Essays in Applied Microeconomics

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To my mother

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Abstract

This thesis is composed of three articles. The first two chapters study the impact of pharmaceutical promotion of opioid analgesics in the US. In the first chapter, we find that counties, where sales representatives of opioid drugs reach more doctors, have higher opioid overdose mortality rates. We also show that doctors receiving promotion for opioid drugs have higher opioid prescription rates. The second chapter examines the role of opioid promotion and opioid painkiller availability on non-poisoning suicide rates. We find that promotion of opioid drugs can increase suicide rates but that this effect is mitigated in counties with strong addiction-help networks. In the final chapter, we analyze the effect of exposure to civil conflict violence on voting behavior. Using data from elections in Bosnia and Herzegovina between 1990 and 2014 and exploiting variation in war intensity across municipalities, we estimate that violence towards civilians had a negative impact on voter turnout.

Resumen

Esta tesis está compuesta de tres capítulos. En los primeros dos capítulos se estudia el impacto de la promoción farmacéutica de los analgésicos opiáceos en Estados Unidos. En el primer capítulo se encuentra que los lugares donde los agentes de ventas farmacéuticas visitaron a un mayor número de doctores, tienen mayores tasas de muertes por sobredosis de opiáceos. Se muestra que los doctores que son visitados por los agentes de ventas escriben más recetas médicas incluyendo opiáceos. El segundo capítulo estudia el rol de la promoción y disponibilidad de opiáceos en suicidios no causados por envenenamiento. Los resultados indican que la promoción de opiáceos puede aumentar la tasa de suicidios pero que este efecto es mitigado en lugares con fuertes redes de apoyo para la adicción. En el tercer capítulo, se analiza el efecto de estar expuesto a violencia causada por un conflicto civil sobre el comportamiento electoral. Usando datos de elecciones en Bosnia y Herzegovina entre 1990 y 2014, y aprovechando diferencias en la intensidad de la guerra entre municipalidades, se estima que la violencia en contra de personas civiles tiene un efecto negativo en la participación electoral.

Preface

This doctoral thesis combines three research projects at the intersection between development economics, health economics and political economy. In the first chapter, co-authored with Fernando Fernandez, we estimate the effect of pharmaceutical promotion of opioid drugs to physicians on opioid-related adverse health outcomes in the US at the county-level. The sales of opioid painkillers nearly quadrupled in the US since 1999. Opioid-related adverse health outcomes such as addiction, overdose, death and the number of babies born with severe withdrawal syndrome after in-utero exposure to opioids increased by similar magnitudes. Our results indicate that counties, where sales representatives of opioid drugs reach more doctors, have higher opioid overdose mortality rates. In addition, we find that infants born in counties with higher opioid promotion during pregnancy are more likely to present symptoms in line with the neonatal abstinence syndrome. We identify the effects by using the presence of state-level bans on pharmaceutical promotion to physicians and the distance between counties and pharmaceutical companies' headquarters to instrument opioid promotion. To study the link between worsened health outcomes and opioid promotion, we use Medicare prescription data and show that doctors receiving promotion for opioid drugs prescribe more opioid painkillers.

In the second chapter, co-authored with Mark Borgschulte, we study the relationship between opioid painkiller availability and non-poisoning suicide rates in the US. We show that more people commit suicide in counties, where many doctors receive pharmaceutical promotion for opioid drugs. The positive relationship between promoting opioid drugs to physicians and suicide rates is mitigated in counties with strong addiction-help networks, suggesting that opioid painkiller availability increases the number of individuals with substance abuse disorders. The positive relationship is also mitigated in states with mandatory access prescription drug monitoring programs, which track patients' and physicians' opioid prescribing history. Doctors in these states react less towards opioid promotion in terms of opioid prescriptions.

The last chapter, co-authored with Caterina Alacevich, investigates the effect of exposure to civil conflict violence on voting. Using data from elections in

Bosnia and Herzegovina between 1990 and 2014 and exploiting variation in war intensity across municipalities, we estimate that violence towards civilians had a negative impact on voter turnout. The effect is stable and persistent over twenty years after the war resolution. Our results are robust to the inclusion of pre and post-war socioeconomic and political characteristics, to instrumental variable estimations based on terrain ruggedness, and to restricting the sample to voters who were too young to be selectively targeted. We measure conflict intensity by the share of war casualties. Distinguishing between civilian and military victims, we show that violence towards civilians is driving the negative effect. Next, we examine different mediating mechanisms related to forced migration and demographic selection, ethnic composition, labor market conditions, physical capital damage, and post-conflict reconstruction. The results support the hypothesis that war exposure directly affects voting behavior. Using survey data, we show that respondents in municipalities with higher shares of civilian casualties report lower generalized trust, lower trust in institutions, and a lower propensity to vote.

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Chapter 1

CAN PHARMACEUTICAL PROMOTION TO PHYSICIANS LEAD TO ADVERSE HEALTH OUTCOMES? EVIDENCE FROM THE OPIOID EPIDEMIC IN THE US

Joint with Fernando Fernandez (UPF)

1.1 Introduction

Every ten minutes one US-American dies from drug overdose (CDC, 2016). Since 1999, the rate of drug overdose deaths has nearly quadrupled, with opioid prescription overdoses accounting for 40% of the overdose deaths in 2014 (CDC, 2015). Incidents of drug overdoses have increased so drastically that all-cause mortality rates for white non-Hispanics in the ages between 45 and 54 years rose in the last decade, reversing the long-run trend of decreasing mortality rates in previous decades (Case and Deaton, 2015). The number of opioid pain relievers

prescribed in the United States skyrocketed in the same period, with no simultaneous increase in pain reported by patients (Chang et al., 2014). The public costs of the epidemic are not limited to higher mortality rates. The misuse of opioids contributed to the increase in hospitalization rates[1] and the number of babies born with neonatal abstinence syndrome. Babies born to women taking opioid drugs during pregnancy are more likely to suffer from respiratory and feeding problems, to be born prematurely and to be admitted to the neonatal intensive care unit (Tolia et al., 2015).

Why did health care professionals increase their opioid prescription rates so extensively in the last two decades? In the 1990s health experts in the US increasingly became concerned with the optimal management of pain. For example, pain was classified as the fifth vital sign, next to body temperature, pulse rate, respiration rate and blood pressure. At the same time, state medical boards started to relax restrictions on prescribing opioid drugs for the treatment of non-malignant chronic pain. Pharmaceutical companies initiated aggressive marketing campaigns to promote opioid medication as an effective treatment option for non-terminally ill pain patients to health care professionals. Some of the manufacturers downplayed the risk of addiction and other adverse health outcomes, partly relying on limited or faulty empirical evidence (Van Zee, 2009).

This study examines the impact of pharmaceutical promotion of opioid analgesics targeted to health care professionals on opioid-related adverse health outcomes in the US in 2014 and 2015. We identify the effects by using the presence of state bans on pharmaceutical promotion to physicians and the distance of the counties to the pharmaceutical companies' headquarters as instruments for receiving pharmaceutical promotion.[2] We find that higher promotional activities for opioid analgesics were associated with higher mortality rates from opioid overdoses in 2014 and 2015. The most conservative estimate of our instrumental variable (IV) regressions indicates that increasing the number of doctors reached by sales

[1] According to the CDC, more than 1,000 US-Americans are admitted to the emergency room every day because of abuse of opioid drugs (Crane, 2013). They also estimate that one out of four patients who receive prescription opioids are struggling with addiction (SAMHSA, 2014).

[2] Engelberg et al. (2014) follow a similar empirical strategy by instrumenting promotion to physicians using the distance to the closest headquarters of pharmaceutical manufacturers. They analyze the prescription behavior of Medicare Physicians in the US in 2013 and consider the promotion of all types of drugs.

representatives by 1% increases overdose deaths by 0.16% (the 95% confidence interval ranges from 0.03% to 0.3%). This means that reducing promotion in the average county to zero would decrease opioid-related overdose death rates by 1.9 per 100,000 inhabitants (0.2 standard deviations). Besides mortality, the use of opioid painkillers has been linked to higher rates of neonatal abstinence syndrome. We, therefore, explore whether promotional intensity of opioid painkillers in a county is also related to adverse neonatal health outcomes. Our IV estimates indicate that ten additional doctors receiving opioid promotion in a county in the nine months prior to birth lead to an increased likelihood for a baby i) to be born with a low birth weight by 0.5 percentage points, ii) to be born prematurely by 1.2 percentage points and iii) to need assisted ventilation by 0.3 percentage points. These numbers are not negligible because the probability of an infant to be born with symptoms in line with withdrawal is low. On average 15 physicians receive opioid promotion in the nine months prior birth in the county of birth. An increase of 15 physicians leads to an increase of babies needing assisted ventilation for more than six hours by 0.5 percentage points which is 5% of the standard deviation of the outcome variable.

To shed light on the mechanism of increased overdose death rates and worsened neonatal health outcomes, we show that doctors receiving promotion for opioid drugs have higher opioid prescription rates. The IV regression implies that promotion has a positive and statistically significant effect on the number of opioid prescriptions with an elasticity of 0.1. The estimates are within the range of elasticity coefficients found in other work analyzing the impact of pharmaceutical promotion on prescription behavior. Kremer et al. (2008) conduct a meta-analysis on the impact of pharmaceutical promotion and find elasticity estimates between 0.05 and 0.15.

Why do physicians prescribe opioid painkillers so extensively despite potential negative health consequences for their patients? One important reason is that medical research on the effectiveness and side effects of opioid analgesics in combating chronic non-cancer pain was scarce, until recently. In recent years, medical research has concluded that there is no evidence for the effectiveness of long-term opioid therapy for improving non-malignant chronic pain, while there is a risk of dependency (Chou et al., 2015). Manchikanti et al. (2012) argue that inappropri-

ate prescription patterns lie at the heart of the epidemic, resulting from knowledge deficits and (wrongly) perceived safety of opioid drugs. They state that 60% of all overdose deaths occur while patients are following the physicians' prescriptions (CDC, 2012). Patients who are prescribed opioids can also acquire opioid painkillers illicitly or switch to illegal opioid drugs, such as heroin. According to the National Survey on Drug Use and Health (NSDUH), between 2002 and 2011 80% of recent heroin initiates report prior use of opioid pain relievers (Muhuri et al., 2013).

The incidence of neonatal abstinence syndrome increased in similar magnitudes as opioid overdose deaths in the last decade (Tolia et al., 2015). Recent medical research shows negative neonatal health outcomes after in-utero exposure to opioids (Patrick et al., 2015). There is no clear empirical evidence yet on the long-run consequences of suffering from neonatal abstinence syndrome. There is, however, evidence for a steep rise in health care expenditures due to increasing hospitalization rates and associated charges (Patrick et al., 2012). Additionally, studies show a significant negative relationship between low birth weight and long-run outcomes, such as educational attainment and earnings (Behrman and Rosenzweig, 2004; Black et al., 2007; Royer, 2009).

We combine county-level data on death rates (CDC Wonder, December 2016) with recently released and rich data on pharmaceutical promotion payments to physicians aggregated at the county-level (CMS, 2016). We first establish that opioid promotion and overdose death rates are positively correlated using OLS estimations. We then use a difference-in-difference estimation to show that the positive correlation between promotion and death rates is not driven by unobservable time-invariant county characteristics. The level of promotion, however, is unlikely to be exogenously distributed across counties with respect to opioid overdose death rates. The promotion of opioid painkillers could, for example, be higher in counties where demand for those products is higher. Promotion could also be higher in places with low demand for opioid drugs if pharmaceutical companies are trying to open new markets.

To establish causality, we adopt an instrumental variable approach, in which opioid promotion is instrumented with the distance of the counties to the pharmaceutical companies' headquarters and the presence of state bans on pharmaceuti-

cal promotion to physicians. The estimation of the causal effect of pharmaceutical promotion on death rates is robust to several specification checks. First, to rule out the concern of endogenous sorting of headquarters, we only include companies that had opened their headquarters before 1995, the onset of large-scale promotional activities of opioid analgesics. Many of these remaining headquarters opened in the 19th century, rendering the concern of endogenous sorting less likely. Second, we control for county characteristics that could potentially correlate with the counties' locations and opioid overdose rates. Economic conditions, such as unemployment rates, are shown to be important determinants of prescription pain reliever use (Carpenter et al., 2017). The robustness of our results to the inclusion of these county characteristics limits the concern that we are solely picking up a county-specific, time variant relationship of higher demand for opioid pain relievers and ultimately more overdose deaths. Third, we take advantage of the fact that the states of Minnesota, Vermont, and Massachusetts introduced some form of ban on pharmaceutical promotion to physicians at different points in time to limit promotional activities towards physicians.[3] Importantly, pharmaceutical promotion to physicians in these states is banned or limited for every type of drug, not opioid painkillers in particular. We show that, prior to the introduction of these bans, the trends in opioid overdose rates of the introducing states were statistically indistinguishable from the rest of the US. This result suggests that the three states did not introduce the state bans as a response to increasing overdose death rates.

To analyze the impact of opioid promotional activities on neonatal health outcomes, we use the CDC 2014 Natality Detail Data Set. We aggregate promotion in the nine months prior to the birth in the county of birth. Medical research points out that in-utero exposure to opioids in the third trimester of the pregnancy is particularly detrimental for neonatal health outcomes (Desai et al., 2015). In line with this finding, we document that promotion in the third trimester of the preg-

[3]Minnesota introduced the law in 1997, while Vermont and Massachusetts introduced it in 2009. Vermont bans most gifts from pharmaceutical manufacturers to health care professionals, while Minnesota allows gifts with a value of less than $50 per year. Massachusetts initially strictly prohibited pharmaceutical and medical device sales representatives from providing any meals of any value but amended the law in 2012. Now meals can be provided to health care professionals if they are of "modest value".

nancy displays the highest correlation with negative health outcomes. This helps us to rule out the concern that counties with high opioid promotion rates are just counties with higher morbidity rates in general and thus adverse neonatal health outcomes. Promotion in the first and the second trimester should show similar correlations with adverse health outcomes if counties with a generally unhealthy population receive high levels of promotion.

To study the link between worsened health outcomes and opioid promotion, we show that pharmaceutical promotion of opioid painkillers increases opioid prescription rates. We use physician level Medicare Part D prescription data for 2013 and 2014 and follow the same empirical strategy as in the county level analysis. We instrument the receipt of a physician's opioid promotion with the proximity of the physician's practice to an opioid producing company's headquarters and the presence of a state ban on pharmaceutical promotion to physicians. Physicians write more opioid prescriptions if they receive opioid promotion in the corresponding year. Their opioid prescription behavior, however, is not influenced by pharmaceutical promotion of other drugs. This substantiates the interpretation that it is not pharmaceutical promotion per se, but the specific promotion of opioid medications, that is driving increases in opioid prescriptions.

We find similar results on the relation between promotion and opioid overdose death rates if, as an alternative, we instrument the number of physicians who receive opioid promotion with the number of physicians in the respective county who receive promotion for drugs unrelated to pain and opioids (such as blood thinner and diabetes medication). The idea behind this alternative instrument is that physicians receive opioid promotion simply because the sales representatives are also promoting unrelated drugs. Finding coefficient estimates of comparable magnitudes increases our confidence of a causal relationship between promotion and overdose deaths.

Since the data on promotional activities is only available from August 2013 onwards, we cannot use our data to explain the overall increase in drug poisoning mortality over time. Our approach, however, is useful to understand why some places have much higher rates of drug overdose mortality than others. McDonald et al. (2012) document large geographic variations in opioid prescription rates in the US in 2008 and argue that differences in morbidity in the population cannot

explain these variations. The total amount of opioids dispensed in counties in the 75th percentile is four times larger than the total amount dispensed in counties in the 25th percentile.

This paper contributes to a growing literature on policies addressing the opioid epidemic in the US. Researchers find that improving access to opioid antagonists such as naloxone can decrease opioid abuse and related health outcomes (Mueller et al., 2015; Rees et al., 2017). Declines of overdose death rates have been found for the introduction of "Good Samaritan Laws" which provide immunity from prosecution for drug possession to anyone who is experiencing an opiate-related overdose or is observing one and is seeking medical attention (Rees et al., 2017). Others analyze the impacts of the introduction of state-level prescription drug monitoring programs (Borgschulte et al., 2018; Kilby, 2015; Dave et al., 2017). Bachhuber et al. (2014) establish that opioid-overdose related death rates decreased in states that legalized the use of medical marijuana. The idea is that the use of opioid painkillers is reduced due to the availability of an alternative non-opioid painkiller to combat chronic or severe pain.

As pointed out, physician knowledge deficits appear to be one of the core causes of the opioid epidemic. Researchers have thus tried to understand which factors determine such deficit. Currie and Schnell (2018) find that physicians who graduated from higher ranking medical schools prescribe significantly fewer opioids. Previous work establishes that pharmaceutical promotion to physicians influences their prescription behavior (Datta and Dave, 2017; Engelberg et al., 2014; Kremer et al., 2008). To the best of our knowledge, this is the first study to examine whether opioid painkiller promotion to physicians plays a significant role in explaining the opioid epidemic.[4]

Our paper also contributes to the literature on the political economy of special interest groups. Special interest groups (SIGs) aim to influence welfare relevant institutions to further their cause. Well-known examples are lobbying groups that intend to influence politicians, bureaucrats and the media (Grossman and Helpman, 2001; Mian et al., 2010; Reuter and Zitzewitz, 2006). Similarly, pharma-

[4]David et al. (2010) find a positive relationship between different kinds of pharmaceutical promotion of drugs for certain conditions and adverse drug events, such as overdoses and allergic reactions, in the US.

ceutical companies affect the prescription behavior of health care professionals through pharmaceutical promotion. The interaction between SIG and institutions may, in principle, benefit welfare as the SIG can share valuable and specific information. However, the SIG's optimal choice of information disclosure does not necessarily maximize public welfare. The opioid epidemic exemplifies the large welfare costs that can arise from such information asymmetry.

The paper is structured as follows. Section 1.2 provides background information on the practice of pharmaceutical promotion to physicians in the US. Section 1.3 describes the data sources and provides basic descriptive statistics. Section 1.4 discusses the empirical strategy, followed by the estimation results (Section 1.5). Section 1.5.3 explores the channel of increasing prescription rates. Section 1.5.4 reports robustness checks and Section 1.6 concludes.

1.2 Background Information: Pharmaceutical Promotion to Physicians

Pharmaceutical promotion to physicians is a common practice in many countries. Pharmaceutical companies in the US spend billion dollars every year on advertisement of their drugs and medical devices. The largest share of their advertisement budget is generally devoted to direct advertisement to physicians and other health care professionals (Cegedim, 2013). In 2012 pharmaceutical companies spent 27 billion USD on promotion – more than 24 billion USD directed towards physicians. A nationally representative study showed that more than 80% of all physicians in the US received some form of gift by a pharmaceutical representative in 2004 (Campbell et al., 2007).

In the economic literature, previous studies show that interactions of physicians with pharmaceutical sales representatives influence the prescribing practices of the former. Engelberg et al. (2014) find that physicians receiving promotion of branded drugs reduce prescription rates for generic drugs and increase prescriptions in favor of the paying firm's drugs (similarly Datta and Dave (2017)). Other work suggests that promotional activities lower the price sensitivity of general practitioners (Windmeijer et al., 2006).

It is important to understand why promotional efforts change prescription behavior: do pharmaceutical companies provide new information or are physicians' incentives distorted due to financial motives? Physicians may act in the best interest of their patients by prescribing the promoted drug if the pharmaceutical company uses the sales representatives visits to inform about new drugs, their effectiveness and side effects. However, patient health may be adversely affected if the provided information is incorrect or the physician's decision making is distorted by rent-seeking behavior. It is difficult to empirically differentiate between the two mechanisms of information acquisition and rent-seeking behavior. Engelberg et al. (2014) find that payments cause shifts in prescriptions towards branded drugs over generic equivalents, arguing that additional information cannot play a significant role in explaining the effectiveness of promotion. Without data on the information provided to the physician, it is impossible to rule out the explanation of new information acquisition as sales representatives can, for example, emphasize that their drug causes fewer side effects even when they are talking about pharmaceutical equivalents.

In promoting directly to physicians, pharmaceutical sales representatives have room for misinformation. Studies show that the information provided by sales representatives is not always accurate. Villanueva et al. (2003) assess the accuracy of promotional material circulated by pharmaceutical companies in Spain and conclude that in 44% of the claims made in advertisements, the references provided did not support the statements. Similar results have been found for promotional material distributed in the US. In the study by Wilkes et al. (1992) they ask medical professionals to assess the accuracy of statements made in pharmaceutical advertisement. For 44% of the claims, the reviewers feel that it would lead to improper prescription behavior if a physician had no other information about the drug.

Purdue Pharmaceuticals was among the first companies promoting the opioid analgesic OxyContin, for the treatment of chronic (non-cancer related) pain in 1996. In its promotional campaign, Purdue asserted that the risk of addiction from OxyContin was extremely small and sales representatives claimed that the risk of addiction was less than 1%, a statement that cannot be backed up with empirical evidence from medical studies (Van Zee, 2009). Purdue's sales grew from $48

million in 1996 to $1 billion in 2000. Simultaneously, its number of sales representatives doubled from 1996 to 2001 (GAO, 2003). During the late 1990s, other pharmaceutical manufacturers followed the promotional efforts of Purdue and extended the marketing of their opioid pain relievers. The key message of these campaigns was that opioids can be used to treat long-term pain of non-terminally ill patients. Promotion was not only directed at pain specialists, oncologists or palliative care specialists but also at primary care physicians (Van Zee, 2009). As stated in the previous section there is no evidence for the superiority of opioid drugs over other medications and forms of therapy in improving non-malignant chronic pain. There is, however, evidence for the risk of dependency, overdose death and negative health consequences for unborn babies who are exposed to opioids in-utero.

A growing number of legal actions against opioid manufacturers suggests that this commercial success has not been harmless. For instance, in 2007 Purdue Pharmaceuticals pleaded guilty to the charges of the misbranding of OxyContin and paid a fine of $634 million. In the past two years, different counties have pressed charges against some of the pharmaceutical companies promoting opioid medications for misbranding and underrepresentation of the risk of addiction.[5] Pfizer Pharmaceuticals and the City of Chicago reached a settlement in 2016 in which Pfizer committed to disclose in their promotional material the risk of opioid medication and stop the promotion for "off-label" uses, such as long-term back pain. Additionally, they admitted that there is no convincing empirical evidence for the long-term use of opioid medication (for more than 12 weeks), in non-terminally ill patients. Compared to the other opioid producing pharmaceutical companies, Pfizer's sales of opioid medications are small.

The Centers for Medicare and Medicaid Services (CMS) publishes data on a yearly basis on the promotional payments made by manufacturers to physicians and teaching hospitals, who are covered under one of the three federal programs. These data on promotional activities are available from August 2013 until December 2015. In Figure 1.2 we split counties into high and low promotional activity

[5]The City of Chicago, Orange County and Santa Clara Counties filed lawsuits against Purdue Pharma LP, Teva Pharmaceutical Industries Ltd, Johnson & Johnson, Endo Health Solutions Inc and Allergan PLC in 2014.

counties and show the evolution of overdose death rates over time. Counties are defined as high promotion areas if promotional activities for opioid medication are above the median level of activity in the years 2013-2015. The median number of physicians receiving opioid-related promotion between 2013 and 2015 is 27 in a given county. Overdose rates between high and low promotion are statistically indistinguishable between 1982 and 1998. Overdose rates for high promotion areas start to increase at a higher rate than in low promotion areas, providing qualitative evidence for our hypothesis.[6]

1.3 Data and Descriptive Statistics

We combine multiple sources of data to conduct our analysis. An overview of all datasets used and the corresponding time periods can be found in Table A-1.

