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Does Intra-household Contagion Cause an Increase in Prescription Opioid Use?

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Abstract

Opioid use claims many thousands of lives each year. This article considers the diffusion of prescription opioid (PO) use within family households as one potential culprit of the proliferation of these medications. In an analysis of hundreds of millions of medical claims and almost 14 million opioid prescriptions in one state between 2010 and 2015, we show that the use of POs spreads within family households. We also show that the treatment effect of exposure to a family member's PO use is driven by an increase in PO consumption for medical conditions that members of treated and untreated families experience at nearly identical rates. This pattern of results suggests household exposure causes an uptick in patient demand for prescription opioids. We use an instrumental variable estimation strategy to address the well-known challenges to estimating a causal effect of intra-household contagion, such as genotypic similarities among family members, assortative matching in partner selection, and clustering of health conditions within households. The results spotlight the salience of the most ubiquitous social structure, the family household, in accelerating opioid consumption to unprecedented levels. The findings also suggest that rather than direct social influence between physicians, the spread of prescription behavior in physician networks may be driven by shifts in patient demand that propagate through the patient sharing network.

Keywords

social networks, diffusion, health outcomes, family, prescription drugs

Consumption of prescription opioids (POs) and street versions of these drugs have caused one of the most significant and tragic public health crises in U.S. history. Between 1999 and 2016, more than 200,000 people in the United States died from overdoses related to prescription opioids (Seth et al. 2018). To contextualize this number, fatal overdoses from opioid analgesics are now more prevalent than deaths from either suicides or automobile accidents.

The opioid crisis and the much broader use of prescription pain medications represents one of the most dramatic and consequential cases of the "medicalization" of modern life (Conrad 1992, 2005; King et al. 2013; King, Jennings, and Fletcher 2014). As an always-growing number of health conditions have an ever-expanding array of pharmaceutical treatment options, the daily use of pharmaceuticals has become commonplace. Indeed, a central culprit of the opioid crisis is the wide availability of these prescription medications. In 2012, the middle year in our data, U.S.

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physicians wrote 255 million opioid prescriptions, which is 81.3 scripts per 100 citizens.[1] This quantity of opioid use equates to a per capita consumption rate in the United States that exceeds the rate in European countries by a factor of 4.5.[2]

The inroads of medication as a mundane occurrence has origins in remarkable technical advances and improved understandings of the biological mechanisms of disease (Cutler and McClellan 2001; Lichtenberg 2001). However, we also know that social factors have much to do with drug use choices (Conrad 1992, 2005; King et al. 2014). Furthermore, social factors influencing decisions about medication use are likely to be particularly salient for health states such as pain, for which there are no definitive biomarkers (Conrad 2005; Fox 1957; Freidson 1962; Shaw and Woodward 2017). Likewise, social considerations loom large in decisions about drugs that have a high potential for abuse. In fact, the individual experience of substance use is virtually always rooted in some type of social context, a phenomenon Galea, Nandi, and Vlahov (2004) aptly label the "social epidemiology of substance use."

In this article, we investigate the role of social contagion in the proliferation of PO use. We hypothesize that family members exposed to POs inside the household are more likely to request them from physicians than otherwise similar individuals in opioid-unexposed households. Specifically, we study whether POs filled by patients cause other family household members to later acquire a PO. A causal association exists if social learning about the palliative or euphoric properties of opioids occurs within households, or if imitation or social influence processes unfold in families.

Because the family is the most prevalent and fundamental organizational structure in human society, we focus on within-household diffusion of PO use. Family circumstances influence nearly all aspects of socioeconomic life, including the formation of social capital (Sanders and Nee 1996) and political views (Flanagan and Tucker 1999), economic opportunities (Renzulli, Aldrich, and Moody 2000), psychological well-being (Helliwell and Putnam 2004), and myriad health practices. Regarding the latter, the family context may play a pivotal role in facilitating the spread of information and behaviors. This occurs because the physical and emotional proximity of household members creates a ripe context for social learning, especially for practices that are risky, controversial, costly to adopt, or private. Therefore, we have reason to anticipate that individual health behaviors are deeply rooted in familial practices.

One challenge, however, is that family studies of the contagion of health behaviors generally suffer from a causal inference problem resulting from the inability to account for similarities between family members and their common exposure to factors that determine the outcomes under examination (Avenevoli and Merikangas 2003). We address this issue by developing an instrumental variable (IV) estimation strategy that relies on the random assignment of patients to emergency department (ED) physicians and the significant variation in the rate at which these clinicians prescribe opioids. Matching between ED physicians and patients is random, so we are able to use the prescribing rate of the assigned physician as an exogenous factor that shifts the probability that ED patients receive an opioid prescription, and therefore introduce this medication to their household.

We find that the diffusion of POs within households fuels consumption of these drugs. We examine instances in which a "source" patient visits the ED ("the index visit") and either does or does not receive an opioid prescription. Family members of these source patients are the "at-risk" group, and we evaluate whether these individuals consume opioids in the year following the index visit. Results show that residing in a household in which a source family member fills a PO increases an at-risk member's subsequent likelihood of obtaining a PO in the following year by 19 percent to over 100 percent, depending on the subpopulation. The effect is remarkably stable across demographic and family characteristics. The one exception is socioeconomic status (SES): the treatment effect is substantially

more pronounced among families that reside in low-SES zip codes.

In a second set of analyses we address a core debate in the literature on medicalization regarding the role that physicians and patients play in the ever-expanding use of prescription medications (Conrad 1992, 2005; Shaw and Woodward 2017). By analyzing the health care consumption behaviors of at-risk family members in opioid-exposed households versus those in untreated families, we show that at-risk patients across the two groups visit primary care physicians (PCPs) and other medical specialists for a remarkably similar set of health reasons.

Conversely, *conditional on visiting a physician for pain symptoms*, we find that at-risk family members of opioid-treated source patients subsequently leave their physicians' offices with a PO at a much higher rate than do individuals in opioid-unexposed households. This pattern is consistent with a mechanism in which treated at-risk patients ask their physicians for an opioid prescription when in pain, and physicians comply with these requests in many cases. This finding also suggests that the clustering of prescription behavior within a network of physicians is not just a function of physicians directly influencing each other via communication channels (Coleman, Katz, and Menzel 1957), but also of physicians steering patient demand.

THE DIFFUSION OF OPIOID CONSUMPTION

Opioid Use and Its Precursors

A growing literature documents sources of variation in prescription opioid use. One strand of work highlights the variation in physician prescribing behavior. Morley-Forster and colleagues (2003), for example, surveyed Canadian PCPs and found that for chronic noncancer pain, 32 percent of physicians used opioids as a first-line treatment, and 35 percent reported they would never prescribe opioids for noncancer pain, even in severe cases. Likewise, a 2013 study of POs in a sample of adult nonsurgical admissions to U.S. hospitals found that, after adjusting for patient characteristics, opioid prescribing rates ranged from 33 to 64 percent (Herzig et al. 2014).

An emerging literature correlates physician characteristics with prescription rates. Volkow and colleagues (2011), for example, found that PCPs write 28.8 percent of all opioid prescriptions. Levy and colleagues (2015) examined differences between specialties and found that POs as a fraction of all prescriptions was highest among specialists in pain medicine (48.6 percent), surgery (36.5 percent), and physical medicine/rehabilitation (35.5 percent). Other research has examined the correlation between prescribing behavior and physician demographics, including physician gender, age, and number of years in practice (Dhalla et al. 2011). None of these correlations are particularly strong.

Evidence also suggests that the aggressive marketing tactics of a few pharmaceutical companies have influenced physician prescribing behavior to the point that these practices constitute a true root cause of the proliferation of POs (Griffin and Miller 2011; Hadland, Krieger, and Marshall 2017). Other scholars highlight the dissemination of misinformation among physicians about the true extent of the addictive properties of prescription opioids, which may have expanded the availability of these medications in the early 2000s (Quinones 2015). A recent study suggests that physician education may limit the influence of marketing efforts and misinformation; there is tentative evidence that physicians who attended highly ranked medical schools may prescribe opioids at lower rates (Schnell and Currie 2017).

Research also documents variation in opioid use between patients. Age is one of the strongest demographic predictors of opioid consumption. In Minnesota in 2015, the number of opioid prescriptions ranged from 1.3 per 100 persons age 2 to 11 to 73.6 per 100 persons age 45 to 64.[3] Patient gender is also correlated with opioid use: women are more likely than men to be prescribed opioids (Campbell et al. 2010). Finally, there is well-documented geographic variation in opioid

use. For example, in 2015 in Louisiana, among individuals age 45 to 64, physicians prescribed 203 POs per 100 persons, which is almost three times the rate of consumption by patients in Minnesota.[4] The extent of the geographic variation in PO use is far too extreme to be entirely driven by the spatial ecology of health statuses.

Despite the stark differences in opioid prescription rates, many of the causes of variation in use remain undocumented. This study contributes to our understanding of this puzzle by developing and testing the argument that learning within social networks may simultaneously increase both demand and supply of these drugs.

Interest in the social structural underpinnings of the diffusion of health practices dates far back in sociology. Indeed, Coleman and colleagues (1957), one of the canonical diffusion studies in the discipline, examined the spread of penicillin use in a network of physicians. They posit that the social networks in which physicians are embedded act as a "chain of influence" that causes physicians to adopt prescription behaviors similar to those of their direct peers. Although the original findings have been revisited and questioned (Van den Bulte and Lilien 2001), this project was one of the first to present evidence that the contagion of a health practice depends on the social structure in a community of actors.

A few notable examples of research on the diffusion of health-related practices include the spread of exercise habits over a social network (Aral, Muchnik, and Sundararajan 2009; Aral and Nicolaides 2017; Centola 2010), latrine ownership (Shakya, Christakis, and Fowler 2014), the incidence of diagnosis of autism (Liu, King, and Bearman 2010), and social influence on fertility behaviors (Balbo and Barban 2014).

Learning in the Family Household

The family household is the most fundamental building block in the organization of society (Coleman 1988). Relationships inside of family households are complex, multiplex, temporally variable, and of heterogeneous valence (Bott 1957). Despite the obvious diversity in intra-familial interactions, literatures in sociology, anthropology, psychology, economics, and public health all suggest that relationships within the family are an important source of influence for the formation of belief systems and for the social spreading of behaviors. Given its ubiquity as an organizational form and the depth of intra-familial relationships, the family household is a critically important social substrate for the contagion of many health behaviors, including the use (and misuse) of prescription medications.

