0.14	4.69
N_{families}	353,403	52,747
$N_{\text{individuals}}$	1,087,353	165,694

Table 7. Relative Incidence of Chronic Conditions

A.3 Identifying Chronic Care Costs

An important component of my model is that chronic illnesses correspond to annual diagnostic and maintenance costs that are not strictly choice variables, in the sense that certain health utilization is more or less required. I identify the costs associated with these illnesses from the claims data as procedures which have the major diagnosis listed on that line item. Additionally, in conjunction with Rebecca Hughes, MD, I identify specific therapeutic classes for prescription medications that are associated with treating each chronic condition, shown in the table below. Empirical distributions of these estimated diagnostic and maintenance costs for each major health event are available upon request.

Major Health Event	Therapeutic Classes
Breast and Prostate Cancer	Antineoplastic Agents Hematopoietic Agents Antiemetics Adrenals Androgens Immunosuppresants Antiinf S/MM, Antibiotics & Comb Antiinf S/MM, Antivirals & Comb Antiinf S/MM, Antifungals & Comb Antiinf S/MM, Scabic/Pediculic Antiinf S/MM, Antiinf Local Misc Antineoplastics S/MM Phosphodiesterase Inhibitors Hormone-Modifying Therapy Molecular Targeted Therapy
Thyroid Cancer	Antineoplastic Agents Antihyperlipidemic Drugs Antiemetics Adrenals Androgens Thy/Antithy, Thyroid Hormones Thy/Anithy, Anithyroid Hormones Immunosuppresants Antineoplastics S/MM Phosphodiesterase Inhibitors
Diabetes (w/ or w/o Complications)	Diabetes Mell/Diab Supply, NEC Antidiabetic Agents, Insulin Antidiabetic Ag, Sulfonylureas Antidiabetic Agents, Misc Antidiabetic Ag, Meglitinides Antidiabetic Ag, SGLT Inhibit Antidiabetic Ag, TZD
Adrenal/Pituitary Disorders	Adrenals Androgens Estrogens & Comb Parathyroid Hormones Pituitary Hormones Progestins Thy/Antithy, Thyroid Hormones Thy/Antithy, Antithyroid Hormones
Chronic Hepatitis	Antivirals Adrenals
Inflammatory Bowel Disease	Antineoplastic Agents Gastrointestinal Drugs Misc. Adrenals Immunosuppresants

Major Health Event	Therapeutic Classes
Rheumatoid Arthritis	Antineoplastic Agents Adrenals Immunosuppressants
Lupus	Quinolones, NEC Adrenals
MDD and Bipolar	Stimulant, Non-Amphetamine Anticonvulsants, Misc. Psychotherapeutics, Antidepressants Psychotherapeutics, Tranq/Antipsychotics Stimulant, Amphetamine Stimulant, Non-Amphetamine ASH, Barbiturates ASH, Benzodiazepines Anxiolytic/Sedative/Hypnotic Antimaniac Agents
Personality Disorder	Stimulant, Non-Amphetamine Anticonvulsants, Misc. Psychotherapeutics, Antidepressants Psychotherapeutics, Tranq/Antipsychotics Stimulant, Amphetamine Stimulant, Non-Amphetamine ASH, Barbiturates ASH, Benzodiazepines Anxiolytic/Sedative/Hypnotic Antimaniac Agents
Multiple Sclerosis	Sympathomimetic Agents Stimulant, Amphetamine Stimulant, Non-Amphetamine Adrenals
Seizures	Anticonvulsants, Misc.
Congestive Heart Failure	Cardiac Drugs, NEC Cardiac, ACE Inhibitors Cardiac, Alpha-Beta Blockers Cardiac, Beta Blockers Cardiac, Calcium Channel Antihyperlipidemic Drugs Repl Preps, Potassium Supp Diuretics, Loop Diuretics Diuretics, Misc. Diuretics, Osmotic Diuretics, Potassium-Sparing Diuretics, Thiazides Diuretics, Carb Anhydrase Inhib
Heart Arrhythmias	Coag/Anticoag, Anticoagulants Coag/Anticoag, Antiheparin Agents Coag/Anticoag, Hemostatics

Major Health Event	Therapeutic Classes
	Cardiac Drugs, NEC Cardiac, ACE Inhibitors Cardiac, Alpha-Beta Blockers Cardiac, Beta Blockers Cardiac, Calcium Channel Antihyperlipidemic Drugs
Asthma	Sympathomimetic Agents Adrenals Leukotriene Modifiers
Fibrosis of Lung	Sympathomimetic Agents Adrenals
Chronic Skin Ulcer	Adrenals

A.4 Identifying Cardiovascular Preventive Medications

Cardiovascular preventive medications are identified using the following set of therapeutic classes

Therapeutic Class	Example Medications
Angiotensin-converting-enzyme (ACE) Inhibitors	benazepril (Lotensin), zofenopril, perindopril
Anticoagulants	warfarin (Coumadin), heparin
Antihyperlipidemic Agents	atorvastatin (Lipitor), fluvastatin, lovastatin
Beta Blockers	propranolol (Inderal), pronethalol
Hypotensive Agents	midodrine (Amatine), norepinephrine

Table 8. Therapeutic Classes Used in Identifying Cardiovascular Preventive Medications

A.5 Identifying Low-Value Services

Low value services are identified at the procedure level using CPT codes for medical procedures and therapeutic classes for prescription medications. I aggregate these services into five broad categories, as illustrated in the following table.