Following the introduction of the Physician Payments Sunshine Act in 2010, all manufacturers of drugs and other medical supplies that have at least one of their products covered by one of the three federal health care programs (Medicare, Medicaid, and State Children's Health Insurance Program), must disclose their financial relationships with physicians and teaching hospitals. Manufacturers are required to submit data on payments made to covered recipients, with information on the amount, the date, the nature of the payment and to which drug it relates to the Centers for Medicare & Medicaid Services (CMS). The CMS provides open access to the payment data (CMS, 2016). The payment data used in this study covers the period from January 2014 to December 2015. The data is available from August 2013 to December 2016. Our main outcome of interest, opioid-related overdose death rates, are only available for the years until 2015. We, therefore, restrict our analysis to 2014 and 2015, the two years for which we have information on both payment data and overdose death rates.

[6]For the years before 1999, we observe overdose mortality rates for opioid-related drugs only in counties with more than 100,000 inhabitants. Calculations in Figure 1.2 are based on 403 counties for which we have data over the entire time span. In the Appendix, we show that before the expansion of pharmaceutical promotion of opioid drugs for non-terminally ill patients in 1996, the mortality rates are following a parallel trend (see Figure A-1a). For the years from 1999 on we have mortality data for all counties. In Figure A-1b, we can see that mortality rates are statistically significantly higher in counties that receive high levels of promotion from 2005 on.

We are primarily interested in payments made to physicians and teaching hospitals regarding opioid medication. These payments can be made for research activities, gifts, in the form of speaking fees, meals, or travel. The dollar amount in the dataset can thus refer to the amount directly paid to the physician for speaking fees or represent the dollar value of the lunch or other gifts.

The payment data provides the National Drug Code (NDC) of the drug the payment was made for. With the NDC Drug Code Directory published by the U.S. Food and Drug Administration (FDA), we obtain details on the drug, such as the substance names that allows us to classify the drug group. We classify a drug an opioid analgesic following the Anatomical Therapeutic Chemical (ATC) Classification System of the WHO (ATC code N02A). We exclude opiates that are given to patients to reverse opioid overdose, such as naloxone.[7] If a payment occurred for more than one drug, we split the amount paid by the number of drugs promoted.

Table 1.1 presents summary statistics for the payments made in 2014 and 2015. On average, 11 doctors in a county received promotion for opioid medication in 2014. Not all payment entries are complete: we can see that in both years around 30% of the payments made do not have a drug identifier. Some measurement error in our independent variable is likely, as there is reason to believe that also some transactions regarding opioid medication are not classified as such. We expect a downward bias in the reporting of the payments. Pharmaceutical companies may have an incentive to under-report payments because it is difficult to detect such underreporting and because the information on the payments made is freely accessible for all patients, all physicians, and their competitors. Patients who observe the financial relations of their physician with pharmaceutical companies may question the physician's prescription recommendation.

On average, pharmaceutical companies spent 1,200 USD per county for opioid promotion in 2014. Average spending on opioid promotion increased from 2014 to 2015 to 2,500 USD. Many counties (in 2015 more than 50%) do not receive any pharmaceutical promotion for opioid medications according to the Open Payment Data. The data indicates that physicians and teaching hospitals receive on average visits by one opioid manufacturer a year. This suggests that the different

[7] See Table A-2 in the Appendix for a list of keywords used.

manufacturers seem not to be competing in convincing physicians to prescribe their opioid over a different opioid (intensive margin). It is possible that manufacturers are targeting physicians to prescribe opioid painkillers over alternative treatment options. Manufacturers spent, on average, 2,400 USD in 2014 to promote painkillers, other than opioid analgesics. In 2015, pharmaceutical companies spent less money on promoting non-opioid painkillers to physicians, compared to 2014.

Our outcome of interest is the count of opioid overdose deaths at the county level. We use the Multiple Cause of Death Data from 1999 to 2015, provided by the Center for Disease Control (CDC Wonder, December 2016). The Multiple Cause of Death Dataset is constructed from summarizing death certificates provided by state agencies. Even though every death certificate includes a single underlying cause of death, up to twenty additional causes can be indicated in the certificate. The death counts reported in this dataset summarize the number of times that a particular cause of death has been mentioned. This means that a deceased person can be counted as having died from an opioid-related overdose and as having died from cancer. The WHO and the CDC (guideline for opioid prescription in March 2016) recommend the prescription of opioid medication for terminally-ill or cancer patients. We do not want to make welfare statements about terminally-ill patients who instead of dying from their fatal disease, die from an overdose of opioid medication. We, therefore, subtract from the count of the fatalities caused not only by overdose but also by neoplasms (ICD-10 Code: C00-D48) the count of deaths by neoplasms only, to obtain the count of fatalities due to opioid overdose only. Table 1.1 summarizes the mortality rates for opioid overdoses for the years 2014 and 2015 (ICD-10 Code: T40.0-T40.4).

To calculate the distance of the counties' centroids to the headquarters of the opioid promotion pharmaceutical companies, we retrieved the location of the headquarters and their opening date from the web pages of the companies. Table A-3 in the Appendix displays the list of companies that have been promoting opioid medication to physicians in 2014 and 2015, according to the CMS Open Payment Data. Headquarters are excluded from our final analysis if they have been opened after 1995 and for pharmaceutical companies that generate most of their

revenues from opioid medication (Purdue, INSYS).[8] We consulted state legislations for the presence of some form of state bans on pharmaceutical promotion to physicians. In Minnesota gifts to physicians with a value of more than 50$ are prohibited since 1997[9], while Vermont[10] and Massachusetts[11] introduced limits on gifts to physicians in 2009. The state of Massachusetts amended the law in 2012, allowing pharmaceutical and medical device representatives to provide meals to health care professionals outside their office of "modest value". This value is not further specified. In none of the states are financial relations between physicians/hospitals and pharmaceutical companies completely banned.

We use the CDC 2014 Natality Detail Data Set to analyze the impact of promotional activities on neonatal health outcomes. The data set contains information on all available births registered in the US in 2014. It provides information on the county and month of birth, mother's characteristics such as demographics and health status, information on delivery and prenatal care and neonatal health outcomes. Summary statistics are depicted in Table 1.2. We calculate promotion exposure by summing the number of physicians that received opioid promotion in the nine months before the birth of the child in the county of birth, normalizing by county population. On average 15 physicians received opioid promotion in the county of birth in the nine months prior to the birth. Neonatal health outcomes in line with the neonatal abstinence syndrome are rare: 8% of all babies are admitted to the neonatal intensive care unit (NICU), 1% of the neonates need assisted ventilation for more than six hours after birth. Around 11% of babies are born prematurely (before gestational week 37) and 8% have low birth weight (less than 2500g).

Another data source used is the Medicare Provider Utilization Data 2013 and 2014 collected by the CMS. These files contain information on Medicare Physi-

[8]The results are not sensitive to the exclusion of these two companies. Results are available upon request.

[9]Minnesota Statues 151.461: `https://www.revisor.mn.gov/statutes/?id=151.461` (accessed on July 31, 2017).

[10]Vermont Statues 18 V.S.A. § 4632: `http://legislature.vermont.gov/statutes/section/18/091/04632` (accessed on July 31, 2017).

[11]Commonwealth of Massachusetts Statues 105 CMR 970.000: `http://www.massmed.org/Advocacy/Regulatory-Issues/Overview-of-Massachusetts-Physician-Gift-Ban-Law/#.WWY6fumxWbg` (accessed on July 31, 2017).

cians, such as their names, specialties and addresses and the number of opioid prescriptions they wrote in 2013 and 2014. These are the two most recent files available. For 2014 we have data on the entirety of payments made, while for 2013 the payments are only available from August to December. We use the prescription data of 2013 to control for the lagged prescription behavior of the physician. We cannot run a difference in difference regression due to the lack of data on payments made before August 2013.

Table 1.3 summarizes the average number of opioid claims made by Medicare Physicians in 2014 and the payments they received from pharmaceutical sales representatives in 2014. The average Medicare Physician prescribes 106 opioid prescriptions per year. 2.6% of all physicians in this dataset receive promotion for opioid medications and 5.5% of the opioid-prescribing physicians. If a physician receives promotion from pharmaceutical companies for opioid, he/she receives a payment of 100 USD in one year, on average. There is large variation across physicians in the number of opioid prescriptions made (up to 26,500 claims) and the average number of all drug services performed by the physician. The mean distance to the closest headquarters of a physician is about 800km and around 5% of Medicare Physicians work in a state that has some form of ban on pharmaceutical promotion to physicians in 2014. To receive more information on the characteristics of the physician, we merge the prescription data from 2014 with the most recent Medicare Physician Compare data provided by the CMS. This data set includes information on the gender of the physician, his/her graduation year and hospital affiliations, if available. Average characteristics can be found in Table 1.3. 60% of doctors for whom this information is available are male and on average they graduated from medical school in 1994. Another characteristic we would like to analyze is whether a physician is affiliated with a hospital with strict conflict of interest policies. Unfortunately, we only have information available on these policies for teaching hospitals in the US and not the universe of hospitals. The AMSA scorecard assigns grades to all medical schools based on policy domains regulating the interaction of the student with the pharmaceutical industries.[12] We can see that this information is only available for 67,000 physicians

[12]These domains are i) whether it is forbidden to accept meals and gifts from pharmaceutical sales representatives, ii) whether sales representatives have access to school facilities, iii) whether

in the Medicare Part D prescription data set and that of 90% are affiliated with a hospital that bans sales representatives from entering the hospital.

Lastly, we collect socio-economic county characteristics that could correlate with opioid overdose mortality rates from different data sources. Medicare Part D enrollment data for 2013-2015 is provided by the CMS. The Bureau of Labor Statistics produces unemployment rates and industry employment shares at the county level for the years 2013-2015. We classify counties into two categories of urbanization (urban/rural) according to the NCHS Urban-Rural Classification Scheme for Counties 2013 (Ingram and Franco, 2012). The U.S. Census Bureau provides in their "Small Area Income and Poverty Estimates (SAIPE) Program" estimates on county poverty rates and median household income levels for the years 2013-2015. Table A-4 summarizes county characteristics for 2014 and 2015.

1.4 Empirical Analysis

1.4.1 Pharmaceutical Promotion and Opioid Overdose Deaths

The goal of the empirical analysis is to test whether pharmaceutical promotion of opioid drugs is related to drug overdose deaths. Our conceptual framework includes three agents: pharmaceutical companies, physicians, and patients. Pharmaceutical companies invest in promotion of their drugs. Physicians decide whether to prescribe opioid drugs or not. Patients receive their treatment and health outcomes (e.g. drug overdoses) are determined. We expect that higher levels of pharmaceutical promotion of opioid drugs are related to higher numbers of fatal drug overdoses through an increase in the prescription of these drugs.

As a starting point, we use cross-sectional variation in pharmaceutical promotion to explain drug overdose deaths by running the following OLS regression:

$$OD_c = \alpha_s + \beta^{OLS} Prom_c + X_c^{'}\Gamma + \varepsilon_c \tag{1.1}$$

the school has a formal curriculum on conflict of interests iv) how well the policies are enforced and sanctioned and v) other domains.

where OD_c denotes the opioid overdose death rate in county c, normalized by the county population (100,000 inhabitants). State fixed-effects are captured by α_s. The vector X is included to control for socio-economic conditions at the county-level such as Medicare enrollment rates, poverty rates, and labor market conditions. Our measure of pharmaceutical promotion at the county level is $Prom_c$. Finally, ε_c denotes the error term.

We observe promotion and overdose deaths for two consecutive years (2014, 2015). This allows us to run a fixed effect regression which controls for time-invariant county characteristics and addresses potential targeting bias at the county level. The next equation we estimate is:

$$OD_{c,t} = \theta_1 CountyFE_c + \theta_2 TimeFE_t + \beta^{FE} Prom_{c,t} + X'_{c,t}\Gamma + \varepsilon_{c,t} \quad (1.2)$$

It is likely that the OLS estimates are biased because of omitted variables and/or measurement error. One possibility is that pharmaceutical companies may be targeting physicians and counties who have a high demand for opioid drugs instead of causing high demand. They could also target counties with initially low demand for opioid painkillers to open new markets by convincing physicians of the advantages of opioid painkillers over alternative treatment options. The fact that physicians are on average visited by one manufacturer only hints to the interpretation that sales representatives try to convince physicians of the superiority of opioid painkillers over alternative treatment options. If pharmaceutical companies were trying to convince physicians, who already write many opioid prescriptions to prescribe their drug, we would observe that multiple manufacturers promote to physicians. Next to the omitted variable bias, OLS regression results may suffer from measurement error. Pharmaceutical companies have, as argued earlier, an incentive to under-report payments made to physicians, especially regarding controlled drugs such as opioids in a period of heightened public attention. 30% of all payments made by manufacturers do not have a drug identifier and it is reasonable to assume that also payments regarding opioid painkillers were not reported.

To overcome these issues, we propose the following IV strategy. We use two instruments for promotion: the distance between the county centroid and the closest headquarters of opioid manufacturers, and the presence of state laws banning

pharmaceutical promotion to physicians. The idea behind the first instrument is that we expect that counties closer to firms' (i.e. opioid producers) headquarters are more likely to receive promotion of opioid drugs. This relationship could arise, for instance, because managers located in the headquarters can monitor sales representatives more easily or sales representatives can reach these counties easier. Additionally, sales representatives are reimbursed for their travel expenses by the manufacturers. The further they travel, the higher the costs for the pharmaceutical company (MedReps, 2017). As described in Section 1.3 three states (Minnesota, Vermont, Massachusetts) have introduced some forms of state bans on pharmaceutical promotion. The three states have introduced state bans for all kinds of pharmaceutical promotion, not opioid medication in particular. We show in the robustness checks (Section 1.5.4) that the introduction was not related to differential trends in overdose death rates in these states. The presence of these state bans thus provides additional exogenous variation in the likelihood of physicians receiving promotional material related to opioid analgesics directly from the manufacturers. The drawback of this approach is that both instruments do not vary over time and we can only exploit cross-sectional variation.

This setup leads us to estimate the first-stage equation:

$$Prom_c = \phi + \rho_1 Dist_c + \rho_2 Ban_c + X_c^{'}\Psi + \mu_c \tag{1.3}$$

where we predict the promotion of opioid drugs, $Prom_c$, with the distance to the closest headquarters of opioid manufacturers, $Dist_c$ and the presence of state bans, Ban_c. We presume ρ_1 to be negative because promotion is expected to be lower in counties further away from headquarters. Similarly, ρ_2 should be negative because counties with bans are less likely to receive promotion. The vector X denotes the above-described county controls. These county characteristics should account for the fact that the location of the counties may be correlated with socio-economic characteristics, which also determine opioid overdose rates.

The second-stage equation is:

$$OD_c = \alpha + \beta^{IV} \widehat{Prom_c} + X_c^{'}\Gamma + \varepsilon_c \tag{1.4}$$

where $\widehat{Prom_c}$ is the prediction from the first-stage (Equation 1.3). The parameter of interest is β^{IV}, which captures the effect of pharmaceutical promotion of opioids on overdose deaths. If this coefficient is positive, it implies that promotion increases deaths related to opioid overdoses. The identifying assumption for the IV estimation is that distance to the closest headquarters and state bans only affect drug overdose deaths through the promotion of opioid drugs. We deal with some concerns related to this assumption in Section 1.5.4.

1.4.2 Pharmaceutical Promotion and Neonatal Health Outcomes

The use of opioid painkillers and illicit opioid in pregnant women increased in the last decade (Desai et al., 2014; Bateman et al., 2014), despite evidence for detrimental health outcomes for unborn babies. With this empirical analysis, we investigate whether the negative health impact we observe in opioid overdose deaths rates can also be found in neonatal health measures. We analyze whether the intensity of opioid promotion in the county of birth of a newborn in the nine months prior to delivery is negatively related to health outcome measures. For this, we regress the number of doctors that received opioid promotion on neonatal health outcomes following the same empirical approach as depicted in Section 1.4.1. We instrument the number of physicians receiving promotion with the distance of the county centroid to the closest headquarters and the presence of a state ban on promotion. We will display OLS regression results and the first and second stage of the 2SLS estimations. In all regressions, we include mother characteristics at birth, such as demographics and health measures, delivery information (prenatal care, the form of delivery, whether a physician attended the delivery) and neonate characteristics (gender, birth order and the number of babies born). We control for month of birth fixed effects and state fixed effects. Medical research has found an increase in respiratory and feeding problems in neonates after in-utero exposure to opioids. The babies are more likely to need assisted ventilation, to be admitted to the neonatal intensive care unit, to have low birth weight and to be born prematurely. We regress the number of opioid receiving physicians in the county of birth on the before mentioned health outcomes. We also analyze the impact on the APGAR 5 score, as it includes a score on how well the infant is breathing after

delivery. The literature has found that these effects are particularly pronounced after exposure in the third trimester and long-term exposure. We, therefore, investigate whether late exposure has larger negative impacts on health outcomes. The variation in promotion is at the county level such that we cluster standard errors at the county level.

1.4.3 Channel: Promotion and Prescriptions of Opioid Drugs

Physicians' prescription behavior is the main channel through which pharmaceutical promotion to physicians affects patient health. To document the relationship between opioid drugs prescription and pharmaceutical promotion of such drugs, we follow the same approach as in Section 1.4.1 using physician-level information. We estimate the following first and second stage equations:

$$Prom_{i,t} = \pi + \gamma_1 Dist_i + \gamma_2 Ban_{is} + \theta Spec_i + \zeta Pres_{i,t-1} + \nu_{iz} \tag{1.5}$$

$$Pres_{i,t} = \lambda + \delta^{IV} \widehat{Prom_{i,t}} + \kappa Spec_i + \eta Pres_{i,t-1} + \epsilon_{iz} \tag{1.6}$$

We instrument opioid promotion to Medicare physicians using the distance of the office to the closest opioid promoting headquarters ($Dist_i$) and the presence of a state ban on promotion (Ban_{is}). We control for the specialty of the physician, denoted by $Spec_i$, and the number of opioid prescriptions issued in the previous year ($Pres_{i,t-1}$) in the first and second stage.

$Pres_{i,t}$ is equal to the number of prescription claims of opioid drugs written by physician i in year t. We use different measures of $Prom_{i,t}$. First, we create a dummy variable equal to one if physician i received payments related to opioid drugs from pharmaceutical companies in the corresponding year, and zero otherwise. Second, we use the (log) dollar amount of the payments made from opioid manufacturers to physician i. We sum up all payments a physician has received in a corresponding year. The error term is denoted by ϵ_{iz}, as we cluster standard errors at the zip-code level. According to our hypothesis, we expect δ^{IV} to be positive, suggesting that higher promotion of opioid drugs is associated with more prescriptions of such drugs.

1.5 Results

1.5.1 Promotion and Mortality of Opioid Overdoses

We begin by presenting the OLS estimates of the association between promotion of opioid drugs and opioid overdose mortality. In Table 1.4, we report the estimated coefficients of Equation 1.1. The point estimates in columns 1 and 2 are both positive and statistically significant, indicating that higher promotion is correlated with higher death rates. These figures imply that increasing the number of doctors reached by sales representatives by 1% increases the number of opioid overdose deaths by 0.1%. Column 3 in Table 1.4 suggests that contemporaneous promotion of opioid medication is related to opioid overdoses while pre-year levels of promotion have no significant relationship with overdoses. The different measures of promotion imply different elasticities: increasing the dollar amount spent on opioid promotion in a county by 1% increases the death rate by 0.05%.

The county fixed effect regressions display smaller coefficients than the OLS results and are less precisely estimated, mainly because we have less variation within counties over time than across counties. In Table 1.5 we can see that increasing the number of physicians receiving promotion by 1%, increases the number of opioid deaths by 0.04%. Again, the coefficients on the dollar amount spent are smaller than the one on the number of physicians reached, but it is not statistically significant at conventional levels. Although these figures are suggestive, it is problematic to provide a causal interpretation to these estimates due to omitted variables concerns.

Thus, we turn to discuss the IV results, reported in Table 1.6. We pool the regression results for all our estimates from here on for the two years 2014 and 2015 together.[13] The OLS estimates display coefficient estimates of the same magnitude for the two years, such that we can pool our data to increase efficiency. In column 1, we use the distance to the closest headquarters as one of the instruments for promotion. One potential concern with this instrument is that firms choose the headquarters location based on factors related to marketing activities. These factors can be correlated with opioid overdose deaths. To deal with this issue, in

[13] IV regression results for 2014 and 2015 separately are very similar and available upon request.

columns 2 and 3 we restrict the headquarters to those opened before 1995, the year before the beginning of promotional activities of opioid drugs.[14] We present both sets of results to demonstrate that endogenous sorting of pharmaceutical headquarters is not a threat to our identification strategy.

The first stage results in Panel A display that the closer a county is to a headquarters, the more doctors receive promotion for opioid medication. This is true for both sets of considered headquarters. Dropping the before described companies decreases the coefficient estimates in the first and second stage. The first stage also reveals that the state bans on pharmaceutical promotion appear to be effective: states with a ban have significantly fewer doctors receiving promotion. The partial F-Value of the two used instruments can be found in the last row of Table 1.6. Our instruments are strong and work in the expected direction.

The second-stage results show that promotion of opioid drugs and overdose deaths are positively linked. The regression results indicate that increasing promotion by 1% in the respective year increases deaths rates by 0.33%. Compared to the OLS estimates, these coefficients are much larger, suggesting that the latter were potentially downward biased. Engelberg et al. (2014) follow the same identification strategy and also find higher coefficient estimates in the IV regression compared to the OLS results. They argue that the IV coefficients may be larger as closeness to headquarters does not only increase the likelihood of receiving promotion that is ultimately displayed in the Open Payment Data but also other forms of promotions, such as marketing events or conferences.

In the third column, we additionally control for county characteristics. The county characteristics we control for are shown to be important determinants of opioid overdose rates (Carpenter et al., 2017). For example, unemployment rates are positively correlated with overdose death rates and explain around 2% variation in deaths in our study period. The characteristics we include are unemployment rates, population, the share of the population that is enrolled in the Medicare Prescription Drug Plan, industry shares, income levels, poverty rates and an urbanization dummy. The coefficient on promotion remains unchanged when we

[14]Table A-3 lists the manufacturers promoting opioid analgesics in 2014 and 2015, the date of their headquarters opening and a dummy indicating whether they are included in the reduced set of headquarters.

control for these variables. The robustness to the inclusion of the county characteristics limits the concern that we are only picking up a relationship of higher morbidity and therefore higher demand for opioid pain relievers and ultimately more overdose deaths. Additionally, other work suggests that state variation in opioid prescription patterns cannot be explained by underlying health status differences of the population (Paulozzi et al., 2014).

We measure the intensity of opioid promotion with the number of doctors receiving promotion for the following reason. We are not differentiating between the informative and persuasive nature of promotion. If additional information is driving changes in prescription rates, there is no reason to believe that every additional dollar given to one physician would change her/his prescription patterns in a linear way. In Section 1.5.4 we perform multiple robustness checks. We can show that our results carry through if instead of proxying promotional levels with the number of doctors we proxy it with the logarithm of the USD amount given to physicians. Again as in the OLS and fixed effect regressions, the coefficients are around half the size compared to the coefficients on the number of physicians. All these findings indicate that the effect on the extensive margin of promotion is larger than on the intensive margin: reaching many physicians with sales representatives has higher elasticities than spending more money on the same physicians.