Why are family ties especially salient in the diffusion of health behaviors? Any analysis of diffusion begins with exposure: knowledge of a practice is a necessary condition for its adoption. The physical and psychological proximity of household members makes it an extremely active stratum for observation, exposure to others' behaviors, and social learning (Axinn and Thornton 1993; Liu et al. 2010; Soons and Kalmijn 2009; Zuckerman, Dasovic, and Fitzgerald 2007). Moreover, members of family households often are embedded in overlapping, external, neighborhood- and community-based social networks. The presence of common, third-party associations fortifies pressures toward behavioral conformity with regard to many beliefs and practices that emerge within families.

In work on the spread of alcohol consumption and smoking, the family has been a particular locus of scholarly attention. In households in which parents smoke, for example, children learn the rudiments of smoking behavior, such as how to light a cigarette and inhale smoke. Observational and reinforcement learning processes ensure that smoking then joins the repertoire of behaviors children discover and become capable of enacting (Darling and Cumsille 2003). Likewise, sibling imitation with regard to smoking and other forms of substance use is common in adolescence (Avenevoli and Merikangas 2003; Hops et al. 2000; Rajan et al. 2003; Vink, Willemsen, and Boomsma 2003). A substantial body of work shows that siblings reciprocally influence one another's smoking and drinking behaviors, leading to

intra-household contagion. However, virtually none of this work presents convincing evidence for a causal social influence process inside the household, generally because unmeasured confounding factors and events induce similarities in sibling-sibling or parent-child behaviors that can easily masquerade as inter-person contagion, even when none is present.

In the case of diffusion of PO use within families, two pathways—*exposure* and *access*—may trigger learning about opioids followed by the adoption of behavior. Both pathways are consistent with at-risk family members learning about opioid analgesics as a result of family members using POs. *Exposure* occurs when an opioid is consumed by a household member and other family members learn about the effects of the medication through first-hand observation of the efficacy of POs for pain mitigation. Likewise, family members may witness the euphoria another household member experiences when consuming these drugs (Woolf and Hashmi 2004). Conrad (2005) describes a similar process that accelerated diagnoses of adult ADHD in the 1990s: by observing similarities between themselves and their ADHD-diagnosed children, adult parents learned to associate their own symptoms with ADHD and to persuade physicians to diagnose them with the condition.

Access occurs because prescription medications often result in an excess supply of pills in the family medicine cabinet (Bicket et al. 2017; Hill et al. 2017), creating an opportunity for drug diversion. Source patients who receive a PO often consume less than the prescribed quantity of pills. If at-risk family members choose to consume any of the leftover pills, they experience the effects of the medication. In addition to learning, at-risk family members may unwittingly initiate the opioid dependence cycle, which can onset after brief periods of opioid use.[5] In survey evidence, drug diversion within families has been identified as a mechanism leading to misuse of prescription medications (Boyd and Mieczkowski 1990; Inciardi et al. 2009). In the opioid context specifically, the majority of individuals who self-report misusing POs first received medication from either a family member or a friend.

Relationships in the family household are more likely than weak and more distant social ties to lead to opioid access and exposure effects. Direct exposure to the source patient's substance use and direct access to excess medication in the home are often limited to members of the family household and, occasionally, close friends. Additionally, due to higher levels of trust and intimacy, social learning about private health matters is more likely between family members than among individuals connected by weaker social bonds. Unlike the spread of an infectious disease that can occur with minimal contact between individuals, contagion of certain health behaviors that are costly to adopt requires a social context of richer, more multiplex relationships.

For these reasons, we expect to observe social spreading of the use of POs within family households:

> *Hypothesis 1:* When one member of a family household (the source patient) receives an opioid prescription, this causes an increase in the subsequent probability that other household members (the at-risk members) later fill an opioid prescription.

To be clear, our hypothesis addresses a stimulant of patient demand for PO analgesics. Insofar as intra-household exposure ignites a learning process, this causes an outward shift in the demand for medication among at-risk members of opioid-exposed households. Yet, no matter how compelling the arguments to believe that intra-household learning about POs induces greater *demand* for these medications, it remains the case that the *supply* of prescribed medications is controlled by physicians. Therefore, this empirical setting contrasts with most research on diffusion of health behaviors, which concerns actions individuals choose to adopt at their sole discretion. For example, the decision to exercise

after encouragement by peers is a choice at-risk actors make on their own accord (Aral and Nicolaides 2017). Cigarette smoking and alcohol consumption likewise are unconstrained by others, except in the case of under-age adopters.

Why do we anticipate that physicians will often comply with patient demand for prescription opioids? This question is central to discussion of the role of medical expertise in the provision of health care (Timmermans and Oh 2010). Training and expertise, certification, and legal protection of the medical profession by the state should allow physicians to treat their patients using scientifically validated knowledge (Freidson 2001; Timmermans and Oh 2010), but several factors may erode physicians' autonomy in medical decision-making.

First, there is often no clear diagnostic test for the extent of pain. Despite the fact that pain is one of the most common medical complaints, we lack objective biomarkers to guide its diagnosis or to dictate choice of treatment (Conrad 2005; Freidson 1962). Patient self-reports of pain remain the primary means of discovery, and patients are often diagnosed with non-malignant chronic pain when they exhibit no identifiable, underlying medical pathology. In this regard, the diagnosis and treatment of pain resembles autism at the ends of the disease spectrum or ADHD: it is characterized by a high degree of diagnostic uncertainty, and treatment is strongly influenced by self-reports (Liu et al. 2010). Of course, this means that falsifying or exaggerating symptoms of pain with the goal of obtaining prescription medications may contribute to the opioid crisis (Bass and Halligan 2014; Bouland et al. 2015; Leavitt and Sweet 1986; LoPiccolo, Goodkin, and Baldewicz 1999).

Second, clinicians may comply with patient demand for prescription opioids because they are motivated, intrinsically and extrinsically, to contribute to patient satisfaction during medical visits (Kolstad 2013). Patient satisfaction scores are increasingly incorporated into insurance reimbursement rates, which creates an incentive for physicians to provide POs to patients who insist on these medications (Kolstad 2013; Zgierska, Miller, and Rabago 2012). Some research suggests that patient requests often shape the outcomes of physician visits, but direct, systematic evidence concerning how patient demand affects prescription rates is lacking (Boath and Blenkinsopp 1997; Conrad 1992; Schwartz, Soumerai, and Avorn 1989; Sun et al. 2009).

If social learning in the family household indeed shifts patient demand for POs and physicians comply with patient requests, the referral and patient sharing networks that link physicians may start to exhibit clustering of prescription behaviors. In other words, physicians who share patients may exhibit more similar prescription behaviors than physicians who share no patients. Importantly, this can occur not just because physicians directly influence each other, but because physicians may shift patients' demands, which affects other physicians with whom they frequently share patients (Coleman et al. 1957; Landon et al. 2012). Thus, in addition to physicians influencing each other through communication, the spread of prescription behaviors may be driven by shifting demand of the patients that physicians share and refer.

EMPIRICAL STRATEGY

Our goal is to determine whether a household member filling an opioid prescription causes another family member to acquire a PO at a future time. To assess this, we compare the rates at which at-risk family members in treated households fill opioid prescriptions to the rate at which at-risk family members in control households fill opioid prescriptions. Using data from the ED allows us to analyze a natural sample of treated (i.e., the patient left the ED with an opioid prescription) and control (i.e., the patient did not receive a PO) family members.

As the literature on statistically identifying social diffusion effects clarifies, demonstrating that a type of behavior clusters within a set of socially connected actors does not in and of itself imply the presence of a causal social diffusion process (Azoulay, Liu, and

Stuart 2017; Christakis and Fowler 2008; Liu et al. 2010; Shalizi and Thomas 2011; Van den Bulte and Lilien 2001). In the case of POs, a temporal clustering of opioid use within family households may result from any of three broad types of processes: (1) social influence or learning, (2) assortative matching and genetic similarities among family members, or (3) common exposure effects.

The empirical challenge we must tackle is to distinguish social contagion from the second and third sets of causes, that is, to parse the true effect of social diffusion from the sociological null hypothesis of the other two sets of factors (Liu et al. 2010). For example, the selection of romantic partners may be driven by characteristics that predispose individuals to consume POs, in which case household clustering of POs could be entirely driven by mate selection. Or, exposure of multiple family members to common risk factors, such as any form of physical, financial, or emotional hardship, may cause temporal clusters of opioid-filling behavior within families. In addition, members of a family sometimes consult the same primary care provider (PCP). Because PCPs exhibit wide variation in the rate at which they prescribe opioids, the correlation in PO use within households may be partly attributable to common physician effects. Although both selection effects and common exposure effects could have important implications for understanding the opioid crisis, our aim is to isolate the effect of social spreading of opioid use within family households.

Consider a model in which an at-risk family member, i, filling an opioid prescription $FILL_{i,t>T}$ at time $t > T$ is a function of whether or not a source family member, j, filled an opioid prescription ($FILL_{j,t=T}$) at time $t = T$:

$$FILL_{i,t>T} = \beta_0 + \beta_1 \times FILL_{j,t=T} + \beta_2 \times X_i + \beta_3 \times X_j + \varepsilon_i \quad (1)$$

The vectors X_i and X_j include control variables pertaining to at-risk and source family members, respectively. The coefficient of interest is β_1, and the empirical task is to obtain an unbiased estimate of β_1 in the presence of either assortative matching or common exposure to factors that affect opioid use. If either process is present in the data and not fully accounted for by control variables, there will be a correlation between treatment, $FILL_{j,t} = T$ and the error term. Formally, the OLS estimate of β_1 is biased because $cov(FILL_{j,t=T}, \varepsilon_i) \neq 0$.

As a first step toward obtaining a reliable estimate of the intra-household treatment effect, we present specifications of Equation 1 in which we control for many possible confounding factors. The data are the complete set of medical claims and extensive health histories of patients, allowing us to include many control variables. These include the at-risk patient's gender, age, pre-treatment prescription medication histories (including benzodiazepine[6] use), the overall rates at which opioids are prescribed for the medical conditions the source patient is diagnosed with, a comorbidity index that captures health status, and so on.