Category	Service	CPT Codes / Therapeutic Classes	Additional restrictions (age/sex restrictions, diagnosis or procedure codes)
All Pediatric	Vitamin D Screening	82306,82652	Age < 18
All Pediatric	Cervical Cancer Screening	87620,87621,87622, 87623, 87624, 87625, 88141, 88142, 88143, 88147, 88148, 88150, 88152, 88153, 88154, 88155, 88164, 88165,88166, 88167, 88174, 88175, G0123, G0124, G0141, G0143, G0144, G0145, G0147, G0148, P3000, P3001, Q0091	Age < 18, age >= 14, female
All Pediatric	Head imaging for headache	70450,70460,70470,70551,70552,70553	Age < 18, Diagnosis codes: 3390, 3391, 3460, 3461, 3462, 3464, 3465, 3467, 3468, 3469, 7840, 3393, G440, G441, G442, G444, G430, G431, G435, G437, G438, G439, 30781,33983, 33984, 33985, R51, R510, R519, G4483, G4484, G4485
All Pediatric	Antibiotics for upper respiratory infections	Antibiotics (multiple classes)	Diagnosis codes: 460,465, J00, J06, H65, H60, H61, H62, 3810, 3814
All Pediatric	Antibiotics for bronchiolitis	Antibiotics (multiple classes)	Diagnosis codes: 46611,46619, J210, J218
All Pediatric	Cough or cold medicine	Antitussives, Expectorants, Mucolytics, Cough/Cold Combinations	Age < 6
Adult Drugs	Opioids to treat migraines	Opiate Agonists, Opiate Part Agonists, Opiate Antagonists	Diagnosis codes: 346**, G43**
Adult Imaging	Head imaging for headache	70450,70460,70470,70551,70552,70553	Diagnosis codes: 3390, 3391, 3460, 3461, 3462, 3464, 3465, 3467, 3468, 3469, 7840, 3393, G440, G441, G442, G444, G430, G431, G435, G437, G438, G439, 30781,33983, 33984, 33985, R51, R510, R519, G4483, G4484, G4485
Adult Imaging	Imaging for lower-back pain	72010, 72020,72052, 72100, 72110, 72114,72120, 72200, 72202, 72220, 72131, 72132, 72133, 72141, 72142, 72146, 72147, 72148,72149, 72156, 72157, 72158	Diagnosis codes: 7213, 7226, 7242, 7243, 7244,7245, 7246,7385, 7393,7394, 8460, 8461, 8462, 8463, 8468, 8469, 8472, M432, M512, M513, M518, M533, M545, M541, M543, M998, 72190, 72210, 72252, 72293, 72402,72470, 72471, 72479, M47817, M532X7, M9903, M9904, S338XXA, S336XXA, S339XXA, S335XXA, M47819, M4647, M4806, M532X8

Category	Service	CPT Codes / Therapeutic Classes	Additional restrictions (age/sex restrictions, diagnosis or procedure codes)
Adult Imaging	Screening for carotid artery disease	36222, 36223, 36224, 70498, 70547, 70548, 70549, 93880, 93882, 3100F	Diagnosis codes: 430, 431, 434, 436, 781, I63, I66, R25, R26, R27, R29, R47, G45, H34, R55, R20, 4350, 4351, 4353, 4358, 359, 3623, 7802, 7820, I609, I619, 43301, 43311, 43321, 43331, 43381, 43391, 99702, V1254, 36284, 78451, 78452, 78459, I6789, I67848, I97811, I97821, Z8673, H3582
Adult Imaging	Cardiac imaging	0144T, 0145T, 0146T, 0147T, 0148T, 0149T, 0150T, 75552, 75553, 75554, 75555, 75556, 75557, 75558, 75559, 75561, 75562, 75565, 75571, 75572, 75573, 75574, 78451, 78452, 78453, 78454, 78460, 78461, 78464, 78465, 78478, 78480, 78459, 78481, 78483, 78491, 78492, 78494, 78496, 78499	
Adult Screening	Vitamin D Screening	82306, 82652	
Adult Screening	Cardiac testing for low-risk patients	93015, 93016, 93017, 93018, 93350, 93351, 78451, 78452, 78453, 78454, 78460, 78461, 78464, 78465, 78472, 78473, 78481, 78483, 78491, 78492, 93303, 93304, 93306, 93307, 93308, 93312, 93315, 93318, 3120F, 93000, 93005, 93010, G0366, G0367, G0368, G0403, G0404, G0405	
Adult Screening	Pre-operative testing before low-risk surgery	71010, 71015, 71020, 71021, 71022, 71023, 71030, 71034, 71035, 93303, 93304, 93306, 93307, 93308, 93312, 93315, 93318, 94010, 78451, 78452, 78453, 78454, 78460, 78461, 78464, 78465, 78472, 78473, 78481, 78483, 78491, 78492, 93015, 93016, 93017, 93018, 93350, 93351	Procedure codes for surgery: 19120, 19125, 47562, 47563, 49560, 58558
Adult Surgery	Arthroscopic surgery for knee osteoarthritis	29877, 29879, G0289	Diagnosis codes: 8360, 8361, 8362, 7170, S832, 71741, M23202, M23205

B Additional Reduced Form Results

B.1 Robustness of Results to Transformations

Table 10 demonstrates that results are robust to two standard transformations for skewed spending variables: the inverse hyperbolic sine transform, as reported in the main text, and the $\log(y + 1)$ transformation.

B.2 Robustness of Results to Event Study Specification

Table 9 shows the standard difference-in-differences coefficients for each of the main event study regressions performed in the main text.

Outcome Variable	Treated _f × Post _t	Adjusted R^2	N
OOP, chronic, full sample	0.09*** (0.012)	0.51	1,538,162
OOP, chronic, zero-deductible plans	0.13*** (0.020)	0.55	390,335
OOP, acute, full sample	0.42*** (0.031)	0.50	1,374,481
OOP, acute, zero-deductible plans	0.39*** (0.063)	0.54	358,860
Billed spending, wellness visits, full sample	0.13*** (0.013)	0.43	1,538,162
Billed spending, wellness, zero-deductible plans	0.18*** (0.027)	0.40	390,335
Cardiovascular Prescriptions, Prob(fill scrip)	2.56 (1.501)	0.42	439,542
Cardiovascular Prescriptions, PDC	1.46 (1.142)	0.48	439,542
Billed Spending, Low Value Services	0.06*** (0.011)	0.20	1,538,162
Utilization, Low Value Services	0.03*** (0.008)	0.20	1,538,162

Table 9. Difference in Differences Coefficients, Main Regressions

Table 11 implements the robust alternative event study estimator described by de Chaisemartin and D'Haultfoeuille (2019).