1.5.2 Promotion and Neonatal Health Outcomes

The positive relationship we have documented between opioid promotion and death rates can also be found in terms of negative neonatal health outcomes. Table 1.7 displays the OLS regression results described in 1.4.2. It shows the relationship between the number of physicians receiving opioid-related promotion in the nine months prior to delivery on the following health outcomes: the infant was admitted to the neonatal intensive care unit (NICU), the infant needed assisted ventilation i) right after birth and ii) for more than six hours, the infant's APGAR score in minute 5, its birth weight and whether she/he was born prematurely. A baby is considered to have low birth weight if its weight is below 2500g. Prematurity is defined by neonates born at less than 37 weeks' gestation. Panel A of Table 1.7 shows that opioid promotion is correlated with more babies being admitted to

the NICU, needing assisted ventilation for more than six hours, being born prematurely, with low birth weight and low APGAR 5 score. There is no statistically significant relationship between promotion and the need for assisted ventilation immediately after birth. Promotion is normalized by ten, meaning that an additional ten physician receiving promotion is associated with a lower birth weight of a baby born in the corresponding county of 4.7 gram. On average 15 physicians in a county receive opioid promotion. The probability of neonates being born with symptoms in line with NAS is generally low. The relationship between promotion and negative health outcomes is therefore sizable: an increase of 15 physicians leads to an increase of babies needing assisted ventilation for more than six hours by 0.1 percentage points which is 10% of the mean of the outcome variable.

Panel B of Table 1.7 splits the promotion into in which trimester of the pregnancy the promotion occurred. In line with previous findings of the medical literature, promotion levels in the third trimester of the pregnancy are associated with the largest impact on negative health outcomes. Low birth weight is positively associated with promotion in all trimesters with similar magnitudes. We are regressing promotion on many health measures and therefore need to account for multiple hypothesis testing. We display the Bonferroni adjusted p-values in Panel A and Panel B. All coefficient estimates in the regressions on promotion during the entire pregnancy are still statistically significant at conventional levels. The coefficient estimate on promotion in the third trimester on low APGAR 5 score loses statistical significance.

Table 1.8 depicts the results of the first and second stage regressions of the 2SLS equation described in Section 1.4.2 and 1.4.1. We instrument the number of physicians that received opioid promotion in the nine months prior to delivery with the distance of the counties centroid to the closest headquarters promoting opioid medication and the presence of a state ban on pharmaceutical promotion to physicians. Panel A shows that again the coefficients following the IV estimation are larger than in the OLS estimation, but we lose precision in the estimates. We find a statistically and economically significant relationship between promotion levels and the probability of neonates being born prematurely, with low birth weight and needing assisted ventilation for more than six hours after birth. Ten additional physicians receiving promotion leads to an increase in the likelihood of

a neonate needing assisted ventilation for more than six hours of 0.3 percentage points, which is one-third of the mean of the outcome variable (0.03 of a standard deviation). For the remaining outcome variables, the coefficient estimates have the same sign as in the OLS regressions, larger magnitudes but lack statistical significance at conventional levels. The coefficient estimates and the partial F-Values of the first stage are displayed in Panel B of Table 1.8. Being born in a county far away from opioid producing headquarters reduces the number of physicians receiving promotion and so does living in a state with a ban on pharmaceutical promotion to physicians. The regression shows a strong first stage with F-Statistics around 40.3.[15]

To be able to derive policy implications, it is important to understand for which mothers opioid promotion appears to have a detrimental effect on the baby's health outcomes. Our data allows us to analyze heterogeneous effects of promotion on health outcomes by the age of the mother, whether the mother is a smoker and by insurance status. Previous research establishes that physicians are more likely to prescribe opioids to Medicare or Medicaid patients (Olsen et al., 2006). Medical research also shows that opioid use is particularly detrimental for the unborn if accompanied with additional risk factors, such as smoking during pregnancy, or alcohol abuse (Desai et al., 2015). The negative effects of promotion on neonatal health outcomes are not driven by mothers who smoke, nor by mothers below the age of 30. The effects are indeed slightly larger for smoking mothers, but also non-smokers are affected. The effect is entirely driven by women who are Medicaid recipients (44% of mother's in our sample are Medicaid recipients). For mothers with private insurance, there is no effect of promotion on neonatal health outcomes. It is possible that receiving Medicaid is a proxy for mothers with worse health status. It is also possible that physicians prescribe opioid painkillers to patients more often if they are covered by Medicaid than to patients who are covered by private fee-for-service insurance. Estimation results of the heterogeneity analysis can be found in Table A-5 in the Appendix.

[15]When we instrument promotion in the third trimester only, our coefficient estimates double in size, in line with the findings of the OLS regressions. Statistical significance does not change compared to the specification in which we measure promotion during the entire pregnancy. Results are available upon request.

1.5.3 Promotion and Prescription Behavior

After establishing a positive link between promotion and opioid overdose deaths and neonatal health outcomes, we turn our attention to the mechanism. The key channel between promotion and negative health outcomes is physician prescription behavior. Table 1.9 reports the OLS estimates from regressing prescription claims on pharmaceutical promotion. Our results show that physicians receiving promotion - measured as the dollar amount of payments or as an indicator of receiving payments - write more prescription of opioid drugs. We control for county fixed effects, the specialty of the physician and opioid prescription rates in the previous year. Column (1) suggests that physicians who receive any promotion write on average 45 opioid prescriptions more than physicians who receive no promotion.[16] The results in column (2) suggest that increasing the dollar amount given to a physician in the form of opioid-medication promotion by 100% leads to an increase of 15 additional opioid prescriptions. Table 1.9 also displays the regression results of the first and second stage of Equations (1.5) and (1.6). As in the regression of overdose mortality rates at the county level, we find that distance decreases the likelihood of receiving pharmaceutical promotion and so does the presence of a state ban. Partial F-statistics of the first stage result can be found in the last row of Table 1.9, showing that our instruments are highly relevant in explaining differences in promotion to physicians. The set of considered headquarters is the reduced set explained in Section 1.3. Our estimates here are very comparable to the coefficients we have found in the OLS estimations. They imply that increasing the USD given to a physician for opioid promotion by 100% increases opioid prescriptions by 14. The elasticity in the OLS and IV regressions are identical and of magnitude 0.1 (see Table A-6). These estimates are in line with elasticity coefficients found in other work. Kremer et al. (2008) conduct a meta-analysis on the impact of pharmaceutical promotion and find elasticity estimates between 0.05 and 0.15.

In Table 1.10 we run a placebo regression to show that it is not promotion per se, but particularly promotion regarding opioid medications, that is driving increases in opioid prescriptions. The regression shows the relationship of the

[16] To see results for other empirical specifications, see Table A-6 in the Appendix.

promotion received by the physician for different drugs and the number of opioid claims by the physician. Payments made for non-opioid non-painkiller drugs have no impact on the number of opioid prescriptions. The positive coefficient we find on painkillers, other than opioids, can be explained by the fact that these two sets of drugs are sometimes prescribed jointly for the pain management of patients. The coefficient on opioid promotion is very comparable to the one we find in Table 1.9, where we do not control for promotion other drugs.

To rule out that opioid promotion is driving up prescriptions for all kinds of drugs, we regress the share of prescriptions for opioid drugs over all prescriptions on opioid promotion. Table A-7 displays regression results for OLS estimates with the share of opioid claims over all claims as a dependent variable. The table indicates that opioid promotion is not driving up total drug claim rates but in particular the share of opioid claims overall drug claims. Again, receiving promotion for non-opioid painkiller drugs or non-painkiller drugs (column 2) does not increase the share of opioid claims.

Next, we investigate which characteristics determine whether a physician receives opioid promotion and how much he or she reacts. Some hospitals have conflict of interest policies in place that are similar to the state bans on pharmaceutical promotion to physicians discussed earlier. Some hospitals ban pharmaceutical or medical device sales representatives from entering the hospital or offer classes on how to deal with conflicts of interest. The American Medical School Association (AMSA) collects data on these policies for all medical schools in the US since 2008. We expect physicians affiliated with a hospital with conflict of interest policies in place to first be less likely to receive opioid-related promotion and second to adjust their opioid prescription behavior less after engaging with sales representatives. Unfortunately, this data is not available for the universe of hospitals but only for teaching hospitals. We can therefore solely analyze the behavior of Medicare Part D physicians who are affiliated with a teaching hospital in 2014. Table 1.11 displays the heterogeneous effects of receiving opioid promotion on opioid prescription rates. Column (1) shows that physicians affiliated to a hospital where sales representatives are not allowed access to any faculty or trainees react less to opioid promotion than physicians who are affiliated with a teaching hospital without such policies. In column (2) we add additional physician characteristics

that could potentially influence the sensitivity towards promotion. Previous literature established that male physicians are more sensitive towards pharmaceutical promotion (Engelberg et al., 2014). We also find that male physicians react more strongly to opioid promotion than female physicians (column (2) in Table 1.11). We do not find that physicians that graduated before 1995 react differentially towards opioid promotion. The idea here is that physicians that graduated before the outbreak of the opioid epidemic may be less trained in pain management using opioid painkillers and thus react more to information provided by sales representatives. Physicians affiliated with a hospital with a ban on sales representatives do prescribe more opioid prescriptions if they receive any kind of promotion regarding opioid drugs. The opioid prescriptions increase is 50% smaller compared to the physicians who are affiliated to a teaching hospital without such a ban. This finding should not be interpreted in a causal manner: physicians with stricter opinions about how health care professionals should interact with the pharmaceutical industry could choose to work for hospitals reflecting his/her opinion. In the last column (3) we analyze which characteristics predict whether a physician receives opioid promotion. Male physicians are more likely to receive promotion and so are physicians who graduated before 1995. Physicians affiliated with a hospital that does not allow sales representatives to engage with its staff are naturally less likely to be visited by a sales representative promoting opioids.

Physicians receiving promotion of opioid medication prescribe more of these drugs because either they receive potentially biased information or because they value the payments made by companies. Although we cannot distinguish the relative importance of these alternative explanations, these estimates indicate that promotion is positively related to prescriptions which lead to adverse health outcomes, such as death and neonates suffering from withdrawal.

1.5.4 Robustness Checks

Our main empirical analysis relies on the assumption of the exogeneity of our instruments. We use the presence of state bans on pharmaceutical promotion and the distance to the closest headquarters to instrument the likelihood of a county receiving pharmaceutical promotion related to opioid analgesics. We show that the

introduction of the state bans was orthogonal to the evolution of opioid-related overdose deaths in the respective year. Readers may be concerned that state legislatures banned pharmaceutical promotion as a reaction to increased opioid misuse. Figure 1.3a plots the differences in overdose rates for Minnesota and the rest of the US from 1987-2007. Minnesota was the first state to introduce a state ban on pharmaceutical promotion in 1997. The graph shows that overdose rates of counties in Minnesota are statistically indistinguishable from other counties in the years leading to the introduction of the state ban. Overdose rates started to decrease in Minnesota compared to the rest of the US one year after the introduction and five years later the gap becomes statistically significant at the 5% level. Figure 1.3a shows the differences in opioid overdose rates of Vermont and Massachusetts compared to the rest of the US, excluding Minnesota from 1999 to 2015. Before the introduction of the state ban in 2009, their overdose death rates are statistically indistinguishable from the rest of the US. After the introduction, death rates do not decline in these two states. It is important to note that Massachusetts and Vermont are small states with 14 counties. Additionally, death rates of opioid overdoses vary substantially from county to county in the late 2000s. Furthermore, Massachusetts amended the law in 2012. Initially, sales representatives were not allowed to provide any meals of any value to health care professionals outside their office. In 2012 this law was updated such that they are not able to provide meals of "modest value". It is, therefore, no surprise to not see any significant decline in the years following the ban for counties belonging to these two states.[17] It is important to note that the ban holds for all types of drugs, not only opioid medication and there is no anecdotal evidence that these bans were introduced as reactions to the opioid epidemic but rather to curtail financial conflicts of interest in general.

Our identification relies on the assumption that the distance to headquarters operating in 2014 and 2015 and promoting opioid drugs to physicians and teach-

[17] In our empirical analysis, we include a dummy for states that have any kind of ban in place in 2014 and 2015. We do not have a measure to which degree the laws prohibit promotion to physicians. As Massachusetts diluted the law in 2012, we perform a robustness check in which only Minnesota and Vermont are coded as states with bans. The partial F-Value of the first stage increases and our second stage coefficients of promotion on overdose death rates are larger. Results are available upon request.

ing hospitals is exogenous to our outcome variable, opioid overdose rates in the respective years. For this we limit our set of pharmaceutical companies to the ones whose headquarters location in 2014/2015 was already determined before 1995.[18] All companies that started operations after 1995 or moved their headquarters after 1995 are dropped from our sample in the main analysis. We also show that opioid overdose rates before 1996 are independent from the distance to headquarters in 2015 in Figure 1.4. The location of the headquarters of the pharmaceutical companies, most of which also produce drugs besides opioid medications, is not significantly related to overdose rates before the large-scale onset of pharmaceutical promotion of opioid medication. Many of the headquarters are located on the East Coast. The reader may be concerned that our results are driven by outliers in terms of opioid death rates, which happen to be located close to the East Coast. West Virginia, Ohio, and Kentucky have been hit particularly hard by the opioid epidemic and are located close to headquarters. Our results are not reliant on the inclusion of these three states. Excluding these states one by one decreases our coefficient estimate from 0.31 to 0.25, but we do still find a positive and statistically significant at conventional levels relationship, confirming that our results are robust to outliers. Our estimates are mainly driven by counties located in the South and Midwest. We cannot capture the relationship of promotion and death for the West Coast, as distance to headquarters in kilometers is not relevant for these counties.[19]

Our instrumental variable model depicted in Equations (1.3) and (1.4) is overidentified. This allows us to look at the regression results using the instruments separately and to test whether the instrument exogeneity condition is valid for one of the two instruments. Table A-8 shows the estimation results of Equations (1.3) and (1.4) regression results if we are using the instruments separately, splitting the sample into different maximum distances to the closest headquarters. All counties are included in the regressions displayed in columns (1) - (3) while only counties within 500 km distance to an opioid producing headquarters are considered in columns (4) - (6). We expect that the distance instrument is valid for counties

[18] Before 1995, there is no evidence of pharmaceutical companies promotion opioid to physicians as treatment options for long-term non-malignant pain patients at large scales.

[19] Results are available upon request.

and physicians within a reasonable distance to the headquarters. Engelberg et al. (2014) follow a very similar empirical strategy and include in their analysis only the prescription behavior of physicians within a 500 km radius of the promoting firm's headquarters. The idea is that these physicians can be reached within a day from the headquarters.[20] As can be seen from comparing the first stage partial F-value in columns (2) and (5), we also find that the distance instrument has a higher first stage if we only consider counties within 500 km distance. In addition, we observe that the second stage coefficients on promotion are very similar using the instruments separately for these counties compared to the regression results including all counties. To analyze whether the instrument exogeneity condition is valid for one of the two instruments, we perform the Sargan overidentification test. The Sargan test examines whether any of the instruments are invalid, assuming that at least enough instruments are valid to exactly identify the equation. If we consider all counties in our overidentified IV regression model, we reject the null hypothesis that both instruments are valid (see p-value of Sargan test in the last row). This implies that the instruments are either correlated with the error term or that they are omitted variables in the regression model. If we only consider counties within a reasonable distance to the headquarters (less than 500 km), we fail to reject the null that all instruments are invalid with a p-value of 0.252. The regression model appears to be misspecified when we include counties for which the distance to the headquarters is irrelevant. Simultaneously, failing to reject the null hypothesis of the Sargan test for counties for which we expect instrument relevance increases the credibility that our instruments are valid.

To show that our results are not driven by small areas where opioid overdose rates are very sensitive to small changes, we repeat our main analysis splitting our sample into two subsamples of counties with more and less than 100,000 inhabitants. Table 1.12 shows that coefficient estimates are identical for small and large counties. This also shows that the relationship we uncover for opioid promotion and overdoses is not exclusive to urban areas.

Although we have shown that overdose death rates of 1995 are unrelated to the location of pharmaceutical company headquarters one may still be concerned that promotion is particularly high in counties that have a high demand for opioid

[20] Excluding the possibility of air travel, for which physical distances are less relevant.

drugs and that the location of the headquarters is related to previous levels of overdose rates. We, therefore, repeat our analysis of Equation (1.3) and (1.4) but additionally control for overdose death rates in the previous years. As seen in Table A-9 overdose mortality rates are autocorrelated. We still find a positive and statistically significant relationship between opioid promotion and overdose death rates in the corresponding year. Our coefficient estimates are smaller once we control for previous death rates. Increasing promotion by 1% led to an increase in opioid death rates by 0.16%, The partial F-Value depicted in the last row of Table A-9 implies that our instruments predict contemporaneous levels of promotion well, even when we control for previous overdose death rates.

Additionally, we show that it is not pharmaceutical promotion per se that is driving opioid overdose rates, but specifically promotion regarding opioid drugs. This helps us to rule out the concern that the counties with high levels of opioid promotion are just counties with high morbidity and high demand for all kinds of drugs. In the last column of Table A-9, we control for pharmaceutical promotion spending of all drugs that are not opioid painkillers. Our coefficient estimates on opioid promotion do not change substantially. As we can see from the reduced partial F-Value in the last row, controlling for promotion of other drugs reduces the predictive power of our two instruments. This can be explained by the fact that the pharmaceutical companies that promote opioid drugs also promote other medication and devices. These estimates nevertheless speak against the interpretation that promotional efforts for all drugs are high due to higher morbidity and thus higher mortality.

Readers may still be concerned that counties with higher morbidity are the ones receiving more pharmaceutical promotion in general. To convince the readers that we are not only picking up the relationship of higher morbidity in general, we perform a placebo test. We show that death rates regarding diseases, that should be unrelated to opioid use and pain in general, are unrelated to opioid promotion. We run the same IV regression as depicted in Equation 1.3 and 1.4 but our dependent variable is now the rate of people that died from diabetes mellitus or a stroke in the corresponding county (ICD Codes: E10-E14 and I60-I69). We pick death related to diabetes mellitus or strokes as our placebo outcomes, as they are among the ten leading causes of death in the US and deaths for which we ex-

pect no systematic relation with opioid misuse and overdose deaths.[21] Table A-10 shows that the number of people dying from diabetes or strokes is not related to opioid promotion. This speaks against the interpretation that opioid death rates and promotion are high in counties with high levels of morbidity in general.

To be able to derive policy implications, it is important to understand whether the promotion of opioid drugs leads to an increase in illicit drug overdoses or prescription opioids. We cannot distinguish whether the death in the mortality database occurred because the deceased followed the prescription of the physician or because he or she obtained the opioid drug through drug diversion or doctor shopping. However, we can distinguish whether an overdose occurred due to the consumption of an illicit (heroin) or legal opioid drug. Overdose death due to heroin intake is classified as T40.1 in the CDC multiple cause of death mortality database. Table 1.13 displays the regression results of our two main regression, comparing the effect on all opioid overdose deaths with the effect on heroin overdoses. The coefficient from the 2SLS regression suggests that opioid promotion has a comparable impact on heroin overdoses as on prescription opioid overdoses. It is claimed that many patients who were prescribed opioid medications and became addicted, substituted to the use of illicit opioid drugs such as heroin. According to the National Survey on Drug Use and Health (NSDUH), between 2002 and 2011 80% of recent heroin initiates report prior use of opioid pain relievers (Muhuri et al., 2013).

Throughout the empirical analysis at the county level, we measure promotion with the number of physicians receiving promotion related to opioid drugs. In the Appendix, we show that if we use the total dollar amount spent on opioid drug promotion instead, we still find a positive and statistically significant relationship with opioid-related overdose rates (Table A-11). As in the OLS regressions, our

[21]The other leading causes of death are heart diseases, cancer, chronic lower respiratory diseases, accidents (unintentional injuries), Alzheimers disease, influenza and pneumonia, nephritis, nephrotic syndrome, and nephrosis and intentional self-harm. None of these deaths would serve as good placebo tests. Research shows that opioid use could have adverse effects on the gastrointestinal, respiratory, cardiovascular, central nervous, musculoskeletal and endocrine system (Baldini et al., 2012). Additionally, the rates of suicides or accidents involving other drugs could be directly affected by the number of opioids prescribed in the county. First, suicide attempts can include opioid drugs or heroin. Second, drug overdoses of other drugs could involve opioids without classification of opioid drugs in the death certificate (Ruhm, 2017). Third, opioid use could lead to addiction and substitution to other drugs.

estimates are half the size compared to the regressions in which promotion is measured by the number of doctors receiving any kind of promotion.

In the last robustness check, we investigate whether our results of a positive causal relationship between opioid promotion and overdose deaths hold if we use an alternative instrument. We instrument the number of physicians who receive opioid promotion with the number of physicians in the same county who receive pharmaceutical promotion for drugs, that are unrelated to pain or opioid medication (such as blood thinner or diabetes medication). The idea is that physicians get opioid promotion solely because sales representatives are also promoting unrelated drugs. This should affect opioid overdose deaths only through opioid promotion. We restrict our set of drugs to the 20 most promoted drugs in the corresponding years that are unrelated to opioids. An overview of the drugs, their purpose and the manufacturer can be found in Table A-12 in the Appendix. Table A-13 displays the results of the first and second stage regressions, based on Equations (1.3) and (1.4). Panel A indicates that the more physicians in a county receive promotion for unrelated drugs, the more physicians receive it for opioid medication as well. Both regressions control for county characteristics and the partial F-Value indicates that our instruments predict our independent variable well. The second stage results show a positive relationship between opioid promotion and opioid overdoses. Again the IV estimates are larger than the OLS estimates in Table 1.4. The coefficient estimates of this alternative instrumental variable approach are close to the estimates of the baseline instrumental variable approach (see Table 1.6). Lastly, to affirm our results of opioid promotion leading to higher prescription rates by Medicare physicians and worsened neonatal health outcomes, we repeat the instrumental variable regressions using this alternative instrument. Results for neonatal health outcomes are depicted in Table A-14. Coefficient estimates are similar for most of the symptoms analyzed. We also confirm the positive and statistically and economically significant relationship between promotion and prescription rates with the alternative instrument. Panel B in Table A-15 shows that Medicare Physicians who receive promotion for medication unrelated to pain and opioids are more likely to receive opioid promotion. More opioid promotion ultimately leads to higher opioid prescription rates, as depicted in Panel A. The coefficient estimates are twice as large as in the OLS specification, mirroring the

results of the analysis on opioid-related overdose death rates.

1.6 Conclusion

The opioid epidemic continues to be one of the most pressing public health concerns in the US. The public costs of the epidemic are staggering: in 2015, 33.000 people died of opioid overdoses. Hospitalization rates for opioid abuse increase steadily (1000 per day in the US in 2015). More and more babies are born with neonatal withdrawal symptoms, following the mothers' usage of opioid during pregnancy.

It is important to understand the causes of the epidemic to create optimal policies fighting the current epidemic and preventing future outbreaks. We show that pharmaceutical promotion is positively related to opioid prescription rates of doctors and ultimately causes the number of overdose deaths to increase. The most conservative estimate from the fixed effect regression suggests that increasing pharmaceutical promotion by 1% from 2014 to 2015 increases death rates by 0.04%. This implies that the promotion of opioid drugs can explain 3% of the variation in death rates. As an interesting case study, we also show that opioid overdose rates are significantly lower in Minnesota, after the introduction of the state ban on pharmaceutical promotion in 1997. Opioid overdose rates before 1995 are unrelated to the closeness of the counties to the headquarters of the pharmaceutical company and states that introduced a ban on promotional activities do not show differential overdose rates before the introduction, supporting the exogeneity assumption of our instruments.