Despite the inclusion of a comprehensive set of control variables, we have no compelling reason to believe that the parameter estimates are immune to unobserved confounders. Therefore, we next rely on an instrumental variables (IV) identification strategy. An IV is a variable, Z_i, that satisfies two conditions. First, the IV must be relevant. In our setting, the IV must significantly affect the likelihood that a *source* patient receives a prescription opioid. Second, the IV must meet the condition of an exclusion restriction: the instrument must affect the at-risk family member's PO use exclusively via its influence on the endogenous treatment variable. In other words, we require a variable that significantly affects the probability that a source patient receives an opioid prescription (treatment), but conditional on all other covariates, the IV can have no independent effect on the likelihood that the at-risk household member receives a PO (outcome), except through its effect on treatment. Formally, a valid instrument requires $cov(FILL_{j,t=T}, Z_i) \neq 0$, and $cov(Z_i, \varepsilon_i) = 0$. The IV must be correlated with treatment, $FILL_{j,t=T}$,

and conditionally uncorrelated with the error term in Equation 1.

The instrument we use relies on a subset of our data in which a natural experiment occurs. Specifically, we limit our analysis to the clinical setting of the emergency department (ED). Compared to nearly all other patient encounters with the health care system, the unique advantage of the ED is that assignment of patients to physicians in the ED is thought to be as-good-as random (Barnett, Olenski, and Jena 2017; Greenwood, Carnahan, and Huang 2018). Moreover, ED physicians who treat similar patient mixes demonstrate substantial variation in the rate at which they prescribe opioids (Barnett et al. 2017).[7]

The logic of the experiment we run is as follows. A source patient experiences an adverse health event and visits an ED. Upon arrival to the ED, the source patient is randomly paired to an on-duty ED physician. If the source patient matches with a high-prescribing practitioner, that patient is more likely to receive a prescription opioid. Because some of the variation in whether a source patient receives a PO is caused by random assignment to a high- versus low-prescribing physician, there is also a random component to an at-risk family member's exposure to opioid use in the household via the source patient. When we implement an IV strategy and limit the estimation of β_1 to the variation in treatment created by the random assignment of source patients to ED physicians, we interpret any difference in the outcome (i.e., the at-risk family member filling an opioid prescription) as a causal effect of changes in opioid introduction in the family that is immune to the concerns of assortative matching and common exposure effects. However, as we will discuss in detail, the IV estimator is interpreted as a local average treatment effect (LATE). It generally does not recover the average treatment effect (ATE) in the overall population; it provides the local treatment effect in the subset of the data for which the IV drives variation in treatment.

Sampling only ED patients has additional benefits. Relying on ED patients eliminates the possibility that family members exhibit similar opioid consumption patterns that result from seeing the same physician. The sole source of introduction of prescription opioids to the household is the ED visit, and neither the source patient nor at-risk household members schedule follow-up visits with ED physicians. Sampling only source family members through ED visits also provides us with a natural sample of "treated" and "control" families. Specifically, 13 percent of ED patients in the sample received an opioid prescription during their visit to the ED, so the ED sample yields a set of treated (13 percent) and control (87 percent) households. Finally, the fact that every family household in our sample enters through a source patient's ED visit is likely to impose some level of balance between the treated and control groups and potentially eliminates concerns about unobserved confounders.

DATA AND SAMPLING

Data Description

The data used in this study come from one primary source: the Massachusetts All Payers Claims Database (MA APCD). The MA APCD is collected and maintained by the Center for Health Information and Analysis (CHIA) and contains remarkably comprehensive information derived from the medical and pharmacy claims of virtually every resident in Massachusetts between January 1, 2010, and December 31, 2014. Massachusetts requires health insurers in the state to report detailed information on every medical and pharmacy claim they receive. CHIA collects these data and prepares them for use in research. For instance, CHIA processes the data to link records of the same individual who has had multiple insurance plans over time.

The MA APCD contains three main data files that we draw from: medical claims, pharmacy claims, and the member eligibility file. Data in the medical claims file include a physician identifier, a patient identifier, diagnosis codes, dates and locations of provider visits,

Table 1. Sample Evaluation

Year	Member Count	Population Count	Absolute Difference	Relative Difference
2010	6,166,446	6,557,300	390,854	5.96
2011	6,340,138	6,611,800	271,662	–4.11
2012	6,466,476	6,657,800	191,324	–2.87
2013	6,632,721	6,708,800	76,079	–1.13
2014	6,883,840	6,755,100	128,740	1.91

medical procedures, and charged dollar amounts. Data in the pharmacy claims file include a unique prescribing provider identifier, a member identifier, medication identifiers, drug supply (in days and dosage), and charged dollar amounts. The member eligibility file is a de facto roster of the insurance coverage of all Massachusetts residents who carry a health insurance plan.

Table 1 shows the number of unique individuals per year in the member eligibility file and the number of residents in Massachusetts according to the U.S. Census. The table shows that the data in the MA APCD are remarkably comprehensive. The differences between the two populations are generally small.[8] The increase in the sample of individuals identified through the member eligibility file is likely caused by an increase in the number of insured Massachusetts residents.

Sampling

To construct our sample, we extracted medical claims based on the medical specialty of the individual provider and the procedure code listed on the claim. Specifically, we extracted all medical claims that had at least one individual provider with "Emergency Medicine" listed as their primary specialty and any of the following Current Procedural Terminology (CPT) codes: 99281, 99282, 99283, 99284, 99285. CPT codes are the U.S. standard for classifying and reporting medical, surgical, radiological, laboratory, anesthesiology, and evaluation and management (E/M) services. CPT codes determine reimbursement rates and are used by all health care providers, payers, and facilities in the United States. The five codes we use to identify ED claims refer specifically to E/M services performed in EDs.

In the next step, we identify in the sample of ED claims all individuals with one or more family members in the same household. To infer family ties we leverage information about individuals covered under the same medical insurance policy number. Specifically, we infer a family tie between individual *j* and individual *i* if they are covered under the same insurance policy in year *t*. This strategy allows us to identify about 1 million families each year, which is about two thirds of the 1.6 million households that the U.S. Census identifies in Massachusetts. Table 2 shows the number of families and household members we are able to identify in each year of the data. We under-observe family households because we rely on joint coverage under a common insurance policy number to identify households. Two members of a household who are on different insurance plans will appear in the data as singletons, and therefore will be excluded from the analysis sample. Families insured through Medicaid will account for a large fraction of the false negative family units, because Medicaid plans are technically individual plans and do not allow us to infer family ties.

If a household member visits the ED at time *t*, this index visit becomes an observation in our sample and it puts all family members, *except the source patient who visits the ED*, at risk of filling an opioid prescription in the 365-day period immediately following the index visit.[9] To be precise, we create a single

Table 2. Families and Members Identified through Insurance Plans

Year	Number of Families	Number of Family Members
2010	952,379	2,961,155
2011	970,927	3,027,702
2012	984,765	3,091,936
2013	1,000,027	3,159,442
2014	1,018,617	3,235,999

observation in the analysis dataset per ED visitor, and the outcome variable corresponding to that observation gauges the opioid-filling behavior of at-risk family members in the year following the day of the visit.[10] Our main specifications are at the family level (i.e., one observation per index visit), but we also present estimates from regressions at the individual level (i.e., multiple observations per index visit if there is more than one at-risk family member in the household).

In determining whether any at-risk family member fills an opioid prescription, we sort through all prescriptions filled by Massachusetts residents. That is, we do not limit the use of POs by at-risk family members to prescriptions that were obtained during an ED visit. This allows us to examine the broader question of whether there is evidence of intra-household diffusion in the use of POs, while using the ED context solely as a means of creating random variation in the initial introduction of opioids to a household via the source patient.

In a series of additional sampling steps, we take further precaution against selection issues that might bias the results. First, despite the fact that prior research shows patient-physician matching in the ED is as-good-as random, patients who repeatedly visit an ED may become knowledgeable about how specific physicians treat their patients, potentially allowing patients to manipulate physician assignment. To guard against this form of non-random physician matching, we include only index visits pertaining to a source patient's first visit to any ED in the data. Likewise, we exclude all households in which any member has been prescribed an opioid in our data at any time in the past. Anecdotal evidence suggests patients addicted to opioids may use the ED to obtain POs. If specific ED physicians acquire reputations for willingness (or reluctance) to supply these medications, it is conceivable that even first-time ED patients may attempt to strategically match to high-prescribing physicians for treatment. We avoid these potential risks to the research design by excluding all repeat ED users and all families with a history of opioid use. Note that these sampling choices will create a relatively healthy, drug-free sample, and may therefore render the estimates conservative relative to the true effect in the population.

To allow for observation of prior medical histories, we also exclude all ED visits in the year 2010. This ensures the observation window for the health histories of all household members is a minimum of one full year prior to the index visit. To account for right censoring, we include only index visits that occurred before December 31, 2013, precisely one year prior to the end of our data. Finally, we exclude children under age 18 from our sample regardless of whether they are the source or an at-risk patient. We do this because children are treated differently from adults, particularly with regard to pain symptoms. In the Appendix, we describe how patients in our analytic sample compare to patients in the full sample.

Table 3 shows descriptive statistics of variables describing the source and at-risk patients. The average source patient is almost 44 years old, and the average at-risk patient is slightly younger. This is not surprising given that the sample of at-risk patients is likely to include more young-adult children. Half of the sample of source patients are female, and about two thirds of the sample are enrolled in

Table 3. Descriptive Statistics, Analytic Sample

	Source Patient					At-Risk Patient				
	Mean	Median	SD	Min.	Max.	Mean	Median	SD	Min.	Max.
Age	43.74	45.00	14.94	18.00	75.00	41.43	44.00	15.29	18.00	75.00
Female	.50	.00	.50	.00	1.00	.51	1.00	.50	.00	1.00
Commercial HMO	.65	1.00	.48	.00	1.00	.66	1.00	.47	.00	1.00
Prior opioid use	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00
Prior benzo use	.38	.00	1.68	.00	6.00	.29	.00	1.44	.00	51.00
Elixhauser	1.08	1.00	1.58	.00	26.00	.87	.00	1.44	.00	28.00
ED opioid	.13	.00	.34	.00	1.00					
ED charge	277.76	212.39	301.75	.00	8860.30					
Number of index visits	253,407									

Note: Age is a continuous variable, *female* is a dummy variable that equals one for female patients, and *commercial HMO* is a dummy variable that equals one if the patient is covered by a commercial HMO plan. *Prior opioid use* and *benzo use* count the number of opioid and benzodiazepine prescriptions that were filled by the patient in the year prior to the index visit. *Elixhauser* is a comorbidity index (Elixhauser et al. 1998) and is computed using the diagnostic codes in a patient's medical claims in the year prior to the index visit. *ED opioid* is a dummy variable that equals one if the source patient filled an opioid prescription in three or fewer days from the index visit. If a patient does so, we assume the patient was prescribed the opioid by the ED physician. This strategy for associating prescriptions with ED visits is consistent with prior work (Barnett et al. 2017). We experimented with different cutoffs and the results are stable. *ED charge* is the total dollar amount billed for the professional services received by the patient in the index visit. Note that this does not include the facility billing (i.e., the amount billed by the hospital for use of its facilities).