	OOP, chronic diagnosis		OOP, acute diagnosis		Wellness spending		Low-value spending	
	$\sinh^{-1}(y)$	$\log(y+1)$	$\sinh^{-1}(y)$	$\log(y+1)$	$\sinh^{-1}(y)$	$\log(y+1)$	$\sinh^{-1}(y)$	$\log(y+1)$
$t-5$	-0.02 (0.028)	-0.02 (0.026)	-0.11 (0.070)	-0.10 (0.064)	-0.09** (0.031)	-0.08** (0.028)	-0.06* (0.033)	-0.05* (0.03)
$t-4$	0.02 (0.024)	0.01 (0.022)	-0.11 (0.059)	-0.10 (0.055)	-0.03 (0.026)	-0.03 (0.024)	-0.04 (0.028)	-0.03 (0.024)
$t-3$	0.00 (0.020)	0.00 (0.018)	-0.02 (0.052)	-0.02 (0.048)	-0.02 (0.022)	-0.02 (0.020)	-0.03 (0.023)	-0.02 (0.021)
$t-2$	-0.00 (0.017)	-0.00 (0.015)	-0.07 (0.045)	-0.06 (0.042)	-0.03 (0.019)	-0.03 (0.017)	-0.01 (0.020)	-0.01 (0.018)
$t-1$	—	—	—	—	—	—	—	—
t	0.08*** (0.014)	0.07*** (0.013)	-0.01 (0.041)	-0.01 (0.037)	0.12*** (0.016)	0.11*** (0.015)	0.05* (0.018)	0.04* (0.016)
$t+1$	0.10*** (0.016)	0.10*** (0.014)	0.10* (0.047)	0.09* (0.043)	0.09*** (0.017)	0.08*** (0.016)	0.05** (0.019)	0.04** (0.017)
$t+2$	0.10*** (0.018)	0.09*** (0.017)	0.06 (0.055)	0.07 (0.050)	0.10*** (0.020)	0.10*** (0.018)	0.05* (0.021)	0.04* (0.019)
$t+3$	0.09*** (0.018)	0.08*** (0.019)	0.10 (0.062)	0.09 (0.057)	0.11*** (0.022)	0.10*** (0.020)	0.04 (0.024)	0.04 (0.021)
$t+4$	0.08*** (0.025)	0.08*** (0.022)	0.14 (0.074)	0.13 (0.068)	0.13*** (0.025)	0.12*** (0.023)	0.09** (0.028)	0.08** (0.024)
$t+5$	0.07*** (0.030)	0.06* (0.028)	0.12 (0.088)	0.12 (0.081)	0.10*** (0.030)	0.09*** (0.027)	0.12*** (0.033)	0.11*** (0.029)
R^2	0.51	0.52	0.50	0.51	0.43	0.44	0.20	0.20
N	1,538,161	1,538,161	1,374,359	1,374,359	1,538,161	1,538,161	1,538,161	1,538,161

Notes: This table presents estimates for the main event study regression results reported in the paper. The first column of each pair of results are the results shown graphically in the text, while the second column uses the log transformation. Standard errors are clustered at the household level.

Table 10. Robustness: Inverse Hyperbolic Sine & Log Transformations

	OOP spending, chronic		OOP spending, acute		Billed spending, wellness		Prescriptions, PDC		Billed Spending, low-value care	
	Standard	Robust	Standard	Robust	Standard	Robust	Standard	Robust	Standard	Robust
t	0.08*** (0.014)	0.06*** (0.013)	-0.01 (0.041)	-0.02 (0.039)	0.12*** (0.016)	0.11*** (0.02)	5.96*** (1.100)	1.33 (1.013)	0.05* (0.018)	0.04** (0.016)
$t + 1$	0.10*** (0.016)	0.08*** (0.016)	0.10* (0.047)	0.06 (0.043)	0.09*** (0.017)	0.07*** (0.018)	2.82*** (1.189)	2.24** (1.137)	0.05*** (0.019)	0.04** (0.016)
$t + 2$	0.10*** (0.018)	0.06*** (0.018)	0.06 (0.055)	0.03 (0.055)	0.10*** (0.020)	0.07*** (0.019)	-1.97 (1.397)	0.12 (1.444)	0.05* (0.021)	0.04* (0.021)
$t + 3$	0.09*** (0.018)	0.04** (0.021)	0.10 (0.062)	0.04 (0.063)	0.11*** (0.022)	0.06*** (0.021)	-3.95* (1.701)	0.46 (1.949)	0.04 (0.024)	0.02 (0.022)
$t + 4$	0.08*** (0.025)	0.02 (0.025)	0.14 (0.074)	0.11 (0.09)	0.13*** (0.025)	0.07** (0.021)	-6.67*** (2.051)	-2.05 (2.685)	0.09** (0.028)	0.06** (0.028)
$t + 5$	0.07*** (0.030)	-0.02 (0.031)	0.12 (0.088)	0.12 (0.107)	0.10*** (0.030)	0.02 (0.034)	-5.42* (2.375)	-0.20 (3.461)	0.12*** (0.033)	0.11*** (0.038)
N	1,538,161	1,538,161	1,374,359	1,374,359	1,538,161	1,538,161	439,542	439,542	1,538,161	

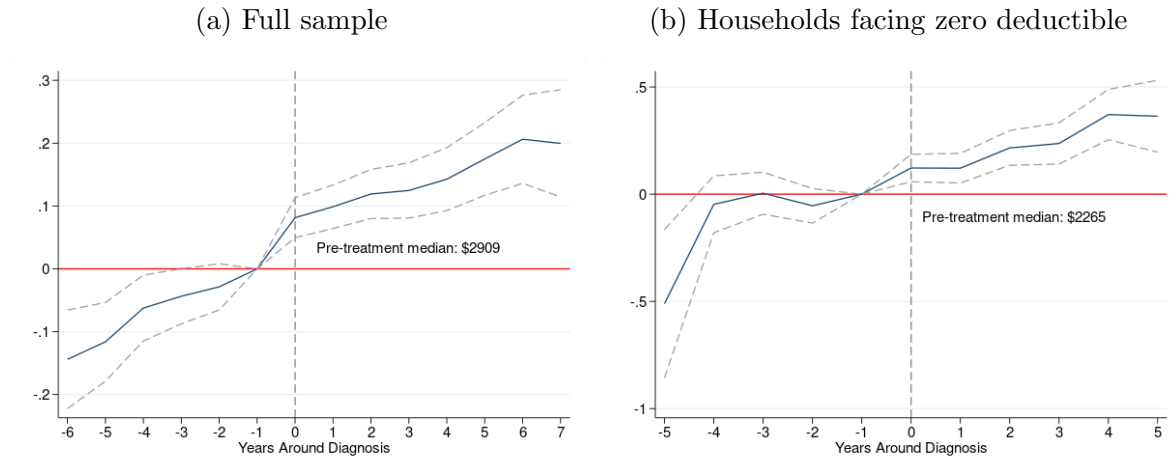
Notes: This table compares regression results from the typical two-way fixed effects event study regression and the robust alternative estimator proposed by de Chaisemartin and D'Haultfoeuille (2019). Estimation is performed using the `did_multiplegt` package in Stata (note that pre-trends are not estimated using this command, hence not reported). Standard errors clustered at the household level are reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table 11. Model Comparison: Event Studies and DID_M