In addition, we find that babies that are born in counties with high levels of pharmaceutical promotion of opioid-related drugs are more likely to be born with health outcomes in line with the neonatal abstinence syndrome: the neonates have lower birth weights, are more likely to be born prematurely and to need assisted ventilation. This negative effect seems to be particularly pronounced for promotion in the third trimester of the pregnancy, consistent with medical research showing that especially late in-utero exposure to opioids has detrimental health impacts for the babies.

We show that prescription rates are higher for Medicare physicians who re-

ceive pharmaceutical promotion for opioid analgesics, and our placebo test indicates that specifically receiving information and financial incentives for opioid analgesics is driving the increase in claim rates, not receiving any kind of promotion per se.

Physician opioid painkiller prescription behavior varies substantially, especially among general practitioners. The more opioid drugs are prescribed, the more people die of opioid-related overdoses (Currie and Schnell, 2018). Currie and Schnell (2018) find that parts of these variations can be explained by the quality of education physicians received in medical school. They argue that they cannot pin down precise differences in the curricula that ultimately lead to diverging prescription rates. One difference between the top and last ranking schools listed in their analysis is the score obtained by the American Medical Student Association on the conflict of interest policies at the medical schools (AMSA, 2016). Top ranking schools have good grades in the AMSA scorecard while low ranking schools show lower grades. Clearly, the presence of conflict of interest policies may correlate with other differences in the curricula of the schools. An interesting question for future research would be to investigate which medical school policies and curricula are the most effective in determining prescription behavior of the physicians. We find that physicians affiliated to hospitals with strict limits on interactions between sales representatives and health care professionals are less sensitive towards opioid promotion than physicians affiliated to teaching hospitals without such bans. In our analysis, unfortunately, we cannot rule out endogenous sorting of physicians nor patients into hospitals with stricter laws on the interaction between health care professionals and the pharmaceutical industry.

One of the causes of the epidemic is the room for misinformation of the pharmaceutical companies in promoting directly to physicians and teaching hospitals. One solution to prevent further misbranding is to increase the FDA's ability to review and verify promotional material before its distribution.[22] In overseeing the

[22] According to the Code of Federal Regulations Title 21 (Food and Administration (2015) and implementing regulations) manufacturers should submit their advertisement material to the FDA before distributing it. The FDA then reviews the material and verifies its accuracy. The FDA has a limited number of staff responsible for the review of all the promotional material. Some opioid-promoting manufacturers distributed promotional material before it was verified by the FDA (Van Zee, 2009).

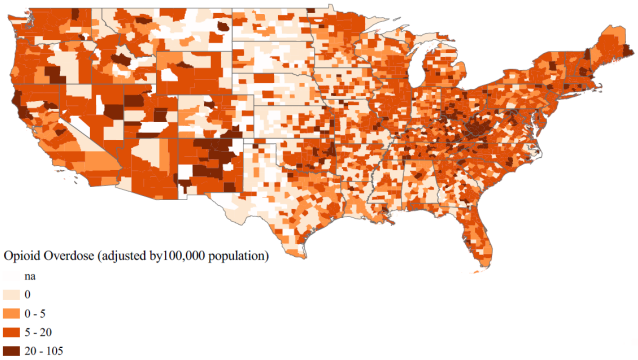
promotional material of prescription drugs, there is no distinction for the FDA between controlled substance and other prescription drugs (GAO, 2003). All controlled substances have per definition potential for abuse and are dangerous when used incorrectly. Pharmaceutical companies are not allowed to run reminder advertisements in television or other forms of broadcast for controlled substance drugs (FDA Code of Federal Regulations 21CFR202.1). Extra caution should also be applied in verifying and controlling information that is distributed to physicians, in particular if it is mostly targeted at primary care physicians who may not have been adequately trained in pain management.

It is beyond the scope of this paper to make welfare statements about the benefits and harms of pharmaceutical promotion of controlled drugs to physicians in the US. Some physicians argue that they perceive promotion as beneficial, as it facilitates the learning about new medications. It is not clear how much physicians incorporate in their decision the fact that this information does not necessarily need to be accurate. To curtail the further spread of the opioid epidemic and to prevent future prescription mistakes we propose that promotional material must be verified by the FDA before manufacturers are allowed to distribute it and that failures to do so must be prosecuted.[23]

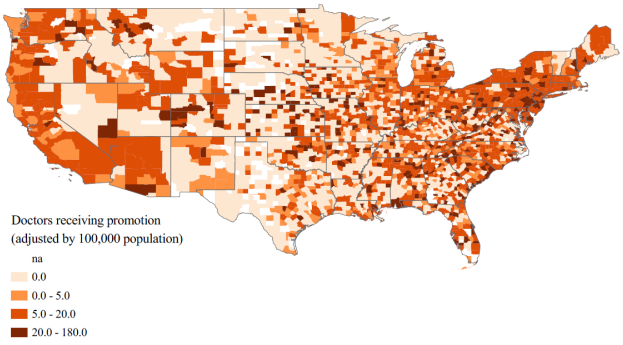
[23] A similar, albeit less demanding, recommendation has been put forth by the Committee on Pain Management and Regulatory Strategies to Address Prescription Opioid Abuse, Board on Health Sciences Policy Health and Medicine Division: "Recommendation 6-5. Strengthen the post-approval oversight of opioids. The U.S. Food and Drug Administration should take steps to improve post-approval monitoring of opioids and ensure the drugs favorable benefit-risk ratio on an ongoing basis. Steps to this end should include [...] aggressive regulation of advertising and promotion to curtail their harmful public health effects." (Sciences et al., 2017).

Tables and Figures

Figure 1.1: Number of opioid-related overdose death rates & opioid promotion in 2014

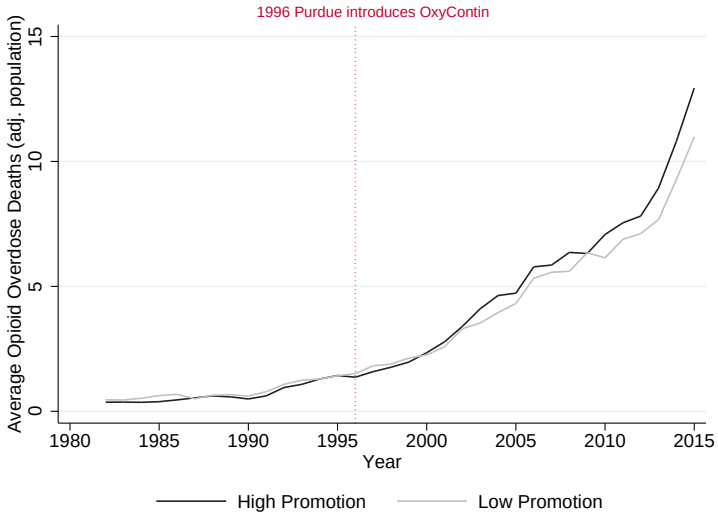


(a) Opioid-related overdoses in 2014. Source: CDC Wonder Mortality MCD Data



(b) Doctors receiving opioid promotion in 2014. Source: CMS Open Payments Data 2014

Figure 1.2: Diverging overdose rates



Average death rates (adj. 100.000 population) for high and low (below median) opioid promotion counties. Data available for 403 counties before 1999, counties with more than 100,000 inhabitants. Source: CMS Open Payments Data and CDC Wonder Mortality MCD Data

Table 1.1: Summary statistics US counties pharmaceutical promotion & opioid-related death rates 2014-2015

	Observations	Mean	Median	Std. Dev	Min	Max
2014						
County Aggregates						
Doctor receiving Opioid Promotion	3142	11.25	1.00	33.96	0	639
Doctor receiving Other Painkiller Promotion	3142	18.84	1.00	61.99	0	1539
Share of Payments with no Drug ID	2958	0.30	0.28	0.19	0.00	1.00
Total Payments for Opioids in $	3142	1161	7.15	19673	0	1067246
Total Payments for Opioids in $ (> 0)	1708	2137	79.62	26648	1.50	1067246
Total Payments for Painkillers in $	3142	2390	16.78	29996	0	1523839
Total Payments for Painkillers in $ (> 0)	1815	4137	156.11	39380	1.18	1523839
Visits to Physicians						
Av. visits by Opioid Sales Rep	1577	2.19	1.67	1.79	1.00	29.34
Av. visits by any Sales Rep	2958	6.56	5.35	4.76	1.00	28.02
Av. number of Manufacturers visiting for opioids	1483	1.00	1.00	0.00	1.00	1.00
Av. number of Manufacturers visiting for any drug	2957	1.25	1.24	0.19	1.00	4.00
Opioid-Related Overdose Death Rates (ICD-10 Code: T40.0-T40.4)						
Total Deaths	2929	9.55	2.00	26.87	0	449
Adjusted by Population (by 100,000)	2929	7.87	5.93	9.19	0	101
2015						
County Aggregates						
Doctor receiving Opioid Promotion	3142	9.88	0.00	33.10	0	729
Doctor receiving Other Painkiller Promotion	3142	21.79	2.00	70.63	0	1681
Share of Payments with no Drug ID	2905	0.29	0.26	0.20	0.00	1.00
Total Payments for Opioids in $	3142	2517	0.00	18510	0.00	439332
Total Payments for Opioids in $ (> 0)	1510	5238	80.41	26436	0.17	439332
Total Payments for Painkillers in $	3142	1952	20.68	12549	0.00	364560
Total Payments for Painkillers in $ (> 0)	1837	3339	160.83	16272	0.16	364560
Visits to Physicians						
Av. visits by Opioid Sales Rep	1511	1.00	1.00	0.10	1.00	5.00
Av. visits by any Sales Rep	2905	7.11	5.65	5.38	1.00	30.43
Av. number of Manufacturers visiting for opioids	1185	1.00	1.00	0.02	1.00	1.50
Av. number of Manufacturers visiting for any drug	2905	1.24	1.23	0.18	1.00	3.00
Opioid-Related Overdose Death Rates (ICD-10 Code: T40.0-T40.4)						
Total Deaths	2915	11.13	2.00	31.96	0	517
Adjusted by Population (by 100,000)	2915	9.00	6.39	10.4	0	131

Source: CMS Open Payment Data 2014 and 2015, CDC Wonder Multiple Cause of Death Data.

Table 1.2: Summary statistics neonatal health

	Observations	Mean	Median	Std. Dev	Min	Max
Health Outcomes						
Admission NICU	3845148	0.08	0	0.27	0	1
Assis. Ventilation Immedi.	3845148	0.04	0	0.18	0	1
Assis. Ventilation $>$ 6 hrs	3845148	0.01	0	0.11	0	1
APGAR 5	3981330	8.78	9	0.84	0	10
Birth Weight	3994708	3272.89	3317	591.69	228	8165
Low Birth Weight ($<$2500g)	3994708	0.08	0	0.27	0	1
Born Prematurely ($<$ 37 weeks)	3994872	0.11	0	0.32	0	1
Mother's Demographics						
Age	3998175	28.35	28	5.89	12	50
Born US (D=1)	3988351	0.78	1	0.41	0	1
White (D=1)	3866633	0.75	1	0.43	0	1
Educ. Attainment	3855275	4.29	4	1.80	1	9
Married	3998175	0.60	1	0.49	0	1
Smoker	3779767	0.08	0	0.28	0	1
Birth Order	3939398	2.48	2	1.57	1	8
Number of Babies born	3998175	1.04	1	0.19	1	5
Gest. Diabetes	3848302	0.05	0	0.23	0	1
Gest. Hypertension	3848302	0.05	0	0.22	0	1
Medicaid Recipient	3819768	0.44	0	0.50	0	1
Mother's BMI	3709225	26.54	25	6.55	13	68.90
Birth Characteristics						
Baby (Boy=1)	3998175	0.51	1	0.50	0	1
Vaginal Delivery	3852663	0.68	1	0.47	0	1
Prenatal Care Start 1st Trim.	3707352	0.77	1	0.42	0	1
Physician attended Delivery	3996146	0.90	1	0.30	0	1
Opioid Promotion: Number of Physicians						
During Pregnancy	3943598	15.89	11.89	14.33	0	235.45
1st Trimester	3952324	3.74	2.65	4.35	0	99.40
2nd Trimester	3943598	5.69	4.18	5.51	0	111.03
3rd Trimester	3943598	6.46	4.82	6.06	0	111.03
Min. Distance HQ in 1000 km	3943598	0.95	0.61	0.90	0	6.46
Presence State Ban (D=1)	3998175	0.04	0	0.19	0	1
Promotion Other Drugs[a]: Number of Physicians						
During Pregnancy	3943598	390.66	326.78	296.67	0	5011.16
1st Trimester	3952324	93.47	73.70	94.04	0	1736.54
2nd Trimester	3943598	136.42	112.79	104.34	0	1885.39
3rd Trimester	3943598	160.56	139.05	113.73	0	1885.39

[a] see Table A-12 for list of drugs

Source: CMS Open Payments Data 2013 and 2014, CDC 2014 Natality Detail Data Set.

Table 1.3: Summary statistics Medicare prescribers 2014

	N	Mean	Std. Dev	Min	Max
Drug Claims 2013 & 2014					
Opioid Claims 2014	753975	106	310	0	26449
Opioid Claims 2014 (if >0)	503757	159	368	11	26449
Opioid Claims 2013	970367	73	262	0	21519
Opioid Claims 2013 (if >0)	414174	173	379	11	21519
Total Drug Claims 2014	1072851	1318	3171	11	226081
Total Drug Claims 2013	970367	1405	3255	11	191530
Share Opioid overall Drug Claims 2014	1072851	0.09	0.16	0.00	1.00
Payments Received					
Payments received for Opioids 2014	1072851	2.57	210.25	0.00	70488
Payments received for Opioids 2014 (if >0)	27729	99	1304	0.21	70488
Payments received for Non-Painkiller 2014	1072851	1130	51439	0.00	43859980
Payments received for Non-Painkiller 2014 (if >0)	430134	2819	81209	0.01	43859980
Payments received for Other Painkillers 2014	1072851	3.62	189	0.00	70249
Payments received for Other Painkillers 2014 (if >0)	33867	115	1059	0.21	70249
Payments received for Drugs Unrelated to Pain 2014[a]	1072851	76.13	1867	0.00	304084
Payments received for Drugs Unrelated to Pain 2014[a] (if >0)	153437	532.29	4914	0.16	304084
Closest HQ Distance & State Ban					
Min. Distance HQ in 1000 km	1072851	0.86	0.88	0	12.5
Presence State Ban (D=1)	1072851	0.05	0.21	0	1
Physician Specialty					
Internal Medicine	1072851	0.12	0.33	0	1
Nurse	1072851	0.10	0.30	0	1
Dentist	1072851	0.12	0.33	0	1
Emergency Medicine	1072851	0.04	0.20	0	1
Pain Management	1072851	0.00	0.06	0	1
Family Medicine	1072851	0.10	0.30	0	1
Others	1072851	0.51	0.50	0	1
Physician Characteristics					
Affiliated to Hospital with Ban on Sales Reps	67675	0.91	0.29	0	1
Physician Male	711125	0.60	0.49	0	1
Graduation Year	673922	1994	12.57	1943	2017

[a] see Table A-12 for list of drugs

Source: CMS Medicare Opioid Prescriber Summary File for Number of Opioid Claims and other Claims 2013 and 2014. Additional physician characteristics from Medicare Compare and AMSA Scorecard.

Table 1.4: OLS: opioid overdose deaths and opioid promotion

Dependent Variable:	(1)	(2)	(3)	(4)	(5)	(6)
log Opioid Overdose Deaths	2014	2015	2015	2014	2015	2015
log Receiving Doctors 2014	0.0921***		0.00151			
	(0.0188)		(0.0221)			
log Receiving Doctors 2015		0.111***	0.110***			
		(0.0185)	(0.0225)			
log USD 2014				0.0554***		0.00892
				(0.00917)		(0.0108)
log USD 2015					0.0575***	0.0529***
					(0.00858)	(0.0104)
Mean Dep. Var.	1.615	1.714	1.714	1.615	1.714	1.714
SD Dep. Var.	1.175	1.204	1.204	1.175	1.204	1.204
Observations	2918	2905	2905	2918	2905	2905
R2	0.322	0.347	0.347	0.326	0.348	0.348
State F.E.	Y	Y	Y	Y	Y	Y
County Characteristics	Y	Y	Y	Y	Y	Y

Estimation result of Equation (1.1). Opioid overdoses and opioid promotion (number of doctors that receive promotion and dollar amount) normalized by county population. State fixed effects included in all regressions. County characteristics included in the regression: unemployment rate, log median income, poverty rate, population, industry shares, share of population enrolled in Medicare Prescription Drug Plan, dummy urban/rural. Standard errors in parentheses adjusted for heteroscedasticity, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: CDC Wonder Mortality Data and CMS Open Payments Data 2014, 2015.

Table 1.5: Fixed effect regression: opioid overdose deaths and opioid promotion

Dependent Variable: log Opioid Overdose Deaths	(1)	(2)
log Receiving Doctors	0.0346*	
	(0.0205)	
log USD		0.0168
		(0.0106)
Mean Dep. Var.	1.689	1.689
SD Dep. Var.	1.181	1.181
Observations	5658	5658
R2	0.0227	0.0227
Year F.E.	Y	Y
County F.E.	Y	Y
Time Varying County Characteristics	Y	Y

Estimation result of Equation (1.2). Opioid overdoses and opioid promotion (number of doctors and dollar amount) normalized by county population. For list of time-varying county characteristics see footnote of Table 1.4. Standard errors in parentheses clustered at state level, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: CDC Wonder Mortality Data and CMS Open Payments Data 2014, 2015.

Table 1.6: 2SLS: opioid overdoses and opioid promotion

	Panel A: First Stage		
Dep. Var.: log Receiving Doctors	(1)	(2)	(3)
Dist. calculated to:	All Headquarters	Opened before 1995	
State Ban (D=1)	-0.913***	-0.803***	-0.963***
	(0.0634)	(0.0628)	(0.0608)
Distance closest HQ in km	-0.596***	-0.226***	-0.172***
	(0.0431)	(0.0243)	(0.0232)
Mean Dep. Var.	1.193	1.193	1.197
SD Dep. Var.	1.278	1.278	1.278
Observations	6284	6284	6266
R2	0.0517	0.0292	0.284
Partial F-Value	131.4	93.80	123.0
County Controls	N	N	Y
Year F. E.	Y	Y	Y

	Panel B: Second Stage		
Dep. Var.: log Opioid Overdose Deaths	(1)	(2)	(3)
Instruments: State ban and Distance to	All Headquarters	Opened before 1995	
log Receiving Doctors	0.687***	0.337***	0.317***
	(0.0652)	(0.0825)	(0.0782)
Mean Dep. Var.	1.664	1.664	1.664
SD Dep. Var.	1.191	1.191	1.190
Observations	5844	5844	5840
County Controls	N	N	Y
Year F. E.	Y	Y	Y

Estimation results of Equations (1.3) and (1.4). Partial F-value of first stage Equation (1.3) displayed in last row in Panel A. Opioid overdoses and the number of doctors receiving opioid promotion both normalized by county population. Control county characteristics: unemployment rate, log median income, poverty rate, population, industry shares, share of the population that is enrolled in the Medicare Prescription Drug Plan, dummy urban/rural. Standard errors in parentheses adjusted for heteroscedasticity, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$).

Table 1.7: OLS: neonatal health and opioid promotion

	(1) Admission NICU	(2) Ventilation Immediately	(3) Ventilation > 6hr	(4) APGAR 5	(5) Birth Weight	(6) Low BW < 2500g	(7) Premature Born
			Panel A: Promotion During Pregnancy				
Promotion 9 Months	0.00516***	0.000689	0.000855***	-0.0112***	-4.711***	0.00277***	0.00236***
before Delivery	(0.000905)	(0.000604)	(0.000281)	(0.00399)	(1.133)	(0.000463)	(0.000562)
R2	0.0710	0.0249	0.0189	0.0291	0.163	0.143	0.102
MHT adj. P-Value	0.00	1.00	0.02	0.04	0.00	0.00	0.00
			Panel B: Promotion By Trimester				
1st Trimester	0.00402**	-0.0000152	0.000210	-0.00793	-5.019***	0.00304***	0.00143
	(0.00161)	(0.00103)	(0.000516)	(0.00762)	(1.946)	(0.000848)	(0.00113)
2nd Trimester	0.00455***	-0.000911	0.000109	-0.00946*	-3.410**	0.00221***	0.00220**
	(0.00118)	(0.000854)	(0.000457)	(0.00546)	(1.629)	(0.000731)	(0.000990)
3rd Trimester	0.00641***	0.00258**	0.00193***	-0.0147**	-5.698***	0.00311***	0.00309***
	(0.00158)	(0.00105)	(0.000450)	(0.00678)	(1.929)	(0.000882)	(0.000898)
Mean Dep. Var.	0.0808	0.0351	0.0112	8.785	3280.2	0.0777	0.110
SD Dep. Var.	0.273	0.184	0.105	0.825	584.9	0.268	0.313
Observations	3436124	3436124	3436124	3429416	3439713	3439713	3440894
R2	0.0710	0.0249	0.0190	0.0291	0.163	0.143	0.102
MHT adj. P-Value	0.00	0.09	0.00	0.21	0.02	0.00	0.00
Mother's Demographics	Y	Y	Y	Y	Y	Y	Y
Birth Characteristics	Y	Y	Y	Y	Y	Y	Y
Month of Birth F.E.	Y	Y	Y	Y	Y	Y	Y
State F.E.	Y	Y	Y	Y	Y	Y	Y

Estimation result of Equation (1.1). Opioid promotion measured as number of doctors receiving opioid promotion in the county of birth during pregnancy (normalized by county population). Mother's characteristics controlled for in all regressions are age, race, educational attainment, marital status, insurance status, mother's health (BMI, hypertension, diabetes), whether mother was born in the US and whether the mother is a smoker. Characteristics of births included in all regressions: vaginal delivery, sex of the baby, birth order, number of babies, early prenatal visits, attendant at birth is physician. State fixed effects included in all regressions. Standard errors in parentheses clustered at county level, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). P-Values adjusted for multiple hypothesis testing (Bonferroni adjustment) displayed for promotion during the entire pregnancy in Panel A and for promotion in the third trimester in Panel B. Source: CDC 2014 Natality Detail Data Set and CMS Open Payments Data 2014, 2015.

Table 1.8: 2SLS: neonatal health and opioid promotion

	Panel A: Second Stage Results						
	(1) Admission NICU	(2) Ventilation Immediately	(3) Ventilation $>$ 6hr	(4) APGAR 5	(5) Birth Weight	(6) Low BW $<$ 2500g	(7) Premature Born
Promotion 9 Months	0.00233	0.00183	0.00340***	-0.0103	-10.79*	0.00497***	0.0124***
before Delivery	(0.00447)	(0.00363)	(0.00129)	(0.0205)	(5.849)	(0.00179)	(0.00291)
Mean Dep. Var.	0.0808	0.0351	0.0112	8.785	3280.2	0.0777	0.110
SD Dep. Var.	0.273	0.184	0.105	0.825	584.9	0.268	0.313
Observations	3436124	3436124	3436124	3429416	3439713	3439713	3440894
Mother's Demographics	Y	Y	Y	Y	Y	Y	Y
Birth Characteristics	Y	Y	Y	Y	Y	Y	Y
Month of Birth F.E.	Y	Y	Y	Y	Y	Y	Y
	Panel B: First Stage Results						
β Dist. HQ 2014	-0.385***	-0.385***	-0.385***	-0.385***	-0.385***	-0.385***	-0.385***
	(0.0560)	(0.0560)	(0.0560)	(0.0560)	(0.0560)	(0.0560)	(0.0560)
β State Ban	-1.360***	-1.360***	-1.360***	-1.360***	-1.360***	-1.360***	-1.360***
	(0.152)	(0.152)	(0.152)	(0.152)	(0.152)	(0.152)	(0.152)
Partial F-Value	46.64	46.64	46.64	46.67	46.69	46.69	46.68

Estimation result of Equations (1.3) and (1.4). Opioid promotion measured as number of doctors receiving opioid promotion in the county of birth during pregnancy (normalized by county population). Partial F-value of first stage Equation (1.3) displayed in last row. Coefficient estimates and standard errors of first stage regression displayed in Panel B. Distance to closest HQ in 2014 measured in 1000km. HQ considered here are reduced set of HQ described in Section 1.1. Mother's characteristics controlled for in all regressions are age, race, educational attainment, marital status, mother medicaid recipient, mother's health (BMI, hypertension, diabetes), whether mother was born in the US and whether the mother is a smoker. Characteristics of births included in all regressions: vaginal delivery, sex of the baby, birth order, early prenatal visits, attendant at birth is physician. Standard errors in parentheses clustered at county level, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: CDC 2014 Natality Detail Data Set and CMS Open Payments Data 2014, 2015.