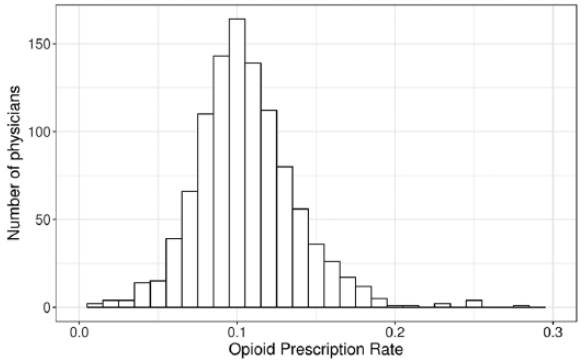

Figure 1. Distribution of Opioid Prescription Rates among ED Physicians in Massachusetts
Note: For each physician treating a patient in the ED, we compute the fraction of visits in which an opioid prescription was written. This graph shows the distribution of prescription rates of all 1,075 physicians in the sample.

a health maintenance organization (HMO) plan. The use of benzodiazepines among source and at-risk patients is relatively low, because these medications are often co-prescribed with opioids and we removed all families with past opioid use.

The Elixhauser index, which predicts health care usage and mortality, counts the number of instances in which a patient has been diagnosed with one of 31 comorbidities (Elixhauser et al. 1998). The average of the index is slightly higher among source patients than among at-risk patients, which is expected because health status weakly predicts current ED visits. Among our sample, 13 percent of patients received an opioid prescription in the ED. We link an opioid prescription to the ED visit if the source patient filled an opioid prescription in three or fewer days from the index visit. This strategy for associating prescriptions with ED visits is consistent with prior work (Barnett et al. 2017). We experimented with different cutoffs (0, 1, and 7 days) and the results are stable.[11] Finally, the average charged amount for professional services provided during the ED visit is almost $280.

Instrumental Variable

For each index visit in the sample, the IV captures the opioid prescription propensity of the ED physician who treats the source patient. The instrument, which is defined for each source patient j assigned to physician p, is technically a leave-out mean:

$$Z_{jp} = d_{jp}\left(\frac{1}{n_p - 1}\right)\left(\sum\nolimits_{k \neq j}^{n_p - 1} PO_k\right),$$

where d_{jp} is binary and indicates whether ED physician p was assigned to ED source patient j; n_p is the total number of patients seen by physician p; and k indexes the ED patients seen by physician p, where PO_k is equal to one if the ED patient filled an opioid prescription following the visit. Thus, the instrument for ED patient j's visit with physician p is the opioid prescription rate of physician p computed on all ED visits that involved physician p, except patient j's own visit. We compute this physician prescribing rate on the complete sample of all ED visits, rather than on the subset of ED visits in the analysis sample. However, we replicated these results when we constructed the instrument based on index visits in the analytic sample and results are similar.

Our analytic sample includes 1,075 ED physicians who practiced in Massachusetts between 2010 and 2015. The mean number of patients treated per ED-physician-day is 9.81. Figure 1 presents the distribution of opioid prescription rates among ED physicians. The median ED physician prescribes opioids to patients in slightly over 10 percent of ED

Table 4. Balance Statistics across Low and High Opioid-Prescribers

	Source Patient			At-Risk Patient		
	Low Prescriber	High Prescriber	*t*-statistic	Low Prescriber	High Prescriber	*t*-statistic
Prior benzo use	.38	.38	.53	.29	.29	.12
Elixhauser	1.08	1.08	–.54	.87	.87	–1.11
Age	43.61	43.88	–4.55	41.23	41.63	–8.03
Female	.50	.50	.34	.51	.51	1.27
Commercial HMO	.65	.65	–1.11	.66	.67	–3.76
Opioid prescription rate	.09	.13	–554.77			

visits. There is substantial variation around the median, with some ED physicians prescribing opioids in close to 0 percent of visits, and others prescribing opioids to over 25 percent of their patients. This significant difference in ED prescribing rates is the source of exogenous variation that we exploit to capture the causal effect of the introduction of opioids into a household on at-risk family members' subsequent opioid use.

In the first and second stages of the IV regressions, we include a vector of month-by-year fixed effects and ED fixed effects. Because patients can sometimes choose between EDs, the ED itself is not randomly assigned. Therefore, physician assignment may only be random conditional on visiting a given emergency room. If high-prescribing ED physicians cluster in EDs, and patients become aware of this and select into EDs with high-prescribing physicians, the exclusion restriction is only satisfied with a full set of ED dummy variables.

Including month-by-year and ED fixed effects effectively limits the comparisons we make to source patients at risk of being assigned to the same set of physicians. With the inclusion of these controls, we can interpret within-cell variation in the instrument, Z_{ij}, as variation in the propensity of a randomly assigned physician to prescribe an opioid relative to other ED visits to the same ED in the same month.[12]

As is always the case with an IV, there is no way to test whether ED patients with family members who have unobservable low (high) probabilities of filling an opioid prescription are assigned to a particular type of physician. However, we can compare differences in all observed characteristics of ED patients and their family members based on whether the index visit was with an ED physician whose prescription propensity is high or low.

Table 4 shows that physicians with above- versus below-median propensities to prescribe opioids are assigned to ED patients who are extremely similar in terms of their age, gender, insurance type, and consumption of benzodiazepines in the year prior to the source patient's treatment in an ED. The two variables that capture medical histories, prior benzodiazepine use and the Elixhauser index, are identical to the level of precision of the third decimal point. In the second set of columns in the table, almost-perfect covariate balance also holds for the at-risk family members of the patient visiting the ED. We do find a few minor differences in observable covariates. Source patients (and therefore at-risk family members) seen by high-prescribing ED physicians are slightly older than source patients seen by low-prescribing ED physicians, but the magnitude of the difference in age is trivial—only three months. Likewise, slightly more HMO-insured at-risk family members are treated by high-prescribing clinicians. The statistics shown in Table 4 are reassuring: they suggest the assumption that patients are assigned in a random fashion to physicians in the ED is plausible.[13]

Table 5. First-Stage Regression Coefficient Estimates

	Dependent Variable		
	Source Family Member Fills Opioid Prescription		
	(1)	(2)	(3)
ED physician opioid prescription rate	1.070***	.993***	.859***
	(.026)	(.033)	(.030)
Demographic controls	No	No	Yes
Visit controls	No	No	Yes
Medical history controls	No	No	Yes
ED FEs	No	Yes	Yes
Month × Year FEs	No	Yes	Yes
Observations	253,407	253,407	253,407

Note: Models 2 and 3 include time fixed effects and ED fixed effects. Model 3 includes demographic controls, visit controls, and medical history controls. Demographic controls included in the model are age, gender, and insurance type. Visit controls include the rate at which opioids are prescribed for the diagnosis of the source patient, whether the source patient was admitted to the hospital, whether the source patient arrived at the ED by ambulance, the dollar amount charged for the visit, and dummies for the day of the week the source patient visited the ED. Medical history controls include prior usage of benzodiazepines and the Elixhauser index.
*$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed tests).

RESULTS

Table 5 shows the first stage of the IV models.[14] The regressions are estimated using linear probability models. The outcome variable in the first-stage regressions is whether an opioid was prescribed to the source patient during the index visit. The *ED physician opioid prescription rate* is the instrumental variable. The first model only includes the main effect of the IV; Models 2 and 3 add successively more control variables. Each model shows that the ED physician's opioid prescription rate is highly predictive of the probability that a source patient receives a PO during the index visit. The effect size is large. In Model 3, with the comprehensive set of controls, a one standard deviation increase in the ED prescribing rate elevates the probability that the source patient fills an opioid prescription from 13.0 to 15.3 percent, which is a 17.7 percent increase. Beyond the fact that this result validates the relevance of the instrumental variable, it also verifies that a sizable fraction of the variance in PO rates in the ED context is determined by physician-specific proclivity to prescribe these medications.

We now estimate the main effect: what is the shift in the probability that an at-risk family member fills a PO as a result of a source family member filling an opioid prescription during an ED visit? To estimate the effect, several design choices are necessary. First, we must decide the duration of the window during which we observe the at-risk patient group. There is reason to believe that the mechanisms responsible for the causal diffusion of opioids within families will decay over time, so longer time windows almost surely will yield lower effect sizes, as both exposure and access should diminish over time. Second, we must decide whether to model the effect at the at-risk individual level or the at-risk family level. At the individual level, the outcome is whether *a specific at-risk family member* fills a PO. At the family level, the outcome is whether *any at-risk family member* fills a PO in the interval. Finally, we must select the control variables to include in the models.

To show that results are not highly dependent on any of these design choices, we estimate all combinations of regressions. We use three different time windows (3, 6, and 12

Table 6. Coefficient Estimates, Target Family Member Filling an Opioid Prescription within a Year

	Dependent Variable				
	Target Family Member Fills Opioid Prescription				
	OLS	OLS	OLS	OLS	IV
	(1)	(2)	(3)	(4)	(5)
Treatment	.023***	.021***	.021***	.027***	.130***
	(.002)	(.002)	(.002)	(.002)	(.038)
Demographic controls, source patient	No	No	No	Yes	Yes
Visit controls	No	No	No	Yes	Yes
Demographic controls, target family member	No	No	Yes	Yes	Yes
ED FEs	No	Yes	Yes	Yes	Yes
Month × Year FEs	No	Yes	Yes	Yes	Yes
Observations	253,407	253,407	253,407	253,407	253,407

Note: Each of the five regressions is estimated at the family level. Models 1 through 4 are estimated using OLS, Model 5 shows the instrumental variable estimate. Models 2 through 5 include time fixed effects and ED fixed effects. Models 3, 4, and 5 include controls for the at-risk family member, and Models 4 and 5 also include index visit controls and controls for the source family member. Because regressions are at the family level, controls for the at-risk family member are averaged if there are multiple at-risk family members.
*$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed tests).

months), two levels of analysis (family and individual), and four step-wise additions of control variables. This yields a total of 24 models using OLS and a paired set of 24 IV regressions. We report four columns of OLS estimates and one set of IV results in table format, and we present the remaining estimates in graphs to economize on space. The estimates in the table use the one-year window and the family as the unit of analysis. The four OLS models progressively add more comprehensive control variables, and the IV regression includes the most complete set of controls.