B.3 Household Response to Major Medical Events

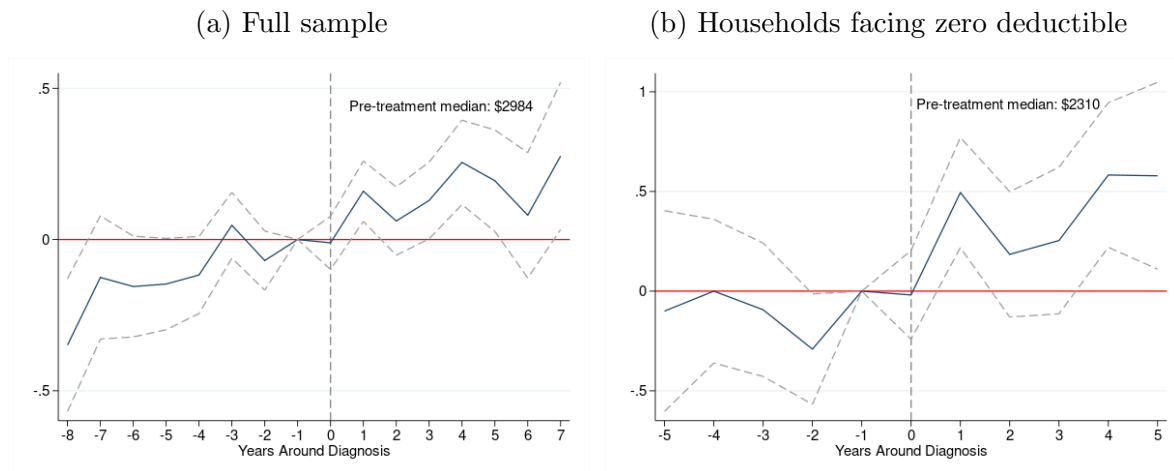
In this section, I include additional results from a suite of two-way fixed effects models estimating the causal effect of major medical events on health behaviors. Figures 12 and 13 illustrate the estimated effect on billed spending for both chronic and acute medical events.

Figure 12. Estimated Effect of a Chronic Diagnosis on Billed Non-Diagnosed Spending



Note: Dependent variable is the inverse hyperbolic sine of total billed spending for all non-diagnosed individuals in a household. Spending is measured in 2020 USD. Standard errors are clustered at the household level.

Figure 13. Estimated Effect of an Acute Health Event on Billed Non-Diagnosed Spending

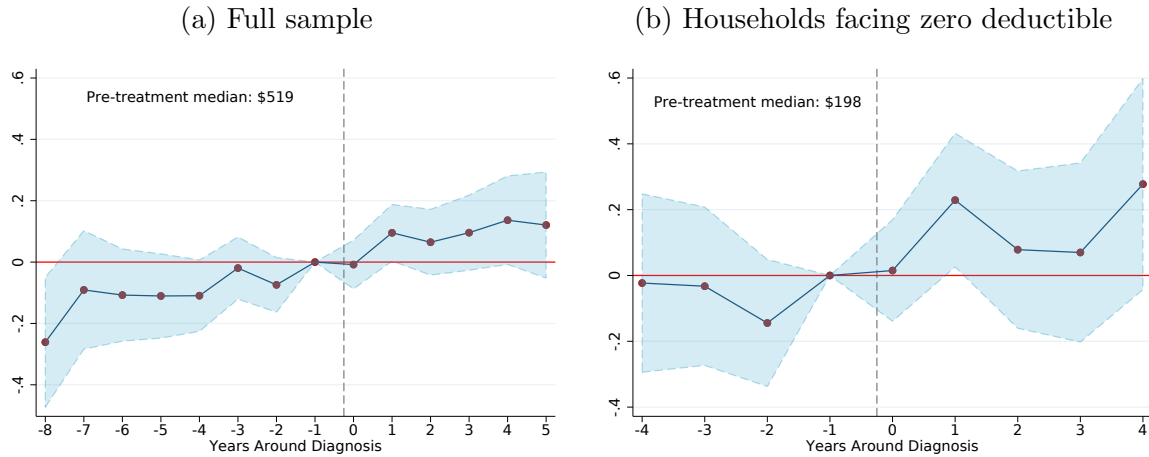


Note: Dependent variable is the inverse hyperbolic sine of total billed spending for all non-diagnosed individuals in a household. Spending is measured in 2020 USD. Standard errors are clustered at the household level.

I also explore the effect of acute health events on household out-of-pocket spending,

similar to Figure 1 in the text. In general, acute events do not generate the same household response that chronic diagnoses do.

Figure 14. Acute Health Events Have no Effect on Non-Diagnosed OOP Spending



Notes: These figures show estimated coefficients and 95% confidence intervals for the effect of an acute hospitalization within the household on medical spending for different populations. For each panel, the dependent variable is the inverse hyperbolic sine of total OOP spending for all non-diagnosed individuals in a household. The first panel includes the full sample, while the second panel limits the sample to those enrolled in plans with zero deductible at the time of the major health event. Spending is measured in 2020 USD. Standard errors are clustered at the household level.