Table 1.9: Opioid prescriptions and opioid promotion: OLS & 2SLS

Method:	OLS		2SLS	
Dependent Variable:	(1)	(2)	(3)	(4)
Opioid Prescriptions	# Pres. 2014	# Pres. 2014	# Pres. 2014	# Pres. 2014
Opioid Promotion (Dummy)	45.54***		42.07**	
	(2.602)		(20.50)	
Opioid Promotion (log USD)		15.55***		13.69**
		(0.895)		(6.644)
# Opioid Pres. 2013	0.976***	0.974***	0.978***	0.976***
	(0.00703)	(0.00707)	(0.00765)	(0.00818)
Mean Dep. Var.	114.9	114.9 .	114.9	114.9
SD Dep. Var.	322.3	322.3	322.9	322.9
Observations	633306	633306	686275	686275
R2	0.888	0.889	0.888	0.888
County F.E.	Y	Y	N	N
Physician Specialty	Y	Y	Y	Y
First Stage Results				
β Dist. HQ 2014			-0.00318***	-0.00970***
			(0.000288)	(0.000981)
β State Ban			-0.0155***	-0.0477***
			(0.000878)	(0.00310)
Partial F-Value			187.6	145.6

Number of opioid claims of Medicare Physicians and opioid-related promotion OLS and 2SLS estimates. 2SLS estimation results of Equations (1.5) and (1.6). First stage results depicted at the end of the Table. Promotion is instrumented with the distance of the physicians office to the closest headquarters (reduced set of headquarters) and the presence of a state ban on promotion. Promotional level measured as dummy for any promotion in column (1) and (3) and as log dollar amount in column (2) and (4), respectively. All regressions control for the specialty of the physician and opioid prescription in the previous year. OLS estimates additionally include county fixed effects. Standard errors in parentheses clustered at zip-code, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: Medicare Opioid Prescriber Summary File and CMS Open Payments Data 2014.

Table 1.10: Placebo: opioid prescriptions and non-opioid promotion

Method:	OLS
Dep. Var.:	(1)
Opioid Prescriptions	# Pres. 2014
Non-Opioid Non-Painkiller Promotion	0.0206
	(0.0671)
Non-Opioid Painkiller Promotion	4.349***
	(0.497)
Opioid Promotion	14.09***
	(0.824)
# Opioid Pres. 2013	0.972***
	(0.00722)
County FE	Y
Physician Specialty	Y
Mean Dep. Var.	114.9
SD Dep. Var.	322.3
Observations	633306
R2	0.889

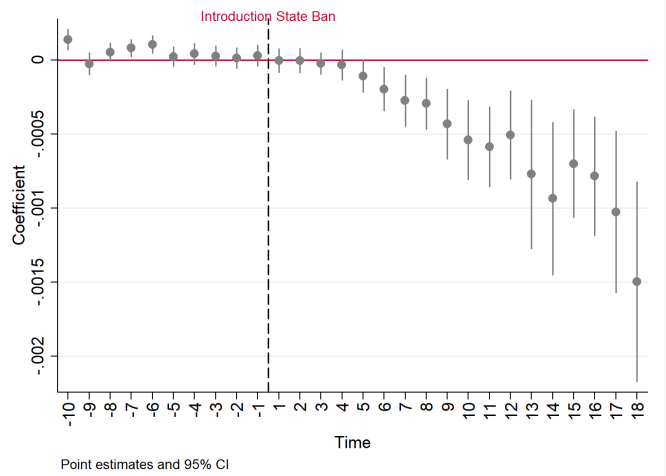
Number of opioid claims of Medicare Physicians and non-opioid and non-painkiller promotion. Promotion measured as log dollar amount received in corresponding year. Estimation result of Equation (1.5). All regressions control for specialty of physician, county fixed effects and opioid prescription in the previous year. Standard errors in parentheses clustered at zipcode level, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: Medicare Opioid Prescriber Summary File and CMS Open Payments Data 2014.

Table 1.11: Opioid prescriptions and opioid promotion: heterogeneity by physician characteristics

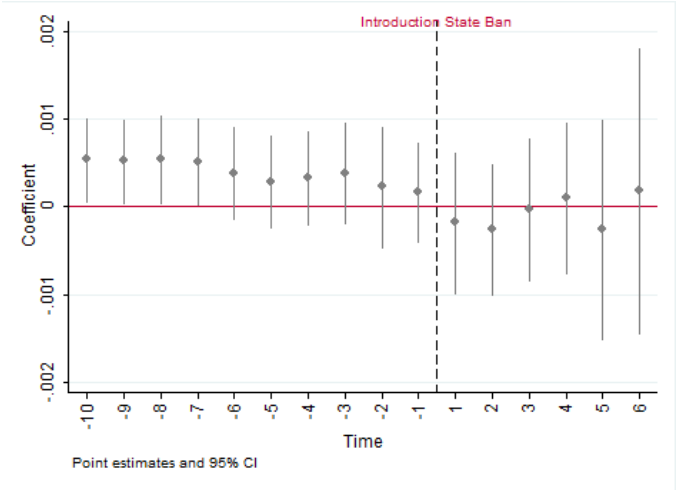
	(1) # Opioid Prescriptions	(2) # Opioid Prescriptions	(3) Received Opioid Promotion (D=1)
Received Opioid Promotion (D=1)	436.3***	278.7***	
	(68.75)	(71.27)	
Sales Rep. Ban	-11.15	-9.233	-0.00811**
	(7.780)	(7.578)	(0.00347)
Sales Rep. Ban * D	-127.8*	-144.5**	
	(72.52)	(72.69)	
Male		24.67***	0.0154***
		(2.245)	(0.00141)
Male * D		178.9***	
		(32.92)	
Graduated before 1995		51.08***	0.0203***
		(2.685)	(0.00166)
Graduated before 1995 * D		29.65	
		(41.31)	
County FE	Y	Y	Y
Physician Specialty	Y	Y	Y
Mean Dep. Var.	118.2	118.0	0.0272
SD Dep. Var.	288.2	288.2	0.163
Observations	43511	43196	67174
R2	0.267	0.280	0.0350

OLS estimates of the relationship between the number of opioid claims of Medicare Physicians and opioid promotion in columns (1) and (2), controlling for physician characteristics and the interactions with the receipt of promotion. The characteristics included are whether the physician is affiliated to a hospital with a ban on sales representatives entering the hospital in place, the gender of the physician and whether she or he graduated before 1995. Last column (3) shows the relationship between these characteristics and the probability to receive promotion for opioid drugs. All regressions control for specialty of physician and county fixed effects. Standard errors in parentheses clustered at zipcode level, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: Medicare Opioid Prescriber Summary File, 2014 AMSA Scorecard and CMS Open Payments Data 2014.

Figure 1.3: Introduction state bans on promotion orthogonal towards opioid death rates

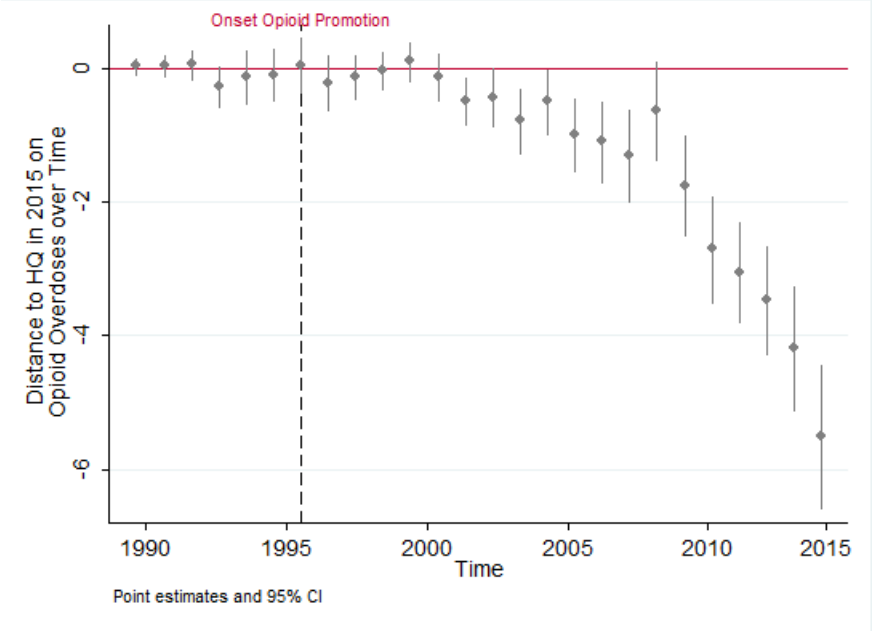


(a) Difference in opioid overdose death rates between Minnesota and the rest of the US, 1987-2015. Source: CDC Wonder Mortality MCD Data



(b) Difference in overdose rates between Massachusetts/Vermont and the rest of the US, 1999-2015 (excl. Minnesota). Source: CDC Wonder Mortality MCD Data

Figure 1.4: Reduced form estimates: distance to headquarters and overdose death rates over time.



Coefficient estimates and 95% confidence intervals of distance of county centroids to opioid promoting HQs (in 1000km) in 2015 on opioid overdose death rates, 1990-2015. Source: CMS Open Payment 2015, CDC Wonder Mortality MCD Data, company homepages for HQ location.

Table 1.12: 2SLS overdoses and promotion: small vs. large counties

Dependent Variable:	(1)	(2)
log Overdose Death in Counties with:	$<$ 100,000 inh.	$\geq$ 100,000 inh.
log Receiving Doctors	0.394***	0.399***
	(0.108)	(0.108)
Mean Dep. Var.	1.525	2.209
SD Dep. Var.	1.252	0.674
Observations	4662	1182
Partial F-Value	78.56	31.35
Year F. E.	Y	Y

2SLS regression results (see Eq. (1.3) and (1.4)), splitting set into counties with less and more than 100,000 inhabitants. Instrument: minimum distance to headquarters, that opened before 1995 and dummy for state ban on promotion. Standard errors in parentheses adjusted for heteroscedasticity, * (p<0.10), ** (p<0.05), *** (p<0.01).

Table 1.13: Illicit vs all opioid overdose deaths

	(1)	(2)	(3)	(4)
Method	OLS		IV	
log Overdose Deaths	All	Heroin	All	Heroin
log Receiving Doctors	0.102***	0.0644***	0.317***	0.336***
	(0.0138)	(0.00997)	(0.0782)	(0.0829)
Mean Dep. Var.	1.664	0.667	1.664	0.668
SD Dep. Var.	1.190	0.906	1.190	0.906
Observations	5823	5823	5840	5840
Partial F-Value	.	.	123.0	81.93
County Controls	Y	Y	Y	Y
Year F.E.	Y	Y	Y	Y

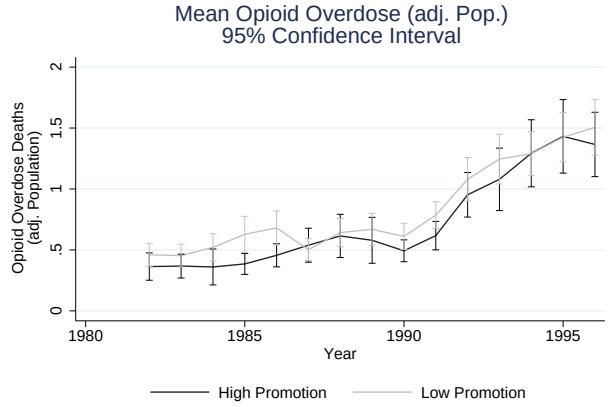
OLS and IV estimates for overdoses only including Heroin (T40.1) compared to all opioid overdoses. OLS estimate from Equation (1.1) and IV following Equation (1.3) and (1.4). Doctors receiving promotion instrumented by the distance to the closest headquarters (opened before 1995) and presence of state ban. First and second stage controls for county characteristics (see Table 1.6). Standard errors in parentheses adjusted for heteroscedasticity, * (p<0.10), ** (p<0.05), *** (p<0.01).

Appendix A

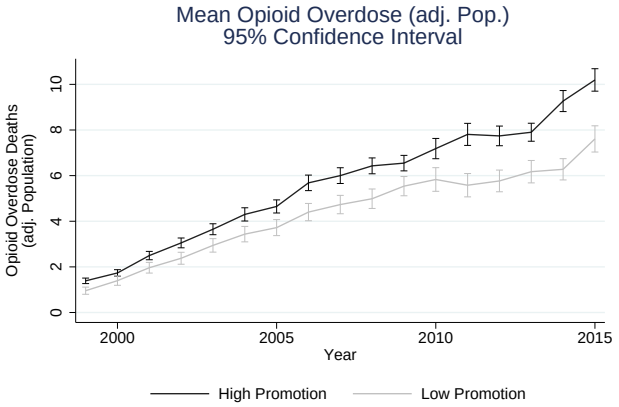
Table A-1: Data availability

Data	Available Time Period	Unit	Source
Pharmaceutical Payment Data	08/2013 - 12/2015	Physician	CMS Open Payments Data
Opioid-related Overdose Death Rates (all counties)	1999-2015	County	CDC Wonder Mortality MCD Data
Opioid-related Overdose Death Rates (counties >100,000 inh.)	1982-2015	County	CDC Wonder Mortality MCD Data
Medicare Physician Prescription Data	2013-2014	Physician	Medicare Part D Provider Data
Medicare Physician Compare	2014-2016	Physician	CMS Physician Compare
AMSA Scorecard Medical Colleges Conflict-of-Interest Policies	2008-2016	Hospital	2014 AMSA Scorecard
Neonatal Health 2014	2014	Birth	CDC 2014 Natality Detail Data Set

Figure A-1: Overdose evolution



(a) Average death rates (adj. 100.000 population) for high and low (below median) opioid promotion counties, before introduction of OxyContin. Data available for 403 counties before 1999, counties with more than 100,000 inhabitants. Source: CDC Wonder Mortality MCD Data & CMS Open Payments Data 2013-2015



(b) Average death rates (adj. 100.000 population) for high and low (below median) opioid promotion counties 1999-2015, 95% confidence interval. All counties included. Source: CDC Wonder Mortality MCD Data & CMS Open Payments Data 2013-2015.

Table A-2: Substance names used to identify opioid analgesic in payment data

Morphine	Opium	Hydromorphone
Nicomorphine	Oxycodone	Papaveretum
Ketobemidone	Pethidine	Fentanyl
Dextromoramide	Piritramide	Dextropropoxyphene
Bezitramide	Methadone	Pentazocine
Phenazocine	Butorphanol	Nalbuphine
Tilidine	Tramadol	Dezocine
Meptazinol	Tapentadol	

Source: Anatomical Therapeutic Chemical (ATC) Classification System WHOCC, ATC Code N02A

Table A-3: List of opioid promoting manufacturers

Manufacturer Operating in 2014	**Headquarters Opening**	**Reduced Set**	**Manufacturer Operating in 2015**	**Headquarters Opening**	**Reduced Set**
Galena Biopharma, Inc.	2015	No	Egalet US Inc	1995	Yes
Janssen Pharmaceuticals, Inc	1993	Yes	Galena Biopharma, Inc.	2015	No
Johnson & Johnson Health Care Systems Inc.	1886	Yes	INSYS Therapeutics Inc	1990	No
Mallinckrodt LLC	1867	Yes	Janssen Pharmaceuticals, Inc	1993	Yes
Marathon Pharmaceuticals, LLC	2010	No	Mallinckrodt LLC	1867	Yes
Mylan Pharmaceuticals Inc.	1976	Yes	Mylan Pharmaceuticals Inc.	1976	Yes
Pfizer Inc.	1961	Yes	Pfizer Inc.	1961	Yes
Purdue Pharma	2000	No	Purdue Pharma L.P.	2000	No
Upsher-Smith Laboratories Inc.	1919	Yes	The Medicines Company	1996	No
			Upsher-Smith Laboratories Inc.	1919	Yes

List of manufacturers promoting opioid medication in 2014 and 2015, respectively. Company dropped from list of headquarters to calculate closest distance if opened after 1995. INSYS Therapeutics Inc dropped for 2015 because most of the revenue generated from opioid medications. Results not sensitive to inclusion of this manufacturer.
Source: CMS Open Payments Data 2014, 2015 and company homepages for headquarters opening dates.

Table A-4: Summary statistics US county characteristics 2014 and 2015

	Mean	Std. Dev	Min	Max
2014				
Promotion (adjusted by population)				
Doctors receiving Opioid Promotion	7.20	11.47	0.00	173.65
Doctors receiving other Painkiller Promotion	11.90	16.42	0.00	165.78
Share of Expenditures spent on opioids	0.004	0.02	0.00	0.66
Minimum Distance to Headquarters (km)	0.60	0.43	0.00	4.24
Socio-economic characteristics				
Rural Dummy	0.42	0.49	0	1
Unemployment Rates	0.06	0.02	0.01	0.24
Population ('000)	101.48	326.17	0.09	10171
Log Median Income	10.73	0.24	9.98	11.74
Poverty Share	16.84	6.46	3.20	52.20
Medicare Part D enrollment	0.11	0.04	0.01	0.27
Share Whites	0.72	0.29	0.00	0.99
Industry Shares				
Natural resources & mining	0.07	0.11	0.00	1.00
Construction	0.06	0.05	0.00	0.71
Manufacturing	0.15	0.12	0.00	0.78
Trade, transportation, & utilities	0.26	0.09	0.00	1.00
Information	0.01	0.01	0.00	0.15
Financial activities	0.05	0.03	0.00	0.37
Professional & business services	0.08	0.06	0.00	0.93
Education & health services	0.17	0.08	0.00	0.82
Leisure & hospitality	0.13	0.08	0.00	0.94
Other services	0.03	0.02	0.00	0.56
Unclassified	0.00	0.00	0.00	0.07
2015				
Promotion (adjusted by population)				
Doctors receiving Opioid Promotion	5.55	9.25	0.00	135.41
Doctors receiving other Painkiller Promotion	13.66	19.45	0.00	224.13
Share of Expenditures spent on opioids	0.004	0.03	0.00	1.00
Minimum Distance to Headquarters (km)	0.57	0.39	0.00	4.24
Socio-economic characteristics				
Rural Dummy	0.42	0.49	0	1
Unemployment Rates	0.06	0.02	0.02	0.24
Population ('000)	102.30	329.21	0.09	10171
Log Median Income	10.76	0.24	10.04	11.74
Poverty Share	16.26	6.44	3.40	47.40
Medicare Part D enrollment	0.11	0.04	0.01	0.27
Share Whites	0.71	0.29	0.00	0.99
Industry Shares				
Natural resources & mining	0.06	0.10	0.00	1.00
Construction	0.06	0.04	0.00	0.75
Manufacturing	0.14	0.12	0.00	0.78
Trade, transportation, & utilities	0.26	0.09	0.00	1.00
Information	0.01	0.01	0.00	0.13
Financial activities	0.05	0.03	0.00	1.00
Professional & business services	0.08	0.06	0.00	0.94
Education & health services	0.17	0.08	0.00	0.79
Leisure & hospitality	0.13	0.08	0.00	0.93
Other services	0.03	0.02	0.00	0.28
Unclassified	0.00	0.00	0.00	0.08

Table A-5: OLS estimates promotion & neonatal health: heterogeneity by mothers' characteristics

	(1) Admission NICU	(2) Ventilation Immediately	(3) Ventilation > 6hr	(4) APGAR 5	(5) Birth Weight	(6) Low BW < 2500g	(7) Premature Born
			Panel A: Mother is a Medicaid recipient				
Promotion 9 months before	0.00352***	0.000257	0.000460*	-0.00260	0.595	0.00102**	0.000570
Delivery	(0.000742)	(0.000533)	(0.000258)	(0.00333)	(1.365)	(0.000469)	(0.000522)
Promotion 9 months before	0.00413***	0.000805*	0.000613***	-0.0125***	-16.48***	0.00556***	0.00436***
Delivery * Medicaid recipient	(0.000751)	(0.000454)	(0.000187)	(0.00266)	(1.899)	(0.000631)	(0.000655)
Medicaid recipient	0.00741***	0.00201*	0.000659*	-0.0144**	-73.68***	0.0119***	0.0176***
	(0.00180)	(0.00113)	(0.000386)	(0.00602)	(5.534)	(0.00144)	(0.00161)
Observations	3755970	3755970	3755970	3750114	3762586	3762586	3762955
R2	0.00333	0.00490	0.00332	0.0138	0.0121	0.00320	0.00438
			Panel B: Mother smokes				
Promotion 9 months before	0.00478***	0.000686	0.000660**	-0.00825**	-5.162***	0.00309***	0.00203***
Delivery	(0.000802)	(0.000516)	(0.000257)	(0.00367)	(1.523)	(0.000560)	(0.000582)
Promotion 9 months before	0.00405***	0.0000883	0.000600***	0.00115	-5.677***	0.00235***	0.00201***
Delivery * Smokes	(0.000963)	(0.000416)	(0.000221)	(0.00217)	(1.330)	(0.000625)	(0.000609)
Smokes	0.0211***	0.0128***	0.00393***	-0.0303***	-194.5***	0.0505***	0.0307***
	(0.00210)	(0.00113)	(0.000587)	(0.00587)	(3.315)	(0.00145)	(0.00143)
Observations	3717944	3717944	3717944	3711537	3724211	3724211	3724514
R2	0.00323	0.00514	0.00343	0.0136	0.0138	0.00461	0.00374
			Panel C: Mother's age				
Promotion 9 months before	0.00446***	0.000499	0.000582**	-0.00533	-2.599*	0.00220***	0.00145**
Delivery	(0.000859)	(0.000577)	(0.000296)	(0.00345)	(1.468)	(0.000532)	(0.000607)
Promotion 9 months before	0.000937**	0.0000632	0.000162	-0.00336***	-4.322***	0.00142***	0.000879**
Delivery * < 30 yrs old	(0.000390)	(0.000303)	(0.000135)	(0.00125)	(0.940)	(0.000301)	(0.000377)
< 30 yrs old	-0.00959***	-0.00220***	-0.00162***	0.00297	-32.06***	-0.00758***	-0.0139***
	(0.000934)	(0.000692)	(0.000290)	(0.00282)	(2.888)	(0.000736)	(0.000880)
Observations	3790596	3790596	3790596	3926976	3940161	3940161	3940321
R2	0.00287	0.00486	0.00329	0.0139	0.00582	0.00167	0.00315
Mean Dep. Var.	0.0824	0.0353	0.0114	8.781	3273.1	0.0805	0.113
SD Dep. Var.	0.275	0.185	0.106	0.839	591.9	0.272	0.316

Estimation result of Equation (1.1). Opioid promotion measured as number of doctors receiving opioid promotion in the county of birth during pregnancy (normalized by county population). Heterogeneity by mother's health insurance status (a dummy for whether she is a Medicaid recipient or not), by mother's age (if she was less than 30 years old at delivery) and whether she reports to be a smoker or not. Standard errors in parentheses clustered at county level, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: CDC 2014 Natality Detail Data Set and CMS Open Payments Data 2014, 2015.