Table 6 reports the OLS and IV estimates.[15] The first four OLS models show a statistically significant effect of an opioid introduced into a household through a source patient's ED visit. The magnitude of the effect is substantial. In Model 4, which contains the complete vector of control variables, having a family member who was prescribed an opioid in the ED increases the probability of an at-risk household member also filling an opioid prescription from 14.6 to 17.3 percent. This is an 18.5 percent increase in the prescription rate.[16]

Model 5 reports the IV estimates. In this model, the treatment effect is statistically significant and the coefficient magnitude is substantially larger in magnitude than the comparable OLS estimate. Specifically, the IV estimate translates to an increase in at-risk family members' likelihood of filling a PO by 95 percent. Before we provide insight into why the IV estimate is substantially larger than the OLS estimate, we will briefly discuss the results of the other 43 regression models we estimated.

In Figure 2, Panels A and B provide information on the 24 OLS regressions, and Panels C and D present information on the 24 IV regressions. Panels A and C show effect sizes expressed as the percentage increase in the probability of filling a PO; Panels B and D show the *t*-statistics associated with the coefficient estimates of the treatment effect. Panel A shows that effect sizes using OLS range from a 14 to a 31 percent increase. Not surprisingly, effect sizes are larger for shorter time

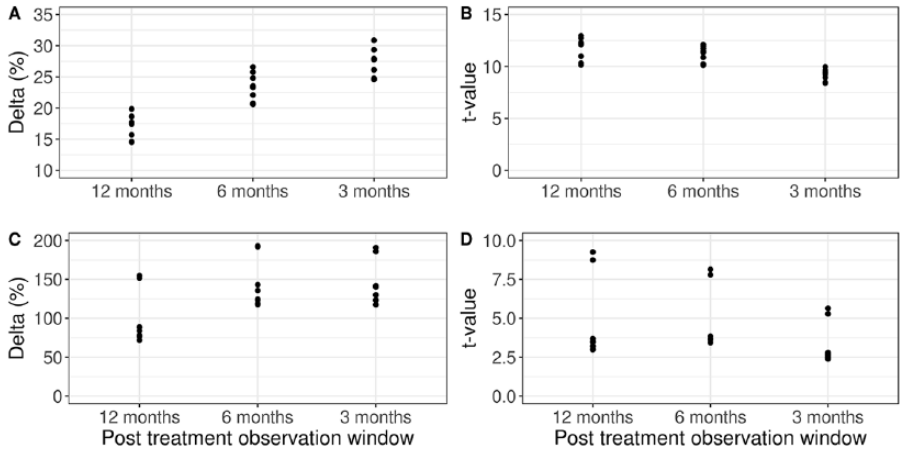

Figure 2. Estimates of Effect Size and *t*-values Using Different Model Specifications
Note: Panel A shows coefficient estimates for different model specifications using OLS; Panel B shows the associated *t*-values. Panels C and D show the coefficient estimates and *t*-values for the same model specifications but using the IV. The model specifications vary in terms of included controls (four options), length of post-treatment observation window (three options), and observations at the individual or family level (two options). The number of point estimates shown in each panel equals the number of unique combinations of model specifications: $4 \times 3 \times 2 = 24$.

windows, which is consistent with a temporal decay in the causal effect and a pattern we expected to observe. Panel B shows that all estimates are statistically different from zero. The IV estimates shown in Panel C range from a 75 percent increase to an almost 200 percent increase in the likelihood that treated, at-risk family members will fill a PO. The *t*-statistics in Panel D show that all effects in the IV regressions are statistically significant.

Why are the IV estimates of the size of the effect of intra-household exposure so much larger than the corresponding OLS estimates? The IV specification always estimates the local treatment effect (LATE), which is the component of the overall sample average treatment effect caused by the exogenous variation introduced by the IV. In our research design, this means variation in the treatment effect of intra-household opioid exposure that is created by source patients' exposure to ED physicians with different opioid prescription rates.

When the LATE exceeds the ATE, the IV estimate is larger than the OLS estimate. In our context, the IV estimate is likely to exceed the OLS equivalent if "medication non-adherence," which occurs when patients choose not to fill a prescription they receive, is common in the data.[17] Fischer and colleagues (2010) find that medication non-adherence is quite prevalent, specifically for pain medication in Massachusetts. Importantly, they also find that medication adherence rates are lower among older adults.

To understand how medication non-adherence can explain the difference between the OLS and IV results, assume there are only two types of people in the data: compliers and non-compliers. Compliers will always fill a PO if their physician recommends one, and non-compliers will never do so, perhaps because they are concerned about the addictive properties of opioids. For simplicity, further assume that family households sort on complier status, so each household contains all compliers or all non-compliers. Because of this sorting process, variation in the prescription rate of ED physicians will only be correlated with treatment status in families of compliers. In other words, the IV only drives variation in treatment in the subsample of compliers; the likelihood of assignment to treatment among non-compliers is independent of the prescribing rate of the ED

Table 7. Coefficient Estimates, Target Family Member Filling an Opioid Prescription within a Year

	Dependent Variable			
	Target Family Member Fills Opioid Prescription			
	OLS	IV	OLS	IV
	(1)	(2)	(3)	(4)
Treatment	.027***	.126***	.027***	.097*
	(.002)	(.038)	(.002)	(.038)
Observations	253,407	253,407	207,316	207,316

Note: Each of the four regressions are at the family level and include source and at-risk patient controls, visit controls, ED FEs, and month x year FEs. Models 3 and 4 include only index visits of patients who are 27 years or older. The estimates clearly show a substantial reduction in the estimated effect using IVs, but not in the standard OLS models, in the "older" sample. This finding is consistent with the mechanics of the IV operating only on compliers.
*$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed tests).

physician. The IV estimate therefore captures the LATE within the subsample of compliers, and the OLS estimate captures the average treatment effect in the full sample.

We know from Fischer and colleagues (2010) that age is correlated with complier status, so it is possible to assess whether this explanation for the difference between the OLS and IV results is consistent with the data. Because the literature establishes that prescription compliance rates are higher among younger adults, we would expect that the difference between the IV coefficient and the OLS will be largest among young adults. Results in Table 7 are consistent with this intuition. Models 1 and 2 show the OLS and IV estimates for the full sample, and Models 3 and 4 show comparable estimates for families in which the source patient is an older adult. As anticipated, the IV estimate attenuates substantially, from .136 to .108, as we move from the full sample to the older-adults-only subsample, whereas the OLS estimate is similar across the full sample and subsample. Limiting the age range of the sample does not fully or even mostly eliminate the difference between the OLS and IV estimates, but the fact that the LATE attenuates and the ATE remains consistent is all we would expect given the noisy proxy for medication adherence and the unrealistic assumptions about complete, within-household sorting based on compliance status.

To better understand the socioeconomic significance of the estimates of the treatment effects in our models, we compare the household exposure effect to other coefficients in the regression (see Appendix Table A3). For example, we know from prior research that women are more likely than men to be prescribed opioids. Our estimates are consistent with these earlier findings. The OLS regression results in Model 1 in Table A3 suggest that women are 2.2 percent more likely to fill a PO in the year following the index visit. In comparison, being exposed to POs through a family member makes one 2.7 percent more likely to consume prescription opioids. Note that the IV model suggests the gap between the two variables (i.e., female versus treatment) is much larger: 2.1 versus 13.8 percent. The effect of exposure to POs in the household is also larger than the effect of benzodiazepine use in the year prior to the index visit: a one standard deviation increase in benzodiazepine use is associated with a 1.8 percent increase in the probability that an at-risk family member will consume a PO.

Another approach to gauge the social impact of the diffusion mechanism is to pose the following question: if no household diffusion in PO use had occurred, how much lower

would the consumption of POs in Massachusetts have been? Answering this question is not straightforward for at least one reason: defining the risk set (i.e., individuals who are at risk of being newly exposed to opioid use) at the population level is complicated by the need to sample only opioid-naive families and by the fact that we are able to identify only two thirds of all families in Massachusetts, not the universe of families in the state. If we assume that all families in which members did not fill a PO in 2010 are opioid-naive, then we have 355,098 "source" patients 18 years and older that filled an opioid prescription between 2011 and 2014. These source patients introduce the medication to opioid-unexposed households with a total of 514,892 at-risk family members who are age 18 or older.

Using the range of estimates of the diffusion effect from the models presented earlier, we get a lower bound of 13,902 for the number of additional patients with opioid prescriptions because of household diffusion.[18] If we instead base the calculation on the highest estimate for the 12-month window, we have 116,520 additional patients with opioid prescriptions caused by social learning in the family. Many assumptions are required to accept these estimates and they are at best suggestive. For instance, the upper-bound estimate does not take into account diffusion of PO use inside of families that are already on opioid trajectories at the start of our observation window. Likewise, the bounds ignore hundreds of thousands of families that we cannot identify because household members are on different insurance plans. In short, these bounds are suggestive of the magnitude of the results, but if we had complete health histories and could identify all families in Massachusetts, these estimates would change significantly.

Which Behavior Does Exposure to Opioids Change?

Having established that opioid use diffuses within family households, this section examines the roles of patients and physicians in creating the link between family exposure and subsequent, higher prescription rates.

One potential source of intra-household diffusion is that at-risk family members learn about the effectiveness of opioids from the source patient and subsequently request a PO from their physician when they experience pain. In this scenario, in the absence of intra-household exposure, the at-risk patient would have been (counterfactually) less likely to request a PO. Post exposure, at-risk family members become more vocal or persistent in their request for medication.

A second possibility is that the euphoric effects of opioids trigger a desire in at-risk family members to consume opioids, even if they are not experiencing true symptoms of pain. This, of course, implies that at-risk family members consume opioids for an unintended use. Moreover, it requires that at-risk family members deceive their physicians to obtain the PO.