To explore the role that these conditional price changes have on the observed spending responses, I first examine the potentially heterogeneous effects of major medical events by families' typical pre-diagnosis deductible contributions. Figure 15 illustrates various difference-in-difference estimates for the effect of a major medical event on billed spending, estimated on the sample of families who contributed up to a certain fraction of their deductible on average prior to diagnosis. For this approach, I examine billed spending instead of OOP spending because OOP spending will mechanically rise more for those who tend to have a larger portion of their deductible to pay off, as the deductible is typically the largest contributor to OOP expenses.

The figure shows much larger utilization effects among families that typically spent less than a quarter of their deductible OOP. In fact, families that spent 10% or less of their deductible on average prior to diagnosis are estimated to increase their utilization by about 50%. These large effects decay as more of the sample is included, and I find that even families spending 50% of their deductible may not increase their health utilization following major medical events. Taken together, these results suggest that the families who experience the largest price reductions in care are not the families increasing their utilization the most, suggesting that demand responses are not the major driver of health behavior changes.

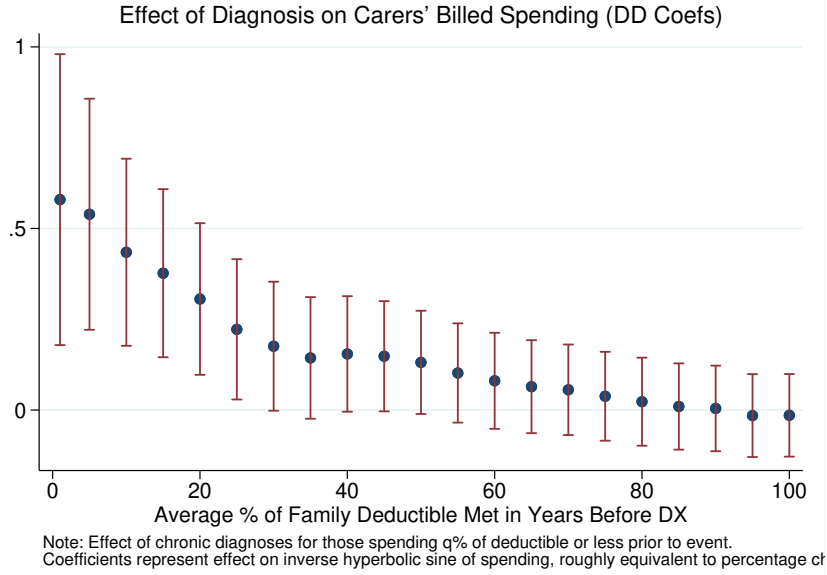


Figure 15. Heterogeneous Utilization Responses based on Pre-Diagnosis Deductible Contributions

Finally, I find a strong extensive margin response among household members who experience major medical events in their families. Table 12 shows that individuals are more likely to spend any positive amount (billed and OOP) on medical care, use any outpatient visits or preventive care, or fill any prescriptions. This effect is strongest in the year of the diagnosis and decays slightly over time, but remains significant for five years following the health event.

B.4 Intra-Familial Relationships

For example, while a diabetes diagnosis is most likely to affect adult household members with similar lifestyles to the original diagnosed individual,³⁷ a mental health diagnosis may have a stronger genetic component. Hence, households where an adult was diagnosed with diabetes may choose to screen other adults, such as spouses, while households where someone received a mental health diagnosis may choose to screen children or siblings of the affected individual.

To assess these potentially heterogeneous effects, I utilize a simple difference-in-differences framework. In Table 13, I present estimation results for the same six diagnosis/outcome pairs shown in Table 3. The dependent variable—either a screening or a new diagnosis—is shown

³⁷The vast majority of diabetes diagnoses in my sample are for Type 2 Diabetes Mellitus, which generally affects adults and risk of which is increased or decreased based on specific lifestyle choices, such as diet and exercise. The same is not as true for Type 1 DM diagnoses.

	Year 0	Years 1–5 (average)
Any Billed Spending	1.54*** (0.08)	0.60*** (0.13)
Any OOP Spending	2.62*** (0.11)	1.41*** (0.18)
Any Outpatient Visits	2.20*** (0.09)	0.65*** (0.15)
Any Preventive Care	3.23*** (0.15)	0.90*** (0.22)
Any Prescription Fills	4.74*** (0.41)	2.45*** (0.53)

Table 12. Estimated Extensive Margin Health Effects of Family Diagnosis

Screening <i>Diagnosis</i>	Hypertension <i>Any Chronic</i>	Diabetes <i>Diabetes</i>	Cholesterol <i>Diabetes</i>	High BMI <i>Diabetes</i>	Cancer <i>Cancer</i>	Depression <i>MDD/Bipolar</i>
$\text{Post}_t \times \text{Diagnosis}_f$	0.39*** (0.03)	-0.85*** (0.21)	-2.20*** (0.29)	-0.38** (0.12)	2.55*** (0.43)	0.30** (0.10)
$\text{Post}_t \times \text{Diagnosis}_f \times \text{Parent}_j$	-0.34** (0.11)	3.49* (1.71)	3.73 (2.26)	1.73* (0.70)	-1.90 (2.49)	-0.93*** (0.13)
$\text{Post}_t \times \text{Diagnosis}_f \times \text{Spouse}_j$	-0.74*** (0.13)	2.54*** (0.45)	5.15*** (0.60)	1.03*** (0.20)	-3.33*** (0.81)	-0.62*** (0.11)
$\text{Post}_t \times \text{Diagnosis}_f \times \text{Sibling}_j$	0.09 (0.04)	0.76 (1.09)	2.89 (1.86)	0.16 (0.69)	1.56 (1.55)	0.68* (0.32)
Observations	4,039,602	3,680,725	3,680,725	3,680,725	3,671,064	3,724,608
Adjusted R^2	0.024	0.217	0.388	-0.025	0.473	0.117

Standard errors in parentheses

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Notes: Table shows results of a difference-in-differences estimation strategy highlighting the potentially differential effects of chronic illnesses on preventive care utilization by household relationships. The primary outcome variable in each column is a screening or new diagnosis, shown in the top row. The specific chronic illness used as the Diagnosis_f dummy is shown in the second row. Standard errors are clustered at the household level.