Table A-6: OLS estimates promotion & prescriptions: different functional specifications

Functional Form	Linear	Log	Elasticity	Deciles
Dep. Var.:	(1)	(2)	(3)	(4)
Opioid Prescriptions	# Pres. 2014	# Pres. 2014	# Pres. 2014 (log)	# Pres. 2014
Opioid Promotion (USD)	0.00526**			
	(0.00238)			
Opioid Promotion (log USD)		15.55***	0.114***	
		(0.895)	(0.00168)	
D=1 Decile 10 (< 11 USD)				25.54***
				(4.576)
D=1 Decile 20 (13 USD)				26.31***
				(4.537)
D=1 Decile 30 (15 USD)				23.49***
				(3.674)
D=1 Decile 40 (18 USD)				22.92***
				(4.904)
D=1 Decile 50 (23 USD)				21.40***
				(4.494)
D=1 Decile 60 (29 USD)				28.96***
				(4.136)
D=1 Decile 70 (38 USD)				38.59***
				(5.560)
D=1 Decile 80 (54 USD)				58.39***
				(6.487)
D=1 Decile 90 (98 USD)				75.21***
				(8.686)
D=1 Decile 100 (> 98 USD)				151.5***
				(12.87)
Mean Dep. Var.	114.9	114.9	2.958	114.9
SD Dep. Var.	322.3	322.3	2.176	322.3
Observations	633306	633306	633306	633306
R2	0.888	0.889	0.752	0.889
County F.E.	Y	Y	Y	Y
Specialty F.E.	Y	Y	Y	Y
Previous Prescription Rates	Y	Y	Y	Y

Number of opioid claims of Medicare Physicians and opioid-related promotion. Estimation result of Equation (1.5). All regressions control for specialty of physician, prescription rates in the previous year and county fixed effects. Standard errors in parentheses clustered at zip-code level, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: Medicare Opioid Prescriber Summary File and CMS Open Payments Data 2014.

Table A-7: Promotion and share of opioid claims over all claims

	(1) % Opioid Claims	(2) % Opioid Claims
Opioid Promotion	0.00239***	0.00400***
	(0.000200)	(0.000215)
Non-Opioid Non-Painkiller Promotion		-0.00156***
		(0.0000692)
Non-Opioid Painkiller Promotion		-0.000390**
		(0.000156)
% Opioid Claims 2013	0.943***	0.943***
	(0.00153)	(0.00153)
County FE	Y	Y
Mean Dep. Var.	0.125	0.125
SD Dep. Var.	0.177	0.177
Observations	633306	633306
R2	0.688	0.689

Outcome variable: share of opioid claims over all claims by Medicare Physicians and pharmaceutical promotion. Estimation result of Equation (1.5), for opioid promotion, painkiller promotion and non-opioid/non-painkiller promotion. Promotion measured as log dollar amount received in corresponding year. All regressions control for specialty of physician, prescription shares in the previous year and county fixed effects. Standard errors in parentheses clustered at zipcode level, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: Medicare Opioid Prescriber Summary File and CMS Open Payments Data 2014.

Table A-8: 2SLS: single instruments and Sargan's overidentification test

	All counties			<500km to HQs		
Dep. Var.: log Opioid Overdose Deaths	(1)	(2)	(3)	(4)	(5)	(6)
Instruments used:	Both	Distance only	Ban only	Both	Distance only	Ban only
log Opioid Promotion Receiving Doctors	0.337***	0.765***	0.152	0.337***	0.279*	0.359***
	(0.0825)	(0.189)	(0.121)	(0.0713)	(0.155)	(0.0981)
Mean Dep. Var.	1.664	1.664	1.664	1.861	1.861	1.861
SD Dep. Var.	1.191	1.191	1.191	1.186	1.186	1.186
Observations	5844	5844	5844	2897	2897	2897
Year F.E.	Y	Y	Y	Y	Y	Y
County Characteristics	Y	Y	Y	Y	Y	Y
Partial F-Value	93.80	21.82	98.64	123.6	28.57	134.2
Sargan P-Value	0.0009	.	.	0.252	.	.

2SLS regression results (see Eq. (1.3) and (1.4)) using i) both instruments, ii) the instruments separately for a) all counties and for b) all counties within 500km distance to the closest headquarters. Partial F-value of first stage Equation (1.3) and P-Value of Sargan overidentification test displayed in last two rows. Standard errors in parentheses adjusted for heteroscedasticity, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$).

Table A-9: IV 2SLS Overdoses and promotion: pre-year level of overdose deaths and non-opioid promotion

Dep. Var.: log Opioid Overdose Deaths	(1)	(2)	(3)
log Opioid Promotion Receiving Doctors	0.317***	0.166**	0.359*
	(0.0782)	(0.0718)	(0.198)
log Opioid Overdose Deaths in t-1		0.418***	
		(0.0180)	
log Non-Opioid Promotion Receiving Doctors			-0.0388
			(0.0829)
Mean Dep. Var.	1.664	1.682	1.664
SD Dep. Var.	1.190	1.182	1.190
Observations	5840	5748	5840
Partial F-Value	123.0	130.2	22.61
County Characteristics	Y	Y	Y
Year F.E.	Y	Y	Y

2SLS regression results (see Eq. (1.3) and (1.4)). First column shows main specification. Second column controls for pre-year level of overdoses. Column (3) controls for non-opioid promotion in the corresponding year. Instrument: minimum distance to headquarters, that opened before 1995 and dummy for state ban on promotion. Standard errors in parentheses adjusted for heteroscedasticity, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$).

Table A-10: Diabetes mellitus/strokes & promotion: 2SLS

Dep. Var.: log Deaths	(1) Diabetes	(2) Stroke
log Receiving Doctors	0.0426	0.0257
	(0.0339)	(0.0252)
Mean Dep. Var.	3.456	3.822
SD Dep. Var.	0.483	0.450
Observations	2861	3624
R2	0.487	0.557
Partial F-Value	44.04	73.52
County Characteristics	Y	Y
Year F. E.	Y	Y

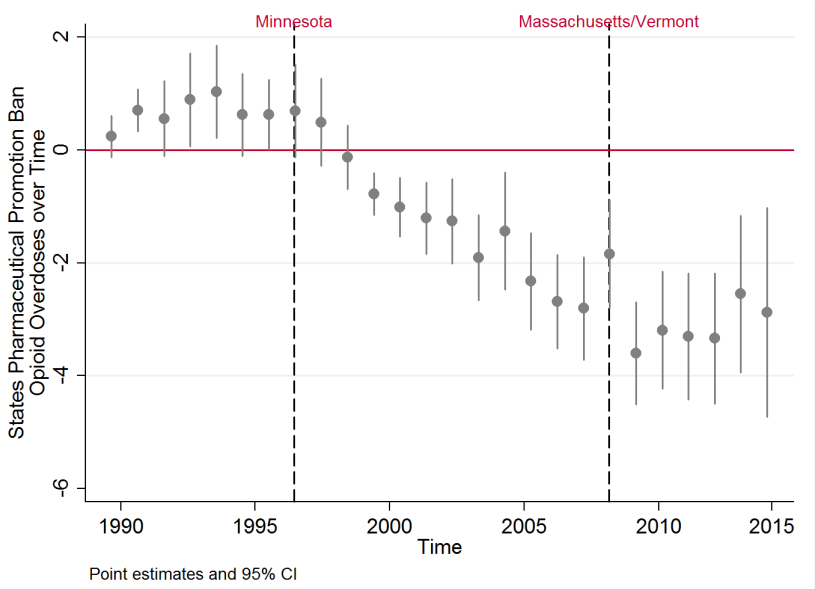
Death rates caused by diabetes or strokes (ICD-Codes: E10-E14 and I60-I69) and opioid-related promotion. Regression results of Equations (1.3) and (1.4). Promotion instrumented with the distance of a county to the closest headquarters and the presence of a state ban. Partial F-Value of first stage displayed in last row. All regressions control for county characteristics (see Table 1.6 for details). Standard errors in parentheses adjusted for heteroscedasticity, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: CDC Wonder Mortality MCD Data and CMS Open Payments Data 2014 and 2015.

Table A-11: Overdose & promotion: 2SLS and OLS promotion in USD

Empirical Strategy	OLS	IV	IV
Dependent Variable:	(1)	(2)	(3)
log Opioid Overdose Deaths			
log Opioid Promotion USD	0.0571***	0.154***	0.132**
	(0.00655)	(0.0449)	(0.0577)
log Non-Opioid Promotion USD			0.0125
			(0.0260)
Mean Dep. Var.	1.664	1.664	1.664
SD Dep. Var.	1.190	1.190	1.190
Observations	5823	5840	5840
R2	0.331	0.147	0.159
Partial F-Value	.	75.00	48.39
County Characteristics	Y	Y	Y
Year F.E.	Y	Y	Y

Number of opioid overdose deaths in a county and opioid-related promotion. Promotion measured as logarithm of sum of USD amount spent on opioid promotion in a given county. Measures adjusted by population (100,000 inhabitants). Regression results of Equations (1.3) and (1.4). Promotion instrumented with the distance of a county to the closest headquarters and the presence of a state ban. In column (3) we additionally control in the first and second stage for all pharmaceutical promotion spending in the county, that is not related to opioid drugs. Partial F-Value of first stage displayed in last row. All regressions control for county characteristics (see Table 1.6 for details). Standard errors in parentheses adjusted for heteroscedasticity, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: CDC Wonder Mortality MCD Data and CMS Open Payments Data 2014 and 2015.

Figure A-2: Reduced form estimates: pharmaceutical promotion state bans and overdose death rates over time.



Coefficient estimates and 95% confidence intervals of state ban dummy (Minnesota, Vermont, Massachusetts) on opioid overdose death rates, 1990-2015. Source: State legislations, CDC Wonder Mortality MCD Data.

Opioid promotion: IV promotion unrelated drugs

In this section we check for robustness of our results by following a different 2SLS IV strategy. We now instrument the number of doctors who receive promotion for opioid drugs in a county with the number of doctors that receive promotion for unrelated drugs. We pick the 20 most promoted drugs in a given year, that are independent of opioid medication, such as drugs for diabetes or hypertension. We follow the same empirical strategy as described in Equations (1.3) and (1.4) and control for county characteristics in the first and second stage. See footnote of Table 1.6 for details on county characteristics.

Table A-12: List of unrelated promoted drugs

Top 20 Promoted Drug in 2014	Purpose	Manufacturer	Top 20 Promoted Drug in 2015	Purpose	Manufacturer
Eliquis	Blood Thinner	Bristol-Myers Squibb & Pfizer	Xarelto	Blood Thinner	Janssen
Myrbetriq	Overactive Bladder	Astellas	Eliquis	Blood Thinner	Bristol-Myers Squibb & Pfizer
Azor	Hypertension	Daiichi Sankyo	Levemir	Diabetes Type 2	Novo Nordisk
Eylea	Retina Diseases	Bayer	Nexplanon	Contraceptive	Merck
Aczone	Acne	Allergan	Victoza	Diabetes Type 2	Novo Nordisk
Prepopik	Clean colon before colonoscopy	Ferring	Cleviprex	Hypertension	Chiesi
Celebrex	Athritis	Pfizer	Pradaxa	Blood Thinner	Boehringer Ingelheim
Bydureon	Diabetes Type 2	AstraZeneca	Quillivant	ADHD	Pfizer
Januvia	Diabetes Type 2	Merck	Namenda	Alzheimer's Disease	Merz
Aptiom	Anti-seizure	Sunovion	Brilinta	Lower risk heart attack	AstraZeneca
Toviaz	Overactive Bladder	Pfizer	Toujeo	Diabetes Type 2	Sanofi-Aventis
Tanzeum	Diabetes Type 2	GSK	Invokana	Diabetes Type 2	Janssen
Novolog	Diabetes Type 2	Novo Nordisk	Vytorin	Reduce Cholesterol	Merck
Quillivant	ADHD	Pfizer	Arestin	Microbial Plaque	Valeant
Victoza	Diabetes Type 2	Novo Nordisk	Bydureon	Diabetes Type 2	AstraZeneca
Apidra	Diabetes Type 1/2	Sanofi-Aventis	Uloric	Gout	Takeda
Brisdelle	Relief Hot Flashes	Sebela	Neox	Ascariasis/Enterobiasis	Bristol-Myers Squibb
Welchol	Diabetes Type 2	Daiichi Sankyo	Duavee	Relief Hot Flashes	Pfizer
Premarin	Relief Hot Flashes	Pfizer	Edarbyclor	Hypertension	Arbor & Takeda
Colcrys	Treat gout attacks	Takeda	Entresto	Heart Failure	Novartis

List of top 20 drugs unrelated to pain or opioid medication, promoted in 2014 and 2015, respectively. Source: CMS Open Payments Data 2014, 2015 and manufacturer homepages for purpose and manufacturer names.

Table A-13: Overdose & promotion: 2SLS IV unrelated drugs

	Panel A: First Stage	
Dependent Variable: log Opioid Promotion	(1)	(2)
log Unrelated Promotion	0.332***	0.349***
	(0.00753)	(0.00829)
log Opioid Overdose Death in t-1		0.0828***
		(0.0126)
Mean Dep. Var.	1.299	1.095
SD Dep. Var.	1.307	1.241
Observations	3133	3133
R2	0.447	0.408
Partial F-Value	1927.8	1805.3

	Panel B: Second Stage	
Dep. Var.: log Opioid Overdose Deaths	(1)	(2)
log Opioid Promotion	0.275***	0.182***
	(0.0289)	(0.0283)
log Opioid Overdose Death in t-1		0.411***
		(0.0146)
Mean Dep. Var.	1.664	1.682
SD Dep. Var.	1.190	1.182
Observations	5840	5748
R2	0.152	0.302
County Characteristics	Y	Y
Year F.E.	Y	Y

Number of opioid overdose deaths in a county and opioid-related promotion (number of doctors receiving promotion). Panel A displays first stage results. Opioid promotion is instrumented with the number of doctors that receive pharmaceutical promotion for unrelated drugs to opioids (see above for description). In the second column we additionally control for opioid overdose death rates in the previous year. All regressions control for county characteristics in Panel A and Panel B. Standard errors in parentheses adjusted for heteroscedasticity, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: CDC Wonder Mortality MCD Data and CMS Open Payments Data 2014 and 2015.

Table A-14: 2SLS IV unrelated drugs: neonatal health and opioid promotion

	Panel A: Second Stage Results						
	(1) Admission NICU	(2) Ventilation Immediately	(3) Ventilation $>$ 6hr	(4) APGAR 5	(5) Birth Weight	(6) Low BW $<$ 2500g	(7) Premature Born
Promotion 9 Months before Delivery	0.00949*** (0.00162)	0.00163 (0.00104)	0.00154*** (0.000473)	-0.0187*** (0.00602)	-11.03*** (1.824)	0.00522*** (0.000773)	0.00503*** (0.000916)
Mean Dep. Var.	0.0808	0.0351	0.0112	8.785	3280.2	0.0777	0.110
SD Dep. Var.	0.273	0.184	0.105	0.825	584.9	0.268	0.313
Observations	3436124	3436124	3436124	3429416	3439713	3439713	3440894
Mother's Demographics	Y	Y	Y	Y	Y	Y	Y
Birth Characteristics	Y	Y	Y	Y	Y	Y	Y
Month of Birth F.E.	Y	Y	Y	Y	Y	Y	Y
State F.E.	Y	Y	Y	Y	Y	Y	Y
	Panel B: First Stage Results						
β Promotion Unrelated Drugs	0.0331*** (0.00175)	0.0331*** (0.00175)	0.0331*** (0.00175)	0.0331*** (0.00175)	0.0331*** (0.00175)	0.0331*** (0.00175)	0.0331*** (0.00175)
Partial F-Value	359.7	359.7	359.7	359.6	360.0	360.0	360.0

Estimation result of Equations (1.3) and (1.4). Opioid promotion measured as number of doctors receiving opioid promotion in the county of birth during pregnancy (normalized by county population). Partial F-value of first stage Equation (1.3) displayed in last row. Coefficient estimates and standard errors of first stage regression displayed in Panel B. Number of physicians receiving opioid promotion instrumented with number of physicians receiving promotion for unrelated drugs (see Table A-12 column 2014 for list of drugs). Mother's characteristics controlled for in all regressions are age, race, educational attainment, marital status, mother medicaid recipient, mother's health (BMI, hypertension, diabetes), whether mother was born in the US and whether the mother is a smoker. Characteristics of births included in all regressions: vaginal delivery, sex of the baby, birth order, early prenatal visits, attendant at birth is physician. State fixed effects included in all regressions. Standard errors in parentheses clustered at county level, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: CDC 2014 Natality Detail Data Set and CMS Open Payments Data 2014, 2015.

Table A-15: 2SLS IV unrelated drugs: physician prescriptions and opioid promotion

	Panel A: Second Stage Results	
Dependent Variable:	(1)	(2)
Opioid Prescriptions	# Pres. 2014	# Pres. 2014
Opioid Promotion (Dummy)	102.3***	
	(9.617)	
Opioid Promotion (log USD)		27.77***
		(2.727)
# Opioid Pres. 2013	0.968***	0.967***
	(0.00797)	(0.00814)
Mean Dep. Var.	114.9	114.9
SD Dep. Var.	322.3	322.3
Observations	633306	633306
County F.E.	Y	Y
Physician Specialty	Y	Y
	Panel B: First Stage Results	
β Promotion Unrelated	0.0826***	0.0660***
Drugs	(0.00114)	(0.000949)
Partial F-Value	5640.5	5187.1

Number of opioid claims of Medicare Physicians and opioid-related promotion. Estimation results of Equations (1.5) and (1.6). Promotional level measured as dummy for any promotion in column (1) and as log dollar amount in column (2). Opioid promotion instrumented with the receipt of promotion for unrelated drugs (see Table A-12 column 2014 for list of drugs). Coefficient estimate and partial F-statistics from first stage displayed in Panel B. All regressions control for specialty of physician, previous prescription rates and county fixed effects. Standard errors in parentheses clustered at zip-code, * (p<0.10), ** (p<0.05), *** (p<0.01). Source: Medicare Opioid Prescriber Summary File and CMS Open Payments Data 2014.

Chapter 2

OPIOID PAINKILLER AVAILABILITY AND SUICIDE RATES IN THE US

Joint with Mark Borgschulte (UIUC)

2.1 Introduction

Between 1999 and 2014, the US experienced a sharp increase in opioid pain reliever prescriptions (Figure 2.1), opioid overdoses, and the number of people with substance abuse disorders (McCabe et al., 2008; Blanco et al., 2007; Okie, 2010). In an influential series of papers, Anne Case and Angus Deaton tie these trends to rising deaths in midlife from suicide and other drug- and alcohol-related causes, and argue that rising mortality rates are symptoms of widespread despair resulting "from a long-standing process of cumulative disadvantage for those with less than a college degree" (Case and Deaton, 2017). This *demand-side* explanation for the opioid epidemic explanation carries with it profound implications about social welfare in affected communities: according to this view, social and economic opportunities for the working class have deteriorated to the point that many lives are not worth living. Policy responses should therefore target economic interventions in conjunction with or ahead of public health measures, and changes in medical

practices.

An important alternative explanation points to a *supply-side* story, driven by changes in prescribing guidelines for opioid analgesics. This explanation is featured in many medical and public health accounts of the epidemic, and imply that changes in prescribing, drug treatment, and public health measures may be most effective in responding to the epidemic. However, the power of the supply side explanation for the rise in all-cause mortality is limited by the role of other diseases of despair, particularly suicide.

In their response to Ruhm (2018), who finds that changes in economic conditions explain only little variation in opioid-involved mortality rates, Case and Deaton argue "We think of all of these deaths as suicides, by a very broad definition, and we attribute them to a broad deterioration in the lives of Americans without a college degree who entered adulthood after 1970." (Case and Deaton, 2018). If deaths of despair are suicides, reforms to reduce drug abuse may have limited effects.

In this paper, we provide the first estimates of the causal effect of increases in the prescribing of opioid analgesics on suicide. To do so, we analyze drug company promotions to doctor, a specific supply shock that we show leads to increases in prescriptions written by doctors. We find that higher promotional activities for opioid drugs are associated with higher suicide rates in 2014 and 2015. The estimates imply that increasing the number of physicians that receive promotion for opioid drugs by 1%, increases suicide rates by 0.07% (the 95% confidence interval ranges from 0.03 to 0.11%). We identify the effect by instrumenting the number of doctors receiving opioid promotion with the number of doctors receiving promotion for drugs unrelated to mental illnesses and pain (e.g., diabetes medication, blood thinner). The results imply that approximately 2% of the variation in non-poisoning suicide rates can be explained by promotion of opioid analgesics to physicians. Fernandez and Zejcirovic (2017) show that pharmaceutical promotion of opioid drugs can explain differences in opioid prescription rates using Medicare Part D prescription data for 2013 and 2014.

While recent medical research has concluded that there is no evidence for the effectiveness of long-term opioid therapy for improving non-malignant chronic pain, there is a risk of dependency (Chou et al., 2015). Substance abuse disorder

is documented to be a high risk factor for suicide (Chesney et al., 2014). Evidence from the medical literature also shows higher suicide risk of individuals diagnosed with opioid use disorder, controlling for comorbid psychiatric and physical conditions (Ashrafioun et al., 2017; Ilgen et al., 2016).

We combine county-level data on death rates (CDC Wonder, December 2016) with recently released data on pharmaceutical promotion payments to physicians aggregated at the county-level (CMS, 2016). We instrument the number of doctors receiving promotion for opioid drugs with the number of doctors receiving promotion for unrelated drugs. The idea behind this instrument is that counties have high numbers of doctors receiving promotion for opioid drugs simply because the sales representatives are also promoting these unrelated drugs. For every year, we look for the 20 most promoted drug unrelated to pain and mental health. We choose the drugs that reach the highest number of doctors.[1]

Our key findings are robust to several specification checks. First, we control for county characteristics that could potentially correlate with pharmaceutical promotion to physicians and suicide rates. Economic conditions, such as unemployment rates, are shown to be important determinants for suicide rates (Classen and Dunn, 2012) and substance abuse disorder involving analgesics (Carpenter et al., 2017). Other confounding factors that we control for are poverty rates, Medicare Part D enrollment rates, divorce rates in 2010, median income and industry shares. The coefficient estimate on the relationship between promotion and suicide rates is robust to the inclusion of these county characteristics. This limits the concern that we are solely picking up a county-specific, time variant relationship between demand for opioids and suicide rates. Second, our instrument may proxy a general high demand for drugs in a county and potentially high prevalence of mental illnesses and suicidal individuals. The reduced form results show that the positive relationship between the number of doctors receiving promotion for unrelated drugs disappears once we include the number of doctors that receive promotion for opioid drugs. This suggests that the positive association we observe in the reduced form operates through opioid promotion.