Because both of these behavioral changes may be behind the data-generating process, our goal in this section is to determine where the preponderance of the evidence lies. In particular, it is important to know whether the diffusion effects identified in Table 6 are more likely to have been driven by the first or second behavioral change. To explore this question, we first assess whether treated at-risk patients are more frequently diagnosed with medical conditions that commonly lead to opioid prescriptions. The rationale for this analysis is that if intra-household opioid exposure causes at-risk family members to acquire POs in situations that are not clinically justified, then self-reported, pain-related diagnoses will be more prevalent among treated family members than among the control group.

We begin by identifying medical claims with diagnostic codes that indicate chronic or acute pain, using a list of diagnoses compiled by Pasquale and colleagues (2014). Each diagnosis is associated with one or more ICD-9 (International Classification of Diseases, 9th revision) codes.[19] Appendix Table A4 shows the list of pain diagnoses we used and the associated ICD-9 codes. Using this

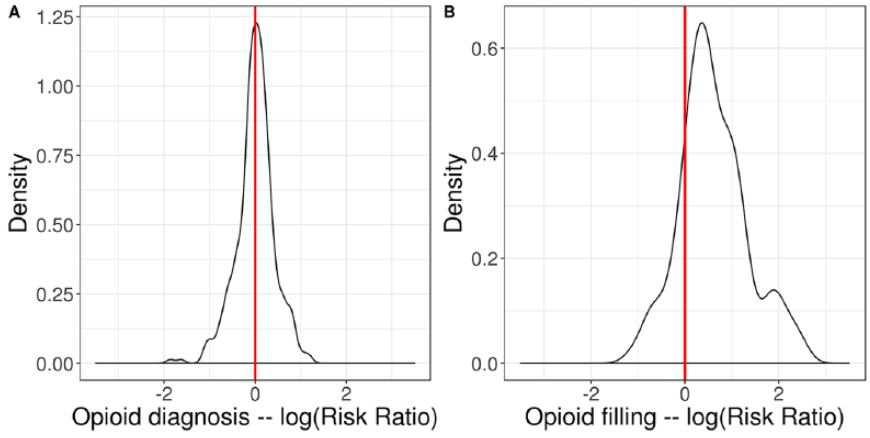

Figure 3. Distribution of Risk Ratios of Pain Diagnoses and Risk Ratios of Opioid Use
Note: In Panel A, for each family, we identify pain diagnoses received by an at-risk family member within a year of the source family member's treatment. We then compute the prevalence of each diagnosis in the sample of treated and control families, and we compute the log of the risk ratio. The graphs plot the distribution of these logged risk ratios. The vertical line indicates the point at which the prevalence is equal among treated and control families. In Panel B, for each family, we identify the first pain diagnosis received by an at-risk family member within a year of the source family member's treatment. We then compute the probability of filling an opioid prescription within three days of being diagnosed and we compute the log of the risk ratio.

list of pain diagnoses, we computed their prevalence among treated versus control, at-risk family members. The incidence of pain diagnoses is remarkably similar among both groups: 40.6 percent of individuals in the treated group were diagnosed with a condition associated with pain in the year following the index visit, compared with 40.3 percent of individuals in the control group. This tiny difference is not statistically different from zero (t-value = 1.283). As an example, consider Lumbago (ICD-9 724.2), a common diagnosis (more than 4 million cases in the data) that frequently results in an opioid prescription (10.4 percent of all cases). Lumbago is the clinical term for lower back pain. Examining the first instance in which an at-risk family member received a pain diagnosis following the index visit, the incidence is 1.5 percent in the treatment group and 1.5 percent in the control group (t-value = .035). Likewise, 3.26 percent of at-risk family members in opioid-exposed families were diagnosed with abdominal pain (ICD-9 789.0), and the incidence was 3.36 percent in control families (t-value = 1.026).

To provide a systematic comparison of the prevalence of pain diagnoses in the treated and control families, we plot the distribution of risk ratios (RR) for the prevalence of pain diagnoses among individuals in the treatment and control groups.[20] The RR is computed as the incidence of a pain diagnosis in the treatment group divided by its incidence in the control group. Panel A in Figure 3 shows the overall distribution of the RRs for 335 pain diagnoses. The vertical line represents the zero point at which the log of the RRs is equivalent in the two samples. Panel A clearly shows that although some diagnoses are more prevalent in one group than in the other, the RRs across the complete set of common pain diagnoses are closely centered around zero. In other words, treated at-risk family members are no more likely to be diagnosed with medical conditions that would increase their odds of receiving a PO than are at-risk family members in untreated families. When we further aggregate the ICD-9 codes to the 41 pain groups in Appendix Table A4, we come to the same conclusion: none of the pain diagnoses are more prevalent in the treatment group than in the control group and vice versa.

To examine whether treated, at-risk family members are more likely to request an opioid from their physician when diagnosed with a painful condition, we extract all cases in which at-risk patients were diagnosed with pain in the year following the index visit. We then computed the probability that an opioid was prescribed in the treated and control groups.[21] Consistent with earlier findings, results indicate that at-risk family members in the treated group are 30 percent more likely to fill an opioid prescription following a pain diagnosis.

We illustrate the overall effect in Panel B of Figure 3, in which we once again extract physician visits with the set of pain diagnoses for at-risk family members in the year following the index visit. Rather than computing the prevalence of pain diagnoses among at-risk patients in treated and control families, we instead compute the rate at which an at-risk family member's physician visit results in the filling of a PO conditional on visiting a clinician for one of these conditions. We then divide the prescription rate (per diagnosis) in the treatment group by the rate in the control group and take the logarithm.

Panel B in Figure 3 clearly shows that, conditional on being diagnosed with a pain diagnosis, treated at-risk patients are far more likely to receive a PO. Or, to express this differently, when a source patient introduces a PO to a household, the other household members become far more likely to leave a medical visit, which they are likely to have scheduled anyway, with a PO. The opioid prescription rate conditional on a diagnosis is unambiguously higher in treated households. Although we do not observe the conversations that occur in medical offices, these analyses strongly suggest that the diffusion of PO use within families occurs because patients in opioid-exposed households request to be treated with opioids when they visit a physician for a pain-related condition.

Heterogeneity in the Treatment Effect

We now explore potential heterogeneity in the treatment effect. In sociological terms, a heterogeneous treatment effect implies a difference between individuals in either the susceptibility of at-risk family members or the strength of social influence exerted from the source patient. We know from an extensive literature that social actors exposed to behaviors exhibited by others may respond differently following exposure.

To gauge whether there is heterogeneity in the treatment effect, we present OLS and IV estimates of the treatment effect interacted with salient observable characteristics of the source patient, the at-risk patient, and the family. Any difference in the estimated effect of treatment across these characteristics is suggestive of a differential response by at-risk family members to the introduction of an opioid to the household. The results are only suggestive because the patient and family characteristics we use to examine heterogeneity in response are unlikely to be exogenous.

As possible factors leading to variation in the treatment effect, we consider (1) gender of the source patient, (2) gender of the at-risk patient, (3) the presence of children in the household, and (4) SES as proxied by the median income in the five-digit residential zip code in which the family resides. We focus on these four characteristics for several reasons. First, each variable is of significant sociological interest. Second, men and women may exert different strengths of social influence (Aral and Nicolaides 2017) and vary in the rate at which they are prescribed opioids. Third, newspaper reports document the devastating impact of opioid addiction on families,[22] particularly for children whose parents are opioid dependent. Recent research also suggests that opioid prescriptions are associated with an increase in the likelihood that children are removed from their homes (Quast, Storch, and Yampolskaya 2018). Finally, a rich literature in sociology describes how health behaviors and the consumption of health care varies with SES. Much of this work documents that health outcomes are worse for low-SES patients, in part due to restricted access to high-quality care. Recent studies suggest, however, that POs are more likely to be written for high-SES patients, which puts these individuals at greater risk of addiction (Joynt et al. 2013).

Table 8. Coefficient Estimates for Heterogeneity in Treatment Effect

	OLS	IV
	(1)	(2)
Female at-risk (at-risk family member is female)		
Female at-risk	.011***	.014**
	(.001)	(.004)
Treatment	.021***	.104**
	(.002)	(.033)
Female at-risk × Treatment	−.002	−.027
	(.003)	(.035)
Female source (source family member is female)		
Female source	−.0003	−.006
	(.001)	(.005)
Treatment	.019***	.065
	(.002)	(.035)
Female source × Treatment	.004	.050
	(.003)	(.036)
Children in family (relationship code = child)		
Children present	.001	.002
	(.001)	(.005)
Treatment	.022***	.095**
	(.002)	(.033)
Children present × Treatment	−.004	−.009
	(.003)	(.036)
Socioeconomic status (median income in five-digit zip code)		
Medicaid pct.	−.00001	.0005***
	(.00002)	(.0001)
Treatment	.020***	.061*
	(.002)	(.029)
Medicaid pct. × Treatment	−.0002**	−.004***
	(.0001)	(.001)
Observations	379,163	379,163

Note: Each of the regressions are at the individual level and include source and at-risk patient controls, visit controls, ED FEs, and month x year FEs. All continuous variables multiplied with treatment status are mean centered.
*$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed tests).

Table 8 reports the results. We find no difference in the effect size of treatment by gender. Looking at the *female source* and *female at-risk* rows in Table 8, none of the coefficients for the interactions are significantly different from zero. F-tests also indicate a lack of variation in the treatment effect by gender. We then examine family structure. Specifically, we estimate whether adult family members with children in the household are more or less likely to fill an opioid prescription when exposed to a PO via another adult member of the family. The results indicate that at-risk parents, compared to at-risk non-parents, are equally likely to fill an opioid prescription when exposed to PO use inside the household.

Finally, we examine spatial data to gauge the socioeconomic context in which families are embedded. We proxy for a household's SES with the 2010 median income of the five-digit zip code in which the family resides.[23] Here, we do observe substantial differences in the treatment effect based on SES.