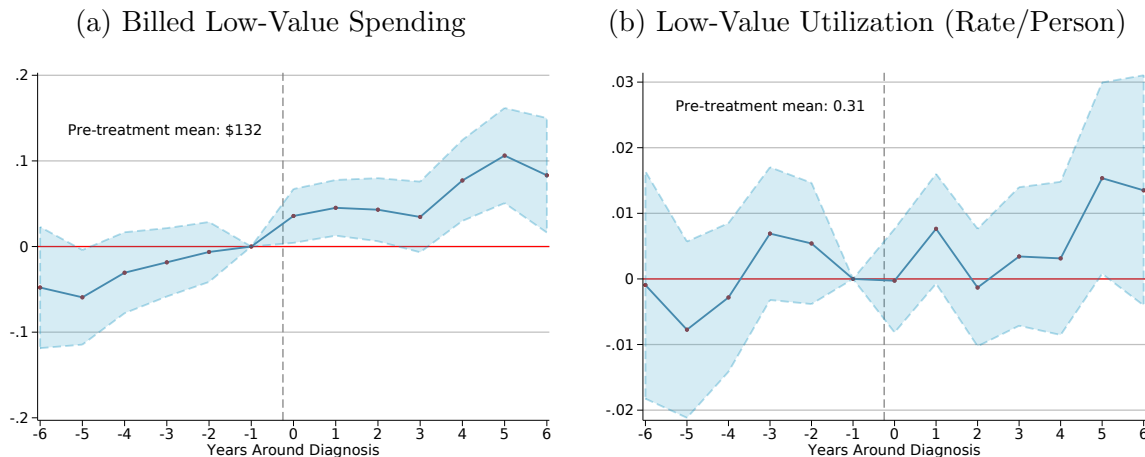
Table 13. DDD Estimates: Disease-Specific Spending

in the top row, with the treatment variable—the chronic illness affecting the household—below in *italics*. I explore the potentially heterogeneous responses for four family relationships: parents, spouses, siblings, and children of the affected individual, with children as the reference group.

Throughout, I find consistent evidence that households respond by not only selecting screenings associated with the health events they experienced, but also selecting which individuals to screen based on their associated risk. New hypertension diagnoses following a chronic event are concentrated among children rather than parents and spouses, suggesting that households are identifying previously ignored risks among the previously lower-risk members of their household. Additionally, households affected with diabetes focus screenings on spouses more than on children, consistent with the lifestyle factors that affect diabetes risk. In contrast, households affected with chronic illnesses that communicate a greater level of genetic risk—cancer and mental health conditions—choose instead to screen children and siblings (in the case of mental health conditions) more than parents or spouses.

B.5 Low Value Care

Figure 16. Chronic Diagnoses Increase Utilization of Low-Value Care



Notes: This figure shows estimated coefficients and 95% confidence intervals for the effect of major health events on the use of low-value services (see Appendix A.5 for definitions). In the first panel, the outcome is the inverse hyperbolic sine of billed spending. In the second panel, the outcome is the number of low-value services used per household member. Spending is measured in 2020 USD. Standard errors are clustered at the household level.

Figure 16 presents estimates for the effect of new chronic diagnoses on the overall utilization of low-value services, including both total spending and overall utilization rates. Major health events are associated with a small increase in overall low-value spending of

<i>Service Category</i>		All Pediatric		Adult Drugs		Adult Imaging		Adult Screening		Adult Surgery	
<i>Outcome Variable</i>		Spending	Rate	Spending	Rate	Spending	Rate	Spending	Rate	Spending	Rate
DiD											
Post _t × Diagnosis _f		0.05*** (0.017)	0.02*** (0.003)	-0.00 (0.000)	-0.00 (0.000)	0.03*** (0.013)	0.01*** (0.002)	0.10*** (0.014)	0.03*** (0.005)	-0.10*** (0.012)	-0.04*** (0.002)
Adjusted R ²		0.192	0.228	0.143	0.259	0.123	0.141	0.163	0.151	0.230	0.255
Event Study											
t − 4		-0.04** (0.014)	-0.02* (0.008)	0.01 (0.003)	0.00* (0.002)	0.01 (0.016)	-0.00 (0.005)	-0.10*** (0.021)	-0.05*** (0.011)	0.09*** (0.012)	0.03*** (0.004)
t − 3		-0.02 (0.012)	-0.01 (0.007)	0.00 (0.002)	0.00 (0.001)	-0.01 (0.013)	-0.01 (0.004)	-0.03 (0.019)	-0.09 (0.010)	0.04*** (0.010)	0.02*** (0.003)
t − 2		-0.01 (0.010)	-0.01* (0.005)	0.00 (0.002)	0.00 (0.001)	0.01 (0.016)	0.00 (0.004)	-0.02 (0.016)	0.00 (0.010)	0.01 (0.009)	0.01** (0.002)
t − 1		—	—	—	—	—	—	—	—	—	—
t		0.02** (0.009)	0.008 (0.004)	0.00 (0.002)	0.00 (0.001)	0.01 (0.010)	0.01 (0.003)	0.03* (0.015)	0.008 (0.008)	-0.03*** (0.008)	-0.01*** (0.002)
t + 1		0.03*** (0.009)	0.01*** (0.005)	0.00 (0.002)	0.00 (0.001)	0.03*** (0.011)	0.01*** (0.003)	0.07*** (0.015)	0.04*** (0.008)	-0.07*** (0.009)	-0.02*** (0.003)
t + 2		0.04*** (0.010)	0.02*** (0.005)	-0.00 (0.002)	0.00 (0.00)	0.02* (0.012)	0.01** (0.003)	0.06*** (0.016)	0.02 (0.009)	-0.08*** (0.011)	-0.03*** (0.003)
t + 3		0.05*** (0.011)	0.02*** (0.006)	-0.00 (0.002)	-0.00 (0.001)	0.03** (0.013)	0.02*** (0.004)	0.07*** (0.018)	0.03** (0.011)	-0.11*** (0.013)	-0.05*** (0.005)
t + 4		0.04*** (0.013)	0.02*** (0.007)	0.00 (0.003)	0.00 (0.002)	0.06*** (0.016)	0.02*** (0.005)	0.10*** (0.021)	0.03* (0.012)	-0.10*** (0.016)	-0.05*** (0.005)
Adjusted R ²		0.192	0.228	0.143	0.259	0.123	0.141	0.163	0.151	0.230	0.255
N		1,538,161	1,538,161	1,538,161	1,538,161	1,538,161	1,538,161	1,538,161	1,538,161	1,538,161	1,538,161