To shed light on why opioid promotion raises non-poisoning suicide rates, we

[1]The drugs, their purpose and manufacturer can be found in Table A-12 in the Appendix of Chapter 1.

perform the following heterogeneity analyses. Opioid use or abuse has been argued to increase suicide rates because it i) increases the means to commit suicide, ii) increases the likelihood that suicidal individuals act on impulses and iii) increases the number of individuals with substance abuse disorder (SUD) of whom some will commit suicide. As we do not have estimates on the number of people with SUDs nor the number of people with suicidal thoughts, we perform heterogeneity analyses to examine potential mechanisms. First, as we are considering non-poisoning suicide rates and find a positive relationship between opioid painkiller availability and suicides, hypothesis i) is unlikely to be the only driving mechanism. Including these deaths increases the coefficient estimates which indicates that the availability of more means, indeed, is an important determinant of suicide rates. Second, we analyze whether the relationship between opioid availability and suicide rates differs by the strength of the addiction-help network in the county. We find that the positive relationship is mitigated in counties with a high number of substance abuse treatment centers. This suggests that the prevalence of SUDs is driving differences between suicide rates. Lastly, we investigate the relationship between opioid promotion and suicide rates by the presence of mandatory-access prescription drug monitoring programs (PDMPs). Prescriptions for controlled substance drugs are collected in a centralized database in these states informing about the behavior of patients and health care providers. Health care providers can check whether their patients received prescriptions for controlled substances from another provider. We find that there is no significant relationship between opioid promotion and suicide rates in states with a mandatory access prescription drug program in place. Using Medicare Part D prescription data, we find that physicians in these states are less sensitive towards opioid promotion.

This study contributes to the literature on the economics of suicide by providing evidence of the importance of opioid painkiller availability in explaining cross-sectional variation in non-poisoning suicide rates.[2] These results comple-

[2]The literature on the economics of suicide has studied the role of socioeconomic factors such as unemployment (Classen and Dunn, 2012), income inequality (Andres, 2005; Daly et al., 2013), divorce laws (Stevenson and Wolfers, 2006), alcohol consumption (Carpenter, 2004) and others. Closely related to our study is the work by Ludwig et al. (2009) showing that increases in sales of anti-depressants (SSRIs) decrease suicide rates.

ment work by Borgschulte et al. (2018) which finds that suicides can increase or decrease in response to supply restrictions to the market for diverted prescription drugs, depending on the availability and efficacy of treatment services. The key feature in our setting is that we study an increase in the supply of opioid drugs. Our hypothesis also closely relates to Ruhm (2018) and Carpenter et al. (2017) which examine the scope for economic explanations of the epidemic. Our work additionally contributes to the literature studying the origins of the opioid epidemic, by highlighting the role of pharmaceutical promotion in opioid prescribing patterns of physicians.[3]

The paper is structured as follows. Section 2.2 provides background information on the practice of pharmaceutical promotion to physicians in the US and the relationship between opioid use and suicide. Section 2.3 describes the data sources and provides basic descriptive statistics. Section 2.4 discusses the empirical strategy, followed by the estimation results in Section 2.5. Subsections 2.5.2 to 2.5.3 explore heterogeneous effects of opioid promotion on non-poisoning suicide rates and on prescription rates. Section 2.6 concludes the chapter.

2.2 Background Information: Pharmaceutical Promotion and Opioid Use and Suicide

2.2.1 Pharmaceutical Promotion

Pharmaceutical promotion to physicians is a common practice in many countries. Pharmaceutical companies in the US spend billion dollars every year on advertisement of their drugs and medical devices. The largest share of their advertisement budget is generally devoted to direct advertisement to physicians and other health care professionals (Cegedim, 2013). A nationally representative study showed that more than 80% of all physicians in the US received some form of gift by a pharmaceutical representative in 2004 (Campbell et al., 2007).

[3] Another study analyzing differences in prescription behavior is the recent work by Currie and Schnell (2018) who find that physicians that graduated from higher ranking medical schools prescribe significantly fewer opioids.

In the economic literature, previous studies show that interactions of physicians with pharmaceutical sales representatives influence the prescribing practices of the former. Engelberg et al. (2014) find that physicians receiving promotion of branded drugs reduce prescription rates for generic drugs and increase prescriptions in favor of the paying firm's drugs (similarly Datta and Dave (2017)). Using Medicare Part D prescription data, Fernandez and Zejcirovic (2017) show that promotion of opioid drugs increases the prescriptions rates for patients enrolled in the program.

In promoting directly to physicians, pharmaceutical sales representatives have room for misinformation. Studies show that the information provided by sales representatives is not always accurate. Villanueva et al. (2003) assess the accuracy of promotional material circulated by pharmaceutical companies in Spain and conclude that in 44% of the claims made in advertisements, the references provided did not support the statements (similar results have been found for the US by Wilkes et al. (1992)).

Purdue Pharmaceuticals was among the first companies promoting the opioid analgesic OxyContin, for the treatment of chronic (non-malignant) pain in 1996. In its promotional campaign, Purdue asserted that the risk of addiction from OxyContin was extremely small and sales representatives claimed that the risk of addiction was less than 1%, a statement that cannot be backed up with empirical evidence from medical studies (Van Zee, 2009). During the late 1990s, other pharmaceutical manufacturers followed the promotional efforts of Purdue and extended the marketing of their opioid pain relievers. Promotion was not only directed at pain specialists, oncologists or palliative care specialists but also at primary care physicians (Van Zee, 2009). There is no evidence for the superiority of opioid drugs over other medications and forms of therapy in improving non-malignant chronic pain. There is, however, evidence for the risk of dependency and overdose death (Chou et al., 2015).

A growing number of legal actions against opioid manufacturers suggests that this commercial success has not been harmless. In the past two years, different counties have pressed charges against some of the companies promoting opioid medications for misbranding and underrepresentation of the risk of addiction.[4]

[4]The City of Chicago, Orange County and Santa Clara Counties filed lawsuits against Purdue

2.2.2 Opioid Use and Suicide

Substance abuse disorder is a high risk factor for suicidal thought and intent. Causally identifying the effect of the disorder on suicide rate is challenging as it is generally accompanied by other mental illnesses or other risky behavior. Different meta-studies show that the presence of a substance abuse disorder has the highest suicide mortality ratio, following the presence of mood disorders (Mościcki, 1995; Cavanagh et al., 2003).

In recent years, medical research has concluded that there is evidence for the risk of dependency of long-term opioid therapy in patients with non-malignant chronic pain (Chou et al., 2015). In addition, there are more people in the US individuals with opioid use disorder (McCabe et al., 2008; Blanco et al., 2007).

Recent evidence from the medical literature indicates positive correlations between opioid abuse and the dosage of opioid use and suicidal intents (Ashrafioun et al., 2017; Ilgen et al., 2016). Ilgen et al. (2016) study the association between prescribed opioid dose and suicide risk in a sample of nearly 5 million veterans. They find that higher dosages of opioids are correlated with increased suicide risk, controlling for demographic and clinical factors, such as pain, psychiatric conditions and the presence of substance abuse disorder. Interestingly, they do not find a relationship between the dosage of acetaminophen and suicide risk, another drug used in pain therapy. The Department of Veterans Affairs and Department of Defense treatment guidelines define higher suicide risk as a relative contraindication for opioid therapy (Department of Veterans Affairs and Department of Defense, 2010). This association is not exclusive to veterans. Analyzing the 2014 National Survey of Drug Use and Health, a nationally representative survey, Ashrafioun et al. (2017) show that prescription opioid misuse is significantly associated with suicidal ideation, planning, and attempts for high-frequency users. This positive correlation survives the inclusion of demographics, overall health conditions and the presence of depression, anxiety and substance abuse disorder.

None of the mentioned studies on substance abuse and suicide attempts address causality. Borgschulte et al. (2018) provide the first quasi-experimental evi-

Pharma LP, Teva Pharmaceutical Industries Ltd, Johnson & Johnson, Endo Health Solutions Inc and Allergan PLC in 2014.

dence linking the rise in prescription drug abuse and suicides among non-Hispanic Whites in the US. They show that the introduction of PDMPs lead to higher decreases in suicides in counties with strong addiction-help networks, implying that there is an inherent risk of suicide associated with prescription drug abuse.

2.3 Data and Descriptive Statistics

We combine multiple sources of data for our analysis. Our outcome variable is the number of people that committed suicide in a county in a given year that did not involve any form of poisoning (ICD Code 10: X70-X84). The CDC Wonder Multiple Cause of Death database provides county-level mortality data based on death certificates for U.S. residents (CDC, 2016). Summary statistics for our study period (2013-2015) can be found in Table 2.1. On average 16.85 people committed suicide per 100,000 inhabitants in a county in a year. 14.81 people died using non-poisoning means.

The second data source we use is the CMS Open Payment Data on payments made by drug and device companies to physicians and other health care professionals in the US (CMS, 2016).[5] The payment data used in this study covers the period from January 2014 until December 2015. Our independent variable of interest is the number of doctors that receive promotion for opioid drugs. We classify a drug an opioid analgesic following the Anatomical Therapeutic Chemical (ATC) Classification Scheme of the World Health Organization (ATC Code N02A). We exclude opiates that reverse opioid overdose, such as naloxone. In every county, on average 6.4 doctors per 100,000 inhabitants received opioid promotion per year. To instrument our independent variable, we use the number of doctors that receive pharmaceutical promotion for drugs unrelated to pain and mental health. For every year, we look for the 20 most promoted drug unrelated to pain and mental health. We choose the drugs that reached the highest number of doctors. Table A-12 lists these drugs, their manufacturer and purpose. On average, 94 physicians receive promotion for these unrelated drugs in every county. The drugs considered treat illnesses such as hypertension, diabetes, and hyperac-

[5] See subsection 1.3 for more detailed information on the database.

tive bladders. These are conditions arguably unrelated to the need or demand of opioid medication.

The third data source is the Medicare Provider Utilization Data 2013, 2014, and 2015 collected by the CMS. These files contain information on physicians and other health care providers paid for under the Medicare Part D Prescription Drug Program. The data includes information on the health care providers' names, specialties, addresses, and the amount of opioid prescriptions written in 2013, 2014, and 2015. The summary statistics depicted in Table 2.1 aggregate the prescriptions written by these health care providers for all drugs and for opioids to the county-year level. On average 25,000 opioid prescriptions were dispensed per 100,000 inhabitants and 403,000 non-opioid prescriptions.

To receive an estimate of the strength of addiction-help networks in every county, we collect county-level information on the number of substance abuse treatment centers from the U.S. Census Bureau's County Business Patterns (CBP). The latest available information is the amount of out- and inpatient treatment centers in every county in 2014. In 2014, there were, on average, 5.6 treatment centers per 100,000 inhabitants in every county.

The information whether a state had a mandatory-access prescription drug monitoring program in place in 2013 is taken from Buchmueller and Carey (2018). 20% of the counties in our study introduced this legislation before 2014.

Finally, we collect data on county characteristics that could correlate with suicide rates and opioid painkiller availability from different data sources. Summary statistics are depicted in the lower panel of Table 2.1. Medicare Part D Prescription Drug Plan enrollment data for 2013-2015 is provided by the CMS. The Bureau of Labor Statistics produces unemployment rates and industry employment shares at the county level for the years 2013-2015. We classify counties into two categories of urbanization (urban/rural) according to the NCHS Urban-Rural Classification Scheme for Counties 2013 (Ingram and Franco, 2012). The U.S. Census Bureau provides in their "Small Area Income and Poverty Estimates (SAIPE) Program" estimates on county poverty rates and median household income levels for the years 2013-2015. Divorce data for 2010 comes from the National Center for Family & Marriage Research with the Center for Family and Demographic Research (Compass, 2016). The divorce rate is calculated by the ratio of the number of di-

vorced people in a county and the married population plus the number of divorced people.

2.4 Empirical Analysis

We are interested in the relationship between opioid promotion and non-poisoning suicide rates. We measure promotion with the number of physicians that receive promotion for opioid drugs. We first show that cross-sectional variation in promotion can explain differences in suicide death rates. For this, we estimate the following OLS regression:

$$SuicideRate_c = \alpha_s + \beta^{OLS} OpioidProm_c + X_c^{'}\Gamma + \varepsilon_c \tag{2.1}$$

where $SuicideRate_c$ denotes non-poisoning suicide rates in county c. Pharmaceutical promotion of opioid painkillers is denoted as $OpioidProm_c$ and state fixed-effects with α_s. The vector $X_c^{'}$ is included to control for time-varying socioeconomic conditions such as Medicare Part D Prescription Drug Plan enrollment rates, poverty rates, and labor market conditions. The error term is captured by ε_c. Dependent and independent variables are adjusted by population and in log.

It is possible that the OLS estimates are biased due to omitted variables. One possibility is that pharmaceutical companies are targeting counties with a high prevalence of mental illnesses or pain to promote opioid drugs. To identify the causal effect of opioid promotion on non-poisoning suicide rates, we estimate the following IV regressions. We use the number of doctors that receive promotion for drugs that are unrelated to mental illnesses or pain. The idea is that physicians receive opioid promotion simply because the sales representatives are also promoting these unrelated drugs. This should effect non-poisoning suicide rates only through opioid promotion. The first stage regression estimated is described by Equation 2.2.

$$OpioidProm_c = \phi + \rho UnrelatedProm_c + X_c^{'}\Psi + \mu_c \tag{2.2}$$

$UnrelatedProm_c$ denotes the number of physicians receiving pharmaceutical

promotion for drugs unrelated to mental illnesses and pain. Our hypothesis is that more doctors receive opioid promotion in counties where many doctors receive promotion for unrelated drugs. Therefore, we expect ρ to be positive.

We estimate the following second stage:

$$SuicideRate_c = \alpha + \beta^{IV} \widehat{OpioidProm}_c + X_c'\Gamma + \varepsilon_c \tag{2.3}$$

where $\widehat{OpioidProm}_c$ is the prediction from the first-stage (Equation 2.2). The coefficient of interest is β^{IV}, which captures the effect of pharmaceutical promotion of opioids on non-poisoning suicide death rates. Our hypothesis is that increasing opioid promotion raises the availability of opioid drugs and therefore suicide rates.

2.5 Results

2.5.1 Opioid Promotion and Suicide Rates

Counties where many doctors receive promotion for opioid drugs have higher non-poisoning suicide rates. Table 2.2 shows estimation results from the OLS regressions depicted in Equation 2.1. Increasing the number of physicians that receive pharmaceutical promotion for opioid drugs by 1% increases the number of people committing non-poisoning suicide by 0.07%. The regressions estimated in columns (1)-(3) differ by whether state and year fixed effects and whether county characteristics are included. The coefficient estimate does not vary substantially between these three specifications. The county characteristics that we include in column (3) are shown or argued to be important determinants of suicide rates. For example, unemployment and divorce rates at the county level are positively correlated with suicide rates. The remaining characteristics we control for are poverty rates, the logged median income, industry shares, whether the county is rural or not, the enrollment rates in the Medicare Part D Prescription Drug Plan and population. Many of these variables are potentially endogenous regressors. We include these county characteristics to show that the positive relationship between opioid promotion and suicide rates survives the inclusion of county characteristics that

could correlate with opioid promotion and suicide rates.

The positive relationship we observe in Table 2.2 could be driven by unobservable county characteristics. We thus turn to the results of our IV regressions. Column (1) in Table 2.3 shows the estimation results of Equations 2.2 and 2.3. The first stage results indicate that the instrument works in the expected direction: the more physicians receive promotion for unrelated drugs, the more physicians receive promotion for opioid drugs. The partial F-statistic of the first stage shows high instrument relevance. A list of the unrelated drugs used as an instrument can be found in Table A-12. The coefficient estimate from our IV regression is indistinguishable from the OLS coefficients depicted in column (2). Again, the results indicate that increasing the number of doctors receiving promotion for opioid drugs by 1% increases suicide rates committed with non-poisoning means by 0.07%. This implies that the promotion of opioid drugs explains approximately 2% in the variation of non-poisoning suicide rates. The coefficient estimate is about 1/3 of the estimate found by Fernandez and Zejcirovic (2017) of opioid promotion on opioid-related overdose death rates. Column (3) displays the reduced form estimates of regressing suicide rates on our instrument. In line with the hypothesis, there is a positive relationship between the number of doctors receiving promotion for drugs unrelated to opioid need and suicide rates. We can see in column (4) that this positive relationship disappears once we include opioid promotion. This suggests that the positive association we observe in the reduced form operates through opioid promotion. This also rules out the concern that our instrument is capturing counties with high medication demand in general and high mental illness prevalence.

For opioid promotion to physicians to affect suicide rates, it must increase the number of prescriptions dispensed. Fernandez and Zejcirovic (2017) show that Medicare Part D physicians receiving opioid promotion have higher opioid prescription rates. In this subsection, we show a positive association between the number of prescriptions written for Medicare Part D patients and suicide rates. Around 18% of the population in the US are enrolled in the Medicare Part D program, of which 50M people are eligible due to their age and 9M people due to disabilities. The count of prescriptions we have at the county-year level is thus not the universe of opioid prescriptions but represents a substantial share.

Columns (4) to (6) of Table 2.2 depict the estimation results of Equation 2.1 where the independent variable is the number of opioid prescriptions for Medicare Part D patients. We find a positive and statistically significant relationship between prescription availability and non-poisoning suicide rates in all three specifications. The most restrictive specification in column (6) implies an elasticity of 0.05 between prescriptions and suicides. These results are indicative that opioid availability is positively associated with suicide rates not involving means of poisoning.

2.5.2 Heterogeneity by Presence of Substance Abuse Treatment Centers

There are different channels through which opioid painkiller availability can increase suicide rates. The first one is that it increases the means of committing suicide. In our analysis, we are excluding all deaths that involved any form of poisoning. Including these deaths increases the coefficient estimates which indicates that the availability of more means, indeed, is an important determinant of suicide rates.[6] The fact that opioid promotion also influences the number of suicides committed without poisoning suggests that opioid use has a direct effect on suicide rates. There is no clear consensus in the medical literature on why there are positive correlations between opioid use and suicidal intents. Possible explanations are that opioid use lowers the hesitance to act on the impulses of suicidal thoughts or that opioid use induces hyperalgesia, i.e., pain sensitivity (Ilgen et al., 2016). Another argument is that it increases the number of people with substance abuse disorder. We do not have county estimates of substance abuse disorders prevalence. We can shed some light on the mechanism by exploring the role of substance abuse treatment centers. If the prevalence of substance abuse disorder is driving increases in suicide rates, we expect that treatment possibilities for substance abuse disorders to mitigate the effect of increasing opioid painkiller availability on suicide rates. We use the number of substance abuse treatment centers and split the sample into counties with many centers in 2014 and counties with few centers (below and above the median of 4 treatment centers per 100,000

[6] Results not included in this article but are available upon request.

inhabitants). Table 2.4 shows the regression results of Equation 2.1 for the entire sample and counties with a high and low number of treatment centers in columns (1) to (3). The coefficient of opioid promotion on suicide rates is much smaller and less precisely estimated in counties with strong addiction-help networks. The coefficient has the magnitude 0.02 and is statistically significant at the 10% level. The elasticity of opioid promotion and suicide rates is four times larger in counties with few treatment centers for substance abuse. The difference in coefficients is statistically significantly different at conventional levels and large in magnitude. When we use the IV strategy, we again receive very similar coefficient estimates but less precisely estimated as can be seen in Panel B of Table 2.4. Importantly, the number of substance abuse treatment centers is not exogenously distributed across counties with respect to suicide rates. The results suggest that strong addiction-help networks can help mitigate the effects of opioid promotion on suicide rates, but we cannot claim causality.

2.5.3 Heterogeneity by Presence of "Must Access" PDMPs

Recent literature argues that inappropriate prescription patterns lie at the heart of the epidemic, resulting from knowledge deficits and (wrongly) perceived safety of opioid drugs (Manchikanti et al., 2012). As a reaction to drug diversion and increasing cases of addiction and overdose, all states but one introduced Prescription Drug Monitoring Programs (PDMP).[7] Prescriptions for controlled substance drugs are collected in a centralized database informing about the behavior of patients and health care providers. Health care providers can check whether their patients received prescriptions for controlled substances from another provider. Law enforcement in some states can also access the database to investigate drug diversion. Previous studies establish the effectiveness of PDMPs in reducing the total volume of opioids dispensed and misuse of opioid drugs in Medicare Part D patients (Moyo et al., 2017; Buchmueller and Carey, 2018). Buchmueller and Carey (2018) highlight the importance of heterogeneity in PDMPS legislations. They find that PDMPs reduce misuse when health care providers *must* access the database while they do not find a reduction for programs without such provisions.

[7] Missouri is the only state that has not passed a PDMP by 2018.

Borgschulte et al. (2018) find that PDMPs reduce suicide rates in counties with strong addiction-help networks, with a larger reduction in states with a mandatory access provision.

The presence of mandatory access PDMPs may influence how opioid promotion affect non-poisoning suicide rates. It directly affects the incentive of the physician to prescribe a controlled substance now that these prescriptions are collected in a centralized database. As the physician now has more information on a patient's potential addiction, it also reduces the probability that the new opioid prescriptions are misused.

Table 2.4 shows that there is no relationship between opioid promotion and non-poisoning suicide rates in counties with a "Must Access" PDMP in place. The comparison counties either have no PDMP in place or no mandatory access provision (column (5)). The difference in coefficients is large in magnitude and statistically significantly different at conventional levels. The positive relationship between suicide rates in promotion is entirely driven by counties with no "Must Access" PDMP. We obtain very comparable results instrumenting opioid promotion with promotion of unrelated drugs (see Panel B of Table 2.4).

The availability of Medicare Part D opioid prescriptions at the county level allows us to investigate whether physicians indeed react less strongly to opioid promotion in states with mandatory access PDMPs. We regress the number of prescriptions dispensed for Medicare Part D patients on the number of doctors receiving promotion. The results are displayed in Table 2.5. Increasing the number of physicians that receive promotion by 1% increases the number of prescriptions dispensed by 0.3%. The elasticities are 50% smaller in counties with "Must Access" PDMPs in place as can be seen from comparing column (2) and (3). The difference is statistically significant at conventional levels. Physicians in these states do react to promotion but with smaller elasticities and it appears that in these states non-poisoning suicide rates are not influenced by promotion of opioid drugs.

2.6 Conclusion

Suicides are a major public health concern and understanding the drivers in suicide rates is crucial to derive prevention policies. We show that pharmaceutical promotion of opioid drugs is positively associated with non-poisoning suicide rates. Pharmaceutical promotion increases the number of opioid prescriptions dispensed, which can raise the number of individuals with substance abuse disorders. Substance abuse disorders are high risk factors for suicidal intent. In line with these arguments, we find that the positive relationship is mitigated in counties with a high number of substance abuse treatment centers. In addition, we find that there is no positive association between promotion of opioid drugs and suicide rates in counties with a mandatory PDMP in place. Doctors in these counties react with smaller elasticities towards opioid promotion in terms of opioid prescriptions. Our results suggest that the negative effects of promotion opioid drugs can be avoided if the right patients receive opioid prescriptions or if people with substance abuse disorder have a path out of addiction.

Pharmaceutical promotion to physicians can influence their prescription behavior (Datta and Dave, 2017; Fernandez and Zejcirovic, 2017). There is evidence for knowledge deficits among some physicians in the US regarding the properties of opioid painkillers (Manchikanti et al., 2012). This study highlights one of the aspects of the high potential social costs associated with underestimating the risk of addiction: an increasing number of suicides.