Specifically, the IV estimates suggest a small, positive, statistically significant main effect of SES on the probability of filling an opioid prescription in the year following the index visit (this association is consistent with previous findings [Joynt et al. 2013]). However, the treatment effect of intra-household exposure to POs is stronger for members of low-SES families than for members of high-SES families. The SES variable is mean centered and the results indicate that for families residing in median-income zip codes, the treatment effect is 20 percent (62 percent) in the OLS (IV) regression. Members of families in zip codes with income one standard deviation below the median, however, are 25 percent (162 percent) more likely to consume an opioid in the year following the source patient's index visit.[24]

We can only speculate on the reasons for this difference. One potential explanation is that individuals in low-SES neighborhoods are, on average, less informed about health care consumption choices, and therefore more influenced by intra-household learning. Given the well-documented and substantial variation in opioid use across spatial areas, we believe it is important to further explore the dynamics of SES and the diffusion of opioid use.

DISCUSSION AND CONCLUSIONS

Summary of the Results

We hypothesized that prescription opioid use diffuses within family households. Acknowledging the difficulties associated with making causal claims in the context of social network data, we took several steps to address these challenges. First, we used a dataset that has unusually rich micro-level data that capture demographic information and behavioral patterns describing health care consumption. Second, we subset the raw data to minimize the potential that confounding factors bias the results. For example, we limited our analyses to opioid-naive families to reduce the risk of other unobserved family characteristics driving our results. Finally, we exploited exogenous variation introduced to the data by the random assignment of physicians with different propensities to prescribe opioids.

Using these empirical strategies, we found a sizable contagion effect: through some combination of social learning and access to excess opioids in the medicine cabinet, the rate of PO use increases in families in which one person introduces these medications to the household. In light of the research design choices we made to estimate the cleanest possible treatment effect, it is probable (although not verifiable) that the intra-family social contagion effect we present is an underestimate of the true incidence of within-household contagion of opioid use in the overall population. If prior opioid use indicates a positive stance toward this class of medications (and prescription drugs more generally), we are effectively oversampling individuals and families that are apprehensive about the use of addictive medications to treat pain. In fact, the consistently larger estimates of the treatment effect in the IV specifications relative to the OLS estimations probably exists because of this latent difference between families in the dataset.

Perhaps the most novel insight to emerge from our study is not that there exists a diffusion process in family households, but that this process interfaces with the way physicians provide medical care. Members of opioid-exposed households and those in opioid-free households exhibit nearly indistinguishable post-exposure or post-pseudo-exposure health events. In other words, when we subset on health conditions that are most likely to result in a prescribed opioid, treated and untreated at-risk family members visit their physicians for these conditions at nearly indistinguishable frequencies. This suggests there is no measurable difference in the future incidence of pain-related medical conditions in the treated and untreated households. What does differ dramatically between treated and untreated household members, however, is the rate at which these two groups are prescribed an opioid when they visit their physician for pain symptoms. We find a substantial

increase in the rate at which members of opioid-exposed households are prescribed these drugs for conditions for which they, counterfactually, would have been much less likely to receive medication if they resided in opioid-unexposed households.

This finding is strongly suggestive of the mechanism that is likely at play in the data. We believe intra-household exposure or access to opioids, even when exposure is randomly generated by the matching process between patients and physicians in the emergency department, causes at-risk family members to become more aware of and knowledgeable about the efficacy of opioids. When that occurs, at-risk family members are likely to request and possibly even insist on an opioid prescription when they subsequently visit a physician for a pain-related condition. In this way, household exposure leads patients to behave differently in the future, and physicians respond because they are sensitive to patient requests for medication.

Contributions to Work on Diffusion and Medicalization

Our research contributes to the literature on diffusion processes in social networks (DiMaggio and Garip 2012). We introduced a novel empirical strategy to recover the causal effect of social learning in a network context. In doing so, we also uncovered the social process that guides the interaction between patient and physician. The findings are also pertinent to research on the role of physician networks in driving variation in costs and health outcomes (Coleman et al. 1957; Landon et al. 2012). Barnett and colleagues (2012), for example, find that the extent to which physicians are connected to one another in a patient sharing network is associated with an increase in medical spending. Our findings suggest this association may not only be driven by connected physicians adopting similar treatment behavior (e.g., high- versus low-cost care), but also by physicians shaping patient beliefs and thereby altering subsequent episodes of patient care. In other words, subsequent to instances of exposure to treatment options, patients themselves become agents of diffusion in the physician referral network.

More generally, we hope our work is a further prompt to sociologists to become more active in examining the provision of health care (Pescosolido 2006). Timmermans and Haas (2008:665), for example, ask, "What are the various health effects of medicalization?" and suggest that "mostly, we do not know because social scientists only exceptionally investigate those health effects." Here, we offer a sociological explanation for why prescription drugs are such a common treatment option for pain, but we also uncover other important puzzles that could be tackled using a sociological lens.

Staying close to the findings presented here, as a byproduct of the IV estimation strategy, we discovered substantial, inter-family heterogeneity in the probability that a treated household member will adopt opioids. The characteristic responsible for heterogeneity in the treatment effect in this context, which we labeled "drug compliance," is a latent variable (at least in our setting), but we also demonstrated that the treatment effect we identified varied substantially with SES. This finding is important because it may be a concrete step toward understanding why PO use is so prevalent in some areas of the United States but not others. Further examination of the relationship between POs and SES may also illuminate differences in the way people navigate the health care domain across the SES distribution.

Suggestions for Future Research

Along similar lines, there is a great deal of work to be done to achieve a deeper understanding of the remarkably heterogeneous opioid prescribing rates that are evident among physicians. For example, although it was not the central focus of our study, the instrumental variable we used required us to estimate the physician-specific prescribing rate for all practicing emergency department

clinicians in Massachusetts. As Figure 1 illustrated, there is substantial variation in prescribing rates in this specialty, even after conditioning out the time and location of service provision and all measurable aspects of the health conditions that are being treated. This residual, unexplained variation provides a fertile avenue for gaining a deeper understanding of the role of physicians in the opioid crisis.

Policy Implications

The results of our study have important policy implications. National Prescription Drug Take Back Day—a united effort by the Drug Enforcement Administration (DEA), the federal government, state governments, and local policymakers—aimed to collect leftover prescription drugs that have the potential for abuse. Of course, opioid analgesics top the list of such medications. The number of returned pills has increased in recent years, but a large amount of unused medication remains in households. We believe policymakers should consider providing incentives to individuals who have been prescribed drugs in large quantities in a targeted attempt to reduce the number of pills that are sitting unused in medicine cabinets across the United States.

A second policy consideration concerns the information contained in Prescription Drug Monitoring Programs (PDMPs). In states with a PDMP, every time a patient fills a prescription, it is recorded in a database. Many states have adopted PDMPs, and the data are accessible to physicians who can then view medication histories to make decisions regarding current prescriptions. Our study suggests policymakers might consider providing physicians access not only to individual patients' prescription histories but also their family members' histories. Insofar as intra-household exposure is a salient form of social learning and influence and it directly shapes patients' demand for pain medications, this information is highly pertinent to making informed decisions about the provision of medication with a high potential for abuse.

APPENDIX

Sampling Strategy

To better understand how our sampling strategy affects some of the key characteristics of the individuals we study, Table A1 shows how several descriptive statistics for our main variables change throughout the sampling process, for the source patient and for at-risk family members. Recall that several sampling steps are involved. First, we move from the full sample to the family sample by removing index visits of source patients who were not jointly covered with family members on an insurance plan in the year of the visit. We then move from the family sample to the analytic sample by including an index visit only if (1) it was the source patient's first visit to any ED in the data; (2) the source patient was part of an opioid-free household; and (3) the visit occurred between 2011 and 2013. We also removed children under age 18 from the set of source patients and from the set of at-risk family members. This sampling strategy brings the number of index visits down from 6,835,765 in the full sample to 1,709,735 in the family sample and 254,327 in the analytic sample.

Comparing source patients in the full sample to source patients in the family sample, average age decreases substantially. This is not surprising: most family health insurance plans are targeted to families with (younger) children, and because we use these plans to identify families, we are oversampling on children and younger parents. This logic also explains why we see a large difference in the percentage of patients in a commercial HMO plan. Oversampling younger ED patients also leads to a reduction in average consumption of opioids and benzodiazepines, anxiety drugs that are habit-forming and often co-prescribed with opioids. Despite the fact that patients in the family sample are younger than patients in the full sample, the rate at which they are prescribed opioids in the ED is fairly similar. Finally, the cost of treatment is slightly higher for patients in the family sample but the difference is small.

Moving from the family sample to the analytic sample, Table A1 shows that

Table A1. Sampling Statistics

	Source Patient					At-Risk Patient				
	Mean	Median	SD	Min.	Max.	Mean	Median	SD	Min.	Max.
Full Sample										
Age	42.75	43.00	21.06	.00	75.00					
Female	.55	1.00	.50	.00	1.00					
Commercial HMO	.26	.00	.44	.00	1.00					
Prior opioid use	.63	.00	1.48	.00	2.00					
Prior benzo use	1.21	.00	3.42	.00	173.00					
Elixhauser	4.26	2.00	5.31	.00	31.00					
ED opioid	.11	.00	.31	.00	1.00					
ED charge	257.27	194.00	277.89	.00	115085.95					
Number of index visits	6,816,455									
Family Sample										
Age	37.22	39.00	19.21	.00	75.00	29.71	24.00	19.33	.00	75.00
Female	.51	1.00	.50	.00	1.00	.50	1.00	.50	.00	1.00
Commercial HMO	.66	1.00	.47	.00	1.00	.67	1.00	.47	.00	1.00
Prior opioid use	.46	.00	1.25	.00	18.00	.28	.00	1.66	.00	172.00
Prior benzo use	.80	.00	2.66	.00	173.00	.29	.00	1.53	.00	62.00
Elixhauser	1.81	1.00	2.67	.00	30.00	.79	.00	1.49	.00	30.00
ED opioid	.12	.00	.33	.00	1.00					
ED charge	274.33	213.48	287.94	.00	14347.30					
Number of index visits	1,701,424									
Analytic Sample										
Age	43.74	45.00	14.94	18.00	75.00	41.43	44.00	15.29	18.00	75.00
Female	.50	.00	.50	.00	1.00	.51	1.00	.50	.00	1.00
Commercial HMO	.65	1.00	.48	.00	1.00	.66	1.00	.47	.00	1.00
Prior opioid use	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00
Prior benzo use	.38	.00	1.68	.00	60.00	.29	.00	1.44	.00	51.00
Elixhauser	1.08	1.00	1.58	.00	26.00	.87	.00	1.44	.00	28.00
ED opioid	.13	.00	.34	.00	1.00					
ED charge	277.76	212.39	301.75	.00	8860.30					
Number of index visits	253,407									

Table A2. First-Stage Regression Coefficient Estimates

	Dependent Variable
	Source Family Member Fills Opioid Prescription
Monday	.003
	(.002)
Saturday	.011***
	(.002)
Sunday	.018***
	(.002)
Thursday	.001
	(.002)
Tuesday	.004
	(.002)
Wednesday	.006*
	(.002)
Age 45 to 54	.011***
	(.002)
Age 55 to 64	.006***
	(.002)
Age 65+	−.022***
	(.002)
Female	−.010***
	(.001)
Benzodiazepine use	.006***
	(.0004)
Elixhauser	.0002
	(.0004)
Commercial HMO	−.001
	(.001)
ED diagnosis opioid prescription rate	1.697***
	(.011)
Hospital admission	.003
	(.002)
Ambulance	.024***
	(.002)
ED charge	.00001***
	(.00000)
ED physician opioid prescription rate	.859***
	(.030)
Constant	−.192***
	(.005)
ED FEs	Yes
Month x Year FEs	Yes
Observations	253,407

*$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed tests).

excluding children naturally pushes up the age of source patients in the analysis sample relative to the average patient in the family sample and full sample. Because we focus on opioid-naive families exclusively, the opioid prescription rate in the year prior to the index visit is reduced to zero and the prescription rate of benzodiazepines declines substantially. The percentage of patients receiving an opioid in one of the index visits remains remarkably stable. Similar patterns hold for at-risk patients.