Notes: Table shows estimated difference-in-difference and event study regression coefficients for the effect of a new chronic diagnosis. Two outcome variables are reported for each category: the inverse hyperbolic sine of billed spending and the number of low-value services used per household member. See Appendix A.5 for service definitions. Spending is measured in 2020 USD. Standard errors clustered at the household level are reported in parentheses. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

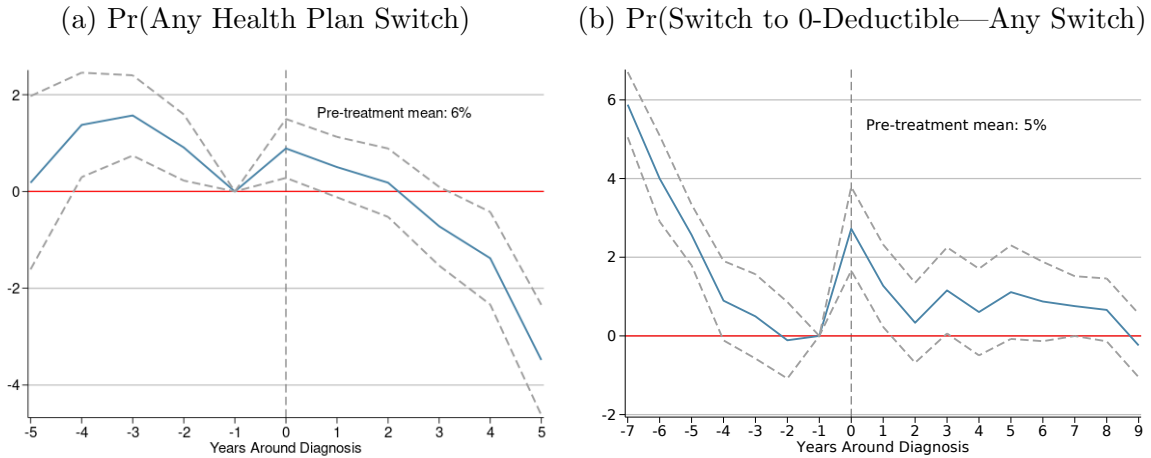
Table 14. Estimated Effects of Chronic Illness on Low-Value Care Utilization, by Category

about 5 percent. In contrast, the average rate of service use among non-diagnosed household members does not change meaningfully following a diagnosis.

B.6 Plan Choices

Finally, using the portion of my sample with identifiable plan choice information, I estimate the effect of chronic health events on household decisions to switch plans. Figure 17 illustrates that affected households are less likely to switch insurance plans following their major health events relative to the general population. I observe both that plan switches do not become more likely overall (Panel (a)), and that even among active choosers, plan switches do not become higher-quality (proxied by the use of zero-deductible plans; see Panel (b)).

Figure 17. Effect of Chronic Diagnoses on Health Plan Switching



Note: These figures assess the impact of major health events on plan switches. The outcome variables are a binary indicator for whether the household switched plans in the first panel, and whether they switched plans to a plan with zero-deductible in the second panel. The second panel restricts the sample to those who ever made an active plan choice. Standard errors are clustered at the household level.

C Additional Modeling Notes

C.1 Solving the Utility Maximization Problem

In the final choice stage of the model, households choose medical spending m_{it}^* based on the realization of their acute shocks $\{\lambda_{it}, m_{ft}^{CH}\}$ and their type parameters $\{p_{it}, \omega\}$. Their expected utility is given by

$$u_{it}(m_{it}) = p \left[(\alpha_1 m_{it} + \alpha_2 m_{ft}^{\text{CH}} - \lambda_{it}) - \frac{1}{2\omega} (\alpha_1 m_{it} + \alpha_2 m_{ft}^{\text{CH}} - \lambda_{it})^2 - c_j(m_{it}) \right] \\ + (1-p) \left[(m_{it} - \lambda_{it}) - \frac{1}{2\omega} (m_{it} - \lambda_{it})^2 - c_j(m_{it}) \right] + \varepsilon_{ijt}. \quad (17)$$

Ignoring the idiosyncratic shock ε_{ijt} , the first order condition for utility maximization implies that optimal spending is given by:

$$m_{it}^* = \frac{1}{1 + p_{it}(\alpha_1 - 1)} \left[\lambda_{it} + \omega(1 - c'_j(m_{it}) + p_{it}((\alpha_1 - 1)\omega - \alpha_2 m_{ft}^{\text{CH}})) \right]. \quad (18)$$

Without the expected utility framework or allowing for state-dependent utility across states, this reduces to the typical solution of $m_{it}^* = \lambda_{it} + \omega(1 - c'_j(m_{it}))$. Here, $c'_j(m_{it})$ depends on the optimal level of spending, with $c' = 1$ when households choose a level of spending below the deductible, and then declining to $c' = c < 1$ when OOP spending is between the deductible and the OOP max, and $c' = 0$ otherwise. The piecewise linear structure of the cost-sharing scheme does not yield a closed form solution for m_{it}^* , but rather implies a discrete set of possible solutions that must be evaluated.

C.2 Alternate Interpretations of p

The evidence presented in Section 3 suggests that health events generate spending responses as household members reevaluate their health risks. This leads to the simple interpretation of the dynamic learning parameter p_{it} as a probability of an adverse health event occurring. However, to the extent that other informational effects affect spending choices in ways that are separate from health risk information, moral hazard effects, or salience effects, these effects may “load” onto the estimated p_{it} parameter, affecting its interpretation. These informational effects may include physician relationship building, increased comfort obtaining care covered by an insurer, or other, more general health information effects, which alter consumer *preferences* for health care rather than their *beliefs* about risk.

The transition probability parameter p_{it} can therefore be interpreted, in part, as an adjustment to consumer preferences for care in addition to risk beliefs. Consider equation 17. If we assume that $\alpha_1 \approx 1$, as estimated in Section 5, the equation reduces to:

$$u_{it}(m_{it}) = m_{it} - \lambda_{it} - c_j(m_{it}) + p_{it}\alpha_2 m_{ft}^{\text{CH}} - \frac{p_{it}}{2\omega} (m_{it} + \alpha_2 m_{ft}^{\text{CH}} - \lambda_{it})^2 - \frac{1-p_{it}}{2\omega} (m_{it} - \lambda_{it})^2. \quad (19)$$

Hence, p_{it} can be construed, together with the estimated parameter α_2 , to be represen-

tative of the preference weight individuals place on chronic care, relative to all non-chronic care. In this setting, the informational effect of health shocks increases individual preferences for chronic care.

D Additional Structural Results

D.1 Estimation Algorithm

I estimate the model described in Section 4 using a maximum likelihood approach similar to Train (2009) and Revelt and Train (1998), with the appropriate extension to a discrete/continuous multi-stage choice model as discussed in Dubin and McFadden (1984). My estimation approach is similar to other models like mine, including Marone and Sabetty (2020). I estimate the parameter values θ that maximize the probability density of households' observed total healthcare spending conditional on their plan choices.

My model allows for individuals to have three type-specific dimensions of unobservable heterogeneity, in addition to the typical Type 1 Extreme Value idiosyncratic shock (which can be integrated out analytically): individual health states, individual beliefs about health risks, and household risk aversion. I therefore must numerically integrate over the three dimensions $\beta_{ft} = (p_{it}, \mu_{\lambda,i}, \psi_{ft}) \in \theta$. Given a guess of θ , I use Gaussian quadrature with 27 support points (three in each dimension) to simulate underlying consumer types, yielding simulated points $\{\beta_{fts}(\theta)\}_s$ and weights W_s .

For each simulation draw s , I can then calculate the conditional density at individuals' observed total healthcare spending and the probability of households' observed plan choices.

D.1.1 Household Spending

Given data on realized choices m_{it} , I construct the distribution of healthcare spending for each individual-year implied by the model and guess of parameters θ . Based on underlying consumer types β_{fts} , I construct individual-level parameters for health states $(\mu_{\lambda,i}, \sigma_{\lambda,i}, \kappa_i)$ based on the parameters β_{fts} and the distributions outlined in Section 4.3.1.

The model predicts that given an acute-chronic health state $(\lambda_{it}, m_{ft}^{\text{CH}})$, households choose total healthcare spending m by trading off the benefit of healthcare utilization with its out-of-pocket cost, as discussed above. Given that m_{ft}^{CH} does not have individual parameters to be estimated (as these values are drawn from an empirical distribution), inverting the expression in equation 18 yields the health state realization λ_{its} that would have given rise to observed spending m_{it} given m_{ft}^{CH} . Given that observed spending is truncated from below at 0, there are two possibilities for the conditional pdf:

$$f_m(m_{it}|c_{jt}, \beta_{fts}, \theta) = \begin{cases} \Phi\left(\frac{\log(\kappa_i) - \mu_{\lambda,i}}{\sigma_{\lambda,i}}\right) & m_{it} = 0 \\ \Phi'\left(\frac{\log(\lambda_{its}) - \mu_{\lambda,i}}{\sigma_{\lambda,i}}\right) & m_{it} > 0, \end{cases} \quad (20)$$

where $\Phi(\cdot)$ is the standard normal cumulative distribution function. In practice, there are iterations where the implied pdf is zero; hence, in order to rationalize the data for any parameter guess, I use a convolution of f_m with a uniform distribution over the range $[-1e-75, 1e-75]$, as done by Marone and Sabety (2020).

D.1.2 Plan Choices

I next calculate choice probabilities for each available health insurance plan. Given θ and β_{fts} I numerically integrate over the joint distribution of acute and chronic health care shocks using $D = 10$ support points in each dimension. The support points for the chronic health care shocks are chosen uniformly across the empirical distribution with the empirical pdf used in calculating the associated weights. For the acute health shocks, support points are calculated over the lognormal distribution as:

$$\lambda_{itsd} = \exp(\mu_{is} + \sigma_{is}Z_d) + \kappa_{is}, \quad (21)$$

where Z_d is the appropriate Gaussian quadrature vector of points (with corresponding weights W_d). The utility maximization framework discussed above (Equation 18) is then used to calculate the optimal spending levels given individual and household shocks and the underlying parameter p_{it} . Expected utility for each support point is calculated as in equation 9 and summed (with weights) over all 100 points.³⁸ Choice probabilities for a plan j are then given by the standard logit formula

$$L_{ftjs} = \frac{\exp(U_{ftjs}/\sigma_\epsilon)}{\sum_{i \in \mathcal{J}_{ft}} \exp(U_{ftis}/\sigma_\epsilon)}. \quad (22)$$

D.1.3 Likelihood Function

Based on the choice probabilities and conditional density functions for observed spending, the likelihood function is approximated by

$$LL_f = \sum_{j=1}^J d_{fjt} \sum_{s=1}^S W_s \prod_{t=1}^T f_m(m_{it}|c_{jt}, \beta_{fts}, \theta) L_{ftjs}, \quad (23)$$

³⁸In practice, to speed up estimation, I ignore points with associated weights smaller than $1e-5$.

where d_{fjt} is an indicator variable equal to one if household f chose plan j at time t and zero otherwise. The log-likelihood function to be maximized is therefore the sum over households:

$$LL(\theta) = \sum_{f=1}^F \log(LL_f). \quad (24)$$

D.2 Additional Parameters

Describe table of mean shifters for preferred specification:

	p_0	λ	κ	ψ_0
Intercept	0.089	0.190	-0.105	0.112
Age	0.084	-0.088	-0.097	
Age ²	0.115	-0.006	-0.087	
Female	0.102	0.219	-0.117	
Individual risk score	0.100			
Any PE condition in family	0.107			
Type		0.152		
Family size				0.107
Average family age				0.052
Average family risk score				0.140

Table 15. Estimated Type Mean Shifting Parameters

Notes: Values reported for preferred specification only.

Include versions of this for other specifications?

D.3 Additional Model Fit

D.4 Additional Simulations