Table A3. Coefficient Estimates, Target Family Member Filling an Opioid Prescription within a Year

	Dependent Variable	
	Target Family Member Fills Opioid Prescription	
	OLS	IV
	(1)	(2)
Age 45 to 54	.003	.004*
	(.002)	(.002)
Age 55 to 64	.003	.004
	(.002)	(.002)
Age 65+	−.001	.001
	(.004)	(.004)
Female	.022***	.021***
	(.002)	(.002)
Commercial HMO	.001	−.0005
	(.005)	(.005)
Benzo use	.011***	.010***
	(.001)	(.001)
Elixhauser	.016***	.016***
	(.001)	(.001)
Family size	.059***	.059***
	(.001)	(.001)
Treatment	.027***	.130***
	(.002)	(.038)
Constant	−.027***	−.019**
	(.005)	(.006)
Demographic controls source	Yes	Yes
Visit controls	Yes	Yes
ED FEs	Yes	Yes
Month x Year FEs	Yes	Yes
Observations	253,407	253,407

*$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed tests).

Full Sets of Coefficient Estimates for First- and Second-Stage Regressions

Table A2 shows the coefficient estimates of the variables included in the first-stage regression. The model presented here is identical to Model 3 in Table 5. The regression estimates show that ED physicians are most likely to prescribe opioids to their patients on weekends, and opioids are most commonly prescribed to patients age 45 to 64. Women are prescribed fewer opioids than men in our ED sample, and patients with a history of benzodiazepine use are more likely to be prescribed opioids. Not surprisingly, if a patient is diagnosed with a condition for which opioids are commonly prescribed, the likelihood of being prescribed an opioid increases. Patients brought into the ED by ambulance are also more likely to receive an opioid prescription. If being brought in by ambulance is an indicator of severity of the condition, this result is not surprising.

Table A3 shows the coefficient estimates of the OLS and IV models that feature the richest set of control variables. Model 1 is identical to Model 4 in Table 6, and Model 2 is identical to Model 5 in Table 6. The coefficient estimates shown here pertain to the variables that describe the at-risk patient. Female at-risk family members are more likely than male at-risk members

Table A4. Pain Diagnoses and ICD-9 Codes

Diagnosis	ICD-9 codes
Back pain	720.0–724.9
Pathologic fracture/osteoporosis	733.XX
Rheumatoid arthritis	714.XX
Myofascial pain	719.4X, 729.5, 729.91, 729.95
Migraine	346.XX, 784.0
Gout	274.XX
Osteomyelitis	730.XX
Sickle cell disease	282.6
Fibromyalgia	729.1
Postamputation	895.XX–897.XX, 353.6
Diabetic peripheral neuropathy	250.6X, 357.2X
Postherpetic neuralgia	053.1
Trigeminal neuralgia	350.1
Chronic postoperative	338.22, 338.28
Chronic pain due to trauma	338.21
Chronic pain syndrome	338.4
Other disorders of peripheral nervous system associated with neuropathic pain	353.1, 353.8, 353.9, 354.1–354.3, 354.5, 354.8, 354.9, 355.1–355.6, 355.8
Abdominal pain	557.1, 789.XX
Dysmenorrhea	625.3, 306.52
Endometriosis	617.XX
Interstitial cystitis	595.1
Chest pain cardiac	786.5x
Chest pain pulmonary	786.0X–786.4X
Depression	296.2X–296.8X, 298.0X, 300.4X, 311.XX
Psychogenic pain	307.8X
Other chronic pain	338.21, 338.29, 338.4
Complex regional pain syndrome	337.21, 337.22, 354.4, 355.71
Central pain syndrome	338.0
Chronic pancreatitis	577.1
Regional sprains or strains	840.XX–847.XX
Burns	94X.X
Postoperative pain	338.12, 338.18
Childbirth	650.XX–677.XX
Spinal cord injury	806.XX, 952.XX
Spine fracture	805.XX
Hip fracture	808.XX, 820.XX, 835.XX
Other fractures	800.XX–804.XX, 807.XX, 809.XX–818.XX, 821.XX–827.XX, 829.XX
Other injuries – dislocations (excluding hip)	830.XX–834.XX, 836.XX–839.XX, 848.XX
Other injuries – wounds, blood vessels, superficials, crushing, injury to nerves (excluding spinal cord)	870.XX–899.XX, 900.XX–924.XX, 926.XX–951.XX, 953.XX–959.XX
Other injuries – vehicular accidents	E80, E82–E84
Acute pancreatitis	577.0

to fill an opioid prescription within a year from the index visit. Less healthy at-risk patients, as captured by their prior benzodiazepine use and their higher Elixhauser index, are also more likely to consume opioids. Finally, family size is positively correlated with an at-risk family member filling an opioid prescription in the year following the index visit.

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Notes

1. For more details, see https://www.cdc.gov/drugoverdose/maps/rxrate-maps.html.
2. For more details, see http://www.alicerap.eu/resources/documents/doc_download/139-policy-paper-4-prescription-opioids-and-public-health.html.
3. See https://www.health.state.mn.us/data/economics/docs/opioidbrief20185.pdf.
4. See http://ldh.la.gov/assets/docs/BehavioralHealth/Opioids/OpioidPrescriptionsFactSheet.pdf.
5. Shah, Hayes, and Martin (2017), for example, find that 20 percent of patients who start using prescription opioids are still consuming these medications a year later.
6. Benzodiazepines are a class of medications commonly co-prescribed with opioid analgesics.
7. Note that we do not require random assignment. All that is required for the instrument to be valid is that the assignment of patients to a physician is independent of the physician's opioid prescription rate.
8. The small differences are likely due to individuals who cross the Massachusetts state line. For example, young adults may move across state lines yet still be insured under their parents' health insurance plan. This group will be included in the CHIA data but will not be tallied in the census data.
9. During the observation window, some individuals switch insurance plans; some transition from individual to family plans and vice versa. We include families in our sample if, at the time of the index ED visit, the source patient is covered under a family plan. We replicated all our results using a looser definition of the family unit. Specifically, our findings are robust to inclusion of all family units in which the family plan existed in the year prior to the index visit. Our findings are also robust to exclusion of all family units in which the insurance plan was terminated within a year of the index visit.
10. The main regression specifications in this article use a one-year window following the index visit, but we re-estimated the effect sizes for multiple time windows and describe results from six-month and three-month windows following the index visit. We anticipate larger effect sizes in shorter time windows, because there will be over-time attenuation in the exposure effect.
11. Note that we only observe prescriptions that are filled. Not all prescriptions written by physicians are filled by patients. We address this issue in more detail when we compare the OLS estimates with the IV estimates for our main model.
12. The instrumental variable calculation is not conditioned on characteristics of the patient or the treatment of the patient, which allows us to directly examine the sensitivity of the results to the inclusion of controls.
13. As mentioned earlier, the minimum requirement for our instrument to be valid is that the patient-physician match is independent of the opioid prescription rate of the ED physician.
14. The full set of coefficient estimates in Model 3 is presented in Appendix Table A2.
15. The full set of coefficient estimates from Models 4 and 5 is shown in Appendix Table A3.
16. The unadjusted difference in the probability of filling an opioid prescription in the year following the index visit is 2.3 percentage points (16.6 percent – 14.3 percent).
17. Note that we observe all prescriptions in Massachusetts that were billed to insurers, but the data do not include prescriptions that were written but never filled by the patient.
18. This estimate uses a 14.6 percent base rate of adoption and a 2.7 percentage point increase in that rate as a result of household exposure as per Table 6, Model 4.
19. The International Classification of Diseases is a hierarchical classification system that organizes medical diagnoses. Diagnosis codes have a minimum of three and a maximum of five digits, but in the MA APCD, less than 1 percent of claims report a three-digit ICD-9 code.
20. We aggregate all claims up to the four-digit level of the ICD-9 hierarchy to make sure there are a substantial number of observations within each diagnostic code. For example, abdominal pain, right upper quadrant (ICD-9 789.01) and abdominal pain, left upper quadrant (ICD-9 789.02) are aggregated up to ICD-9 code 789.0 (abdominal pain). Because there are seven times as many observations in the control group as in the treatment group, we only compare the four-digit ICD-9 bins that contain at least seven control-group cases.
21. To link an opioid prescription to the visit in which the patient was diagnosed with any of these pain diagnoses, we use the strategy described earlier: opioids filled within three days of a visit are associated with that visit. For this variable, we also experimented with different cutoffs (0, 1, and 7 days) and the results are stable across each definition.

22. See, for example, https://www.nytimes.com/2017/12/28/opinion/opioid-crisis-children-foster-care.html.
23. We also constructed the proportion of Medicaid-insured patients in the five-digit zip code, which varies by year. The two measures yield nearly indistinguishable results.
24. Note that it is again likely that the gap between the OLS and the IV estimate is caused by differences in medication adherence between low- and high-SES families.

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