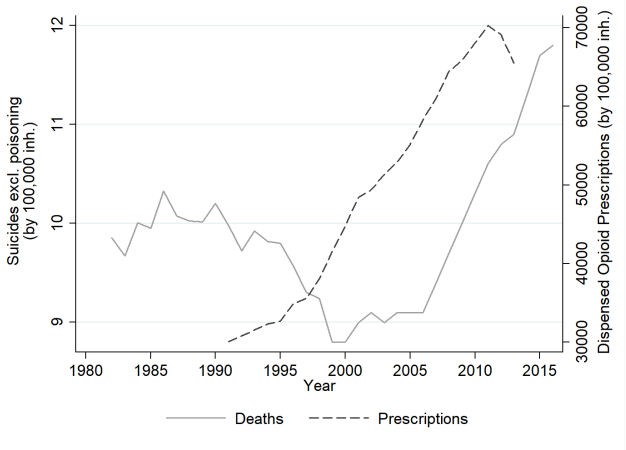
It is important to note that the heterogeneity analyses we perform, rely on the assumption that the considered policies, e.g., PDMPs, and the presence of substance abuse treatment centers, are exogenous regressors. It is possible that opioid promotion influences legislators' decision to pass or fund any of these programs/legislation.

Another drawback of our analysis is that we cannot show an increase in the number of individuals with substance abuse disorder empirically, but we can only provide indirect evidence using the presence of substance abuse treatment centers. More research is needed on understanding the relationship between opioid abuse and suicide rates, complementing the medical research that shows a positive correlation between opioid abuse and suicidal intent (Ashrafioun et al., 2017; Ilgen

et al., 2016), even controlling for comorbid psychiatric and physical conditions.

Tables and Figures

Figure 2.1: Suicide crude rates (1982-2016) and opioid analgesic prescriptions dispensed from US retail pharmacies (1991-2014).



Source: CDC Wonder Mortality MCD Data and IMS Health National Prescription Audit

Table 2.1: Summary statistics

	Observations	Mean	Median	SD	Min	Max
Deaths, prescriptions, promotion						
Suicide rates	8765	16.85	14.88	11.65	0	180.87
Suicide rates (excl. poisoning)	8765	14.81	12.99	10.88	0	158.26
Opioid prescription Medicare Part D	9118	25298	21999	19601	0	505408
Non-opioid prescriptions Medicare Part D	9118	402822	369043	262187	0	5588897
Doctors receiving opioid promotion	6302	6.38	1.44	10.45	0	173.65
Doctors receiving unrelated drug promotion	6302	93.98	69.57	105.50	0	2155.27
State laws & treatment centers						
Must access PDMP (D=1)	9372	0.20	0	0.40	0	1
Drug abuse treatment centers	9425	5.58	3.90	7.25	0	107.58
County characteristics						
Rural (D=1)	9453	0.42	0.00	0.49	0.00	1.00
Medicare Part D enrollment	9406	0.11	0.11	0.04	0.01	0.27
Median income (log)	9422	10.73	10.72	0.24	9.98	11.74
Share living below poverty	9422	16.78	15.80	6.51	3.00	55.10
Population	9425	101475	25716	325914	86	10170292
Unemployment rate	9417	0.06	0.06	0.02	0.01	0.26
Divorce rate 2010	9288	0.36	0.37	0.11	0.00	1.00
Industry shares						
Natural resources & mining	9424	0.07	0.03	0.10	0.00	1.00
Construction	9424	0.06	0.05	0.04	0.00	0.75
Manufacturing	9424	0.14	0.12	0.12	0.00	0.78
Trade, transportation, & utilities	9424	0.26	0.25	0.09	0.00	1.00
Information	9424	0.01	0.01	0.01	0.00	0.16
Financial activities	9424	0.05	0.04	0.03	0.00	1.00
Professional & business services	9424	0.08	0.07	0.06	0.00	0.94
Education & health services	9424	0.17	0.16	0.08	0.00	0.82
Leisure & hospitality	9424	0.13	0.11	0.08	0.00	0.94
Other services	9424	0.03	0.03	0.02	0.00	0.56
Unclassified	9424	0.00	0.00	0.00	0.00	0.08

Unit of analysis: county-year. Study period: 2013-2015. All numbers in upper panel adjusted by population (100,000). Source: CDC Wonder MCD Data, CMS Open Payment, US Census Bureau County Business Patterns.

Table 2.2: OLS: opioid promotion and suicides, excl. poisonings

	(1)	(2)	(3)	(4)	(5)	(6)
Dep. var.:	Suicides excl. poisonings	Suicides excl. poisonings	Suicides excl. poisonings	Suicides excl. poisonings	Suicides excl. poisonings	Suicides excl. poisonings
Doc. receiving opioid promotion	0.0683***	0.0743***	0.0611***			
	(0.00969)	(0.0103)	(0.0115)			
Opioid prescriptions				0.0608***	0.0563***	0.0514***
				(0.0122)	(0.0128)	(0.0133)
Mean dep. var.	2.482	2.480	2.485	2.465	2.465	2.467
SD dep. var	0.925	0.925	0.919 .	0.922	0.922	0.918
Observations	5844	5825	5790	8644	8644	8590
R2	0.00890	0.0677	0.0850	0.00623	0.0595	0.0815
Year F.E.	N	Y	Y	N	Y	Y
State F.E.	N	Y	Y	N	Y	Y
County characteristics	N	N	Y	N	N	Y

Suicide rates, opioid promotion, and opioid prescriptions normalized by county population. Dependent and independent variable in log. Estimation results of Equation 2.1. County characteristics: unemployment rate, log median income, poverty rate, population, industry shares, share of population enrolled in Medicare Prescription Drug Plan, dummy urban/rural, divorce rate in 2010. Standard errors in parentheses adjusted for heteroscedasticity, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: CDC Wonder Mortality Data, CMS Open Payments Data 2014, 2015, and Medicare Provider Utilization Data 2013, 2014, and 2015.

Table 2.3: IV: opioid promotion and suicides, excl. poisonings

	(1)	(2)	(3)	(4)
Method	IV	OLS	OLS reduced form	OLS
Dep. var.:	Suicides excl. poisonings	Suicides excl. poisonings	Suicides excl. poisonings	Suicides excl. poisonings
Doc. receiving promotion for opioid drugs	0.0684***	0.0697***		0.0704***
	(0.0209)	(0.00975)		(0.0127)
Doc. receiving promotion unrelated drugs			0.0302***	-0.000889
			(0.00926)	(0.0118)
Mean dep. var.	2.482	2.482	2.482	2.482
SD dep. var.	0.925	0.925	0.925	0.925
Observations	5844	5844	5844	5844
R2	0.00950	0.00950	0.00319	0.00950
First stage				
ρ Doc. receiving promotion unrelated drugs	0.411***			
	(0.00574)			
Partial F-Statistic	3980.5	.	.	.

Suicide rates and opioid promotion normalized by county population. Dependent and independent variable in log. Partial F-Statistic of first stage displayed in last row. Column (1) shows estimation results of Equations 2.2 and 2.3, while columns (2)-(4) show estimation results of Equation 2.1. Standard errors in parentheses adjusted for heteroscedasticity, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: CDC Wonder Mortality Data and CMS Open Payments Data 2014, 2015.

Table 2.4: Heterogeneity by presence of substance abuse treatment centers and "Must Access" PDMP

Counties:	(1) All	(2) High number treatment centers ($>$ 4 per 100,000 inh)	(3) Low number treatment centers ($\leq$ 4 per 100,000 inh)	(4) Must access PDMP	(5) No must access PDMP
Dep. var.:	Suicides excl. poisonings	Suicides excl. poisonings	Suicides excl. poisonings	Suicides excl. poisonings	Suicides excl. poisonings
			Panel A: OLS estimates		
Doc. receiving promotion for opioid drugs	0.0697***	0.0216*	0.0872***	0.0225	0.0797***
	(0.00975)	(0.0122)	(0.0162)	(0.0191)	(0.0112)
			Panel B: IV estimates		
Doc. receiving promotion opioid drugs	0.0684***	0.0227	0.0724*	0.0165	0.0737***
	(0.0209)	(0.0193)	(0.0425)	(0.0491)	(0.0235)
Mean dep. var.	2.482	2.556	2.415	2.607	2.448
SD dep. var.	0.925	0.591	1.138	0.791	0.953
Observations	5844	2750	3094	1201	4624
R2 (Panel A)	0.009	0.003	0.008	0.003	0.012
Partial F-Statistic (Panel B)	3980.5	1716.1	1053.0	567.5	3204.6

Suicide rates, number of treatment centers, and opioid promotion normalized by county population. Dependent and independent variables in log. Estimation results of Equation 2.1 in Panel A, estimation results of Equations 2.2 and 2.3 in Panel B. Partial F-Statistic of first stage displayed in last row. Number of substance abuse (inpatient and outpatient) centers in 2014. Sample split whether above or below median value of number of treatment centers in columns (2) and (3). Sample split by whether county belongs to a state with a mandatory access prescription drug monitoring program in place in 2013 in columns (4) and (5). Standard errors in parentheses adjusted for heteroscedasticity, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: CDC Wonder Mortality Data, CMS Open Payments Data 2014, 2015, and US Census Bureau County Business Patterns. PDMP presence from Buchmueller and Carey (2018).

Table 2.5: Opioid promotion and prescription rates: heterogeneity by "Must Access" PDMPs

	(1)	(2)	(3)
Method:	OLS	OLS	OLS
Counties:	All	Must access PDMP	No must access PDMP
Dep. var.:	Opioid prescriptions Medicare Part D	Opioid prescriptions Medicare Part D	Opioid prescriptions Medicare Part D
Doc. receiving promotion for opioid drugs	0.305***	0.217***	0.321***
	(0.0116)	(0.0167)	(0.0136)
Mean dep. Var.	9.808	10.04	9.749
SD dep. Var.	1.248	0.761	1.336
Observations	6075	1218	4857
R2	0.0978	0.129	0.0945

Opioid prescription and promotion normalized by county population. Dependent and independent variables in log. Standard errors in parentheses adjusted for heteroscedasticity, * ($p<0.10$), ** ($p<0.05$), *** ($p<0.01$). Source: Medicare Provider Utilization Data 2013, 2014, and 2015, PDMP presence from Buchmueller and Carey (2018), CMS Open Payments Data 2014, 2015.

Chapter 3

WAR AND POLITICAL PARTICIPATION IN BOSNIA AND HERZEGOVINA

Joint with Caterina Alacevich (UPF)

3.1 Introduction

Formal institutions and informal norms such as civic capital and trust between market participants contribute crucially to economic development (Guiso et al., 2011; Algan and Cahuc, 2010). The establishment of institutions and democratic participation in the aftermath of a civil war is vital for the reconstruction of war-torn economies and societies. In particular, elections can be decisive for the achievement of peaceful resolutions of political controversies (Korth, 2011). In ethnically divided communities, institutions play an essential role in restraining competing groups from further social unrest (Montalvo and Reynal-Querol, 2005).

A growing literature shows that the experience of violence and war-related aggression may alter social capital, trust, preferences for market participation, and political preferences (Voors et al., 2012; Rohner et al., 2013; Cassar et al., 2013). Political participation and institutional representation ultimately determine the al-

location of public and private resources (Miguel and Roland, 2011). It is therefore important to understand how voting behavior differs for individuals who were more affected by civil war than others. Does the intensity of civil conflict lead to differential political participation among victims?

The direction in which exposure to violence may affect voter turnout through a change in behavior and preferences is ambiguous a priori. Within a growing literature that studies the consequences of conflict and exposure to violence on political and social behavior, there is mixed evidence. War may foster cooperation among neighbors to organize common defense, attack, and coping strategies. Bellows and Miguel (2009) find that conflict in Sierra Leone fostered political involvement and engagement in community meetings. The authors argue that increased participation is triggered by a change in behavior, in response to external aggressions. Blattman (2009) shows increased political engagement among former abducted combatants in Uganda, in line with experiences of post-traumatic psychological growth. Voors et al. (2012) estimate higher levels of in-group altruism among individuals in Burundi whose villages directly experienced high levels of violence. In contrast, Cassar et al. (2013) argue that having experienced violence during the Tajik civil war led to decreased levels of trust and preferences for market participation. The civil war in Tajikistan was characterized by intra-group fighting. Civil war victimization may thus lead to reduced society-wide trust if individuals were attacked by someone with whom they previously lived together and interacted peacefully.

This paper contributes to the literature by showing that violence against civilians led to a negative and persistent impact on voter turnout in post-war democratic elections when social interactions and political institutions are defined along "out-group" divisions. Using data from local and central elections in Bosnia and Herzegovina's (BiH) municipalities and Bosnian War casualties, we estimate that a one standard deviation increase in civilian casualties, 1.24% of pre-war population, decreases turnout by up to 4.6 percentage points. With an average turnout of 55-60% and casualties reaching up to 7%, the effect is large in magnitude and statistically significant at conventional levels for every election in our dataset. The impact persists over twenty years after the conflict resolution, up until the latest

general elections of 2014.[1]

Relying on a database recording war casualties by municipality of origin and electoral statistics, we base our estimates on objective measures of conflict exposure and voting. The use of objective data minimizes measurement errors and overcomes the concern of self-reporting bias which characterizes some of the existing studies based on survey data.[2]

Most existing empirical analyses rely on the total number of casualties and do not consider the identity of the victims. Our study introduces a crucial distinction between civilian and military casualties and shows that one can reach different conclusions on the relationship of war exposure and voting behavior depending on how war intensity is measured. The literature suggests that the experience of kinship-targeted violence can have stronger traumatic effects (Kalyvas et al., 2006). Azam and Hoeffler (2002) assert that perpetrators can intentionally use violence against civilians with the strategic purpose of undermining the sense of society. Rohner et al. (2013) find that the negative impact of violence exposure in Uganda on social capital is larger when they consider violence committed against civilians. Consistent with this hypothesis, we find that social and political apathy arose specifically from violence towards civilians. In contrast, higher exposure in terms of military casualties had no statistically significant relationship with post-war turnout rates nor social capital measures.

The literature shows that war and conflict can have extensive consequences on societies and economies (Acemoglu et al., 2011), including the depletion of human capital (Akresh and de Walque, 2008; Leon, 2012), demographic shifts and ethnic homogenization (Swee, 2015), and changes in social, risk, and time preferences (Voors et al., 2012; Miguel and Roland, 2011; Bellows and Miguel, 2009; Blattman, 2009). To address different potential conflict outcomes that may correlate with political participation and mediate the effect of war violence, we add demographic, social, political, and economic indicators to our estimations. We show that the relationship between war casualties and voter turnout is robust to alterna-

[1] This persistence is in line with the findings of Bauer et al. (2016) who – pooling several studies with different time horizons– show that the effect of violence exposure on social capital does not seem to dissipate over time (similarly, see Cassar et al. (2013) and Grosjean (2014))

[2] Dellavigna et al. (2017) find that individuals who voted tend to tell the truth, while non-voters have a higher propensity to lie about their electoral behavior.

tive explanations of forced migration, demographic selection, ethnic composition, labor market and economic activity, human capital, physical capital damage, and post-conflict reconstruction. The results support the hypothesis that exposure to war affects citizen behavior in a direct way. To shed more light on this channel, we then use a nationally representative individual-level survey on social and political preferences (EBRD, 2006). The survey allows us to follow the same empirical strategy developed at the municipality-level, controlling for recalled trust and political engagement before the War. We find that respondents in municipalities exposed to intensive violence towards civilians report a lower propensity to vote, to trust other people, and to trust political and economic institutions such as the presidency, the cabinet of ministers, the parliament, political parties, the court, military forces, the police, and the financial system.[3] The analysis suggests that the effect of exposure to violence towards civilians on voting is channeled by a depletion of social preferences, undermining trust in institutions and interpersonal ties.

In a recent meta-analysis on conflict and cooperation, Bauer et al. (2016) conclude that individuals experiencing violence generally develop pro-social preferences toward "in-group" members, while there is not much evidence on preferences towards "out-group" members. Before the outbreak of the war, BiH was characterized by a substantial ethnic fractionalization, with Serbs, Croats, and Bosniaks living together and with numerous multi-ethnic households. The ethnic nature of the Bosnian civil conflict determined clear "out-group" divisions.[4] Whitt (2010) shows that Bosnians have low levels of generalized trust, and more than 65% of survey respondents mistrust members of opposed ethnicity as well as co-ethnics.[5] Analogously, ethnic divisions play a major role in the political debate. Parties define their core policies along co-ethnic favoritism and war-time divides are thus still salient in political agendas. Our findings of decreased voter

[3] Alike in the municipality-level analysis, we show that there is no evidence of selected targeting of individuals based on their pre-war levels of social capital, nor do results differ for younger respondents that were less likely to be selectively attacked.

[4] During the War, there were also instances of intra-group fighting among ethnic affiliates (Christia, 2008), and several victims knew their aggressors in person (Kalyvas and Sambanis, 2005).

[5] In another study, he shows that levels of fairness elicited with behavioral games do not display differences across ethnicities for the majority of the respondents (Whitt, 2014).

turnout and lower generalized trust in high-violence areas, therefore, reconcile with the existing literature because social and political interactions encompass all individuals, including different ethnic groups and former war opponents.

An estimation challenge arises from the fact that conflict intensity is unlikely to be exogenously distributed with respect to municipality characteristics that correlate with social capital measures. For example, communities with higher civic capital may be more efficient in organizing defense and attack. Additionally, the consequences of war extend to a broad range of economic, demographic, social, and political outcomes that may correlate with political behavior and cause omitted variable bias. We explore the possibility of a selective targeting towards less politically engaged municipalities by correlating pre-war voter turnout and war casualties. We find the opposite scenario of a positive, rather than a negative, association between casualties and pre-war turnout. To tackle endogeneity concerns, we adopt several empirical strategies. One advantage of the Bosnian context is that the country had its first free and democratic elections before the outbreak of the war. This allows us to follow a difference-in-differences strategy. Similarly to Rohner et al. (2013), we take the first difference between post-war and pre-war turnout rates and regress it on the intensity of violence towards civilians from the same municipality of origin. Another advantage is that in BiH, the same parties that emerged shortly before the war still define the political scene nowadays. This is a crucial feature that makes pre and post-conflict political participation comparable.[6] The absence of multiple democratic elections before the War, however, impedes a usual test for pre-conflict parallel trends. For this reason, the results need to be interpreted with caution. Next, we thus additionally assess the validity of our findings through alternative empirical specifications and other measures of war exposure. We show that our estimates are robust to the inclusion of pre and post-war geographical, demographic, and economic municipality characteristics that the literature identifies as important determinants of conflict risk (Novta, 2013; Weidmann, 2011; Kalyvas and Sambanis, 2005). Similarly to Bel-

[6]Most existing analyses of war exposure and political participation rarely discuss the "supply side" of politics. The stability of the political scenario determines whether past elections can be an appropriate control. Understanding who the electorate can vote for after the resolution of a conflict helps to shed light on the opposing effects found in the literature. If parties emphasize war-time divides in their policies, victims of the war may become more politically apathetic.

lows and Miguel (2009), we investigate heterogeneous effects for voters who were too young to be directly targeted in the war, compared to older cohorts. We do not find any statistically significant difference, ruling out the hypothesis of selective victimization. Finally, we instrument variation in conflict intensity with measures of terrain ruggedness. The literature suggests that mountainous environments are conducive to conflict risk because they facilitate sheltering and attack in the adjacent valleys (Kalyvas and Sambanis, 2005). All our estimation specifications confirm that conflict exposure decreases voter turnout.

This study contributes to the existing literature in several ways. It suggests that research on the impact of violence on political participation needs to carefully address i) the measure of war exposure, (ii) the in-group versus out-group nature of formal institutions and informal norms, and (iii) the "supply-side" of policies and political parties. Contrasting results in the existing literature could be attributed to different definitions of war intensity and changes in the political landscape. Our results of decreased voter turnout conciliate with and complement the existing evidence of lower trust toward out-group community members (Cassar et al., 2013). Because in this context political and social participation imply "out-group" division, our findings also complement and reconcile with opposite results of increased in-group 'parochial' trust and civic engagement found by a number of recent micro-level studies (Bauer et al., 2016).

The paper is structured as follows. Section 3.2 describes the historical and institutional background of our analysis. Section 3.3 summarizes the sources of data at the municipality level and the individual-level survey, and their descriptive statistics. Section 3.4 presents the empirical strategy for the municipality-level analysis, followed by the estimation results (Section 3.5). Section 3.5.2 explores the potential channels, and Section 3.5.3 elaborates the analysis of conflict exposure and individual levels of generalized trust, trust in institutions and political participation. Section 3.5.4 reports robustness checks and Section 3.6 concludes.

3.2 Historical Background and Institutional Setting

The Bosnian War originated with the breakup of the Socialist Federal Republic of Yugoslavia (SFRY). Former Member of the SFRY, BiH declared its sovereignty in

October 1991, after Slovenia and Croatia. At that time, BiH had a total population of 4.3 million and included several ethnic groups such as Muslim Bosniaks (44 percent), Orthodox Serbs (31 percent), Catholic Croats (17 percent), and other minorities.

On February 29th of 1992, Bosnia was asked to express its opinion regarding independence from the SFRY. Political representatives of the Bosnian Serbs boycotted the referendum and responded with the mobilization of armed forces. A majority of Croats and Bosnian Muslims voted for independence and obtained recognition by the international community. On the contrary, Bosnian Serbs wanted to keep the annexation with Yugoslavia, to unify Serbian-majority territories, and to form an ethnically homogeneous "Greater Serbia". Municipalities with a Serbian majority had already declared mistrust toward Muslim leaders of Bosnia and had started forming armed municipal Crisis Staffs late in 1991. The Yugoslav Army, under the guidance of Milosevic, transferred Serbian soldiers to local units in Bosnia. Initial tensions quickly escalated into an armed conflict and into a brutal ethnic civil war that lasted four years. The war was fought along ethnic and territorial control lines.

The conflict ended in 1995 with the negotiation of the Dayton Peace Agreement between representatives of all Bosnian parties and the neighboring Federal Republic of Yugoslavia and Republic of Croatia. As established in the Agreement by all parties, Bosnia and Herzegovina (BiH) was then proclaimed a federal democratic republic composed by two main entities, the Federation of Bosnia and Herzegovina (FBiH) and the Republika Srpska (RS), and, since March 2000, the Brcko District.[7] An Inter-Entity Boundary Line separated the three units. The Agreement mandated that internal administrative units will be equally governed by all groups (Bosniaks, Croats, and Serbs). FBiH was further divided into ten cantons and 79 municipalities, and RS into 63 municipalities.

The political landscape is thus characterized by a three-members rotating presidency, elected by popular vote within the three major ethnic groups (Bosniak, Croat, and Serb). The national government has responsibilities limited to security and defense, borders and immigration, fiscal and monetary policy, and inter-entity

[7] Brcko is a self-governing neutral administrative entity under the sovereignty of BiH, internationally supervised.

coordination and regulation. The confederate sub-entities have a large degree of autonomy. They are in charge of public goods provision, social services, education, housing, and health care. Municipal councils are in charge of local public management and services delivery.

The first elections after the war (1996-97) were supervised and monitored by the Organization for Security and Co-operation in Europe (OSCE). Despite OSCE's auditing activity, they were characterized by irregularities, frauds and harassment, both during the registration and in the voting process (Pugh and Cobble, 2001). The elections in 2002 can be considered as the transition to an autonomous self-administration: international authorities still had a prominent role, but the Bosnian institutions officially took responsibility (OSCE, 2002). Our empirical analysis starts from the elections of 2004. In 2004, the government had full responsibility for the elections, for the first time, without the supervision of the OSCE.

A crucial analytical advantage of the Bosnian context is the stability of the political landscape. The main political parties and subjects that ran in the pre-conflict elections of 1990 still participated in all post-war elections. This makes the pre and post-war electoral scenario comparable. Table B-6 in Appendix displays the names and evolution of political parties over time.

3.3 Data and Descriptive Statistics

We combine several sources of data, following the general criterion of favoring objective measures over self-reported information.

To obtain objective measures of war intensity by municipality, we use the "Bosnian Book of the Dead" (BBD), a registry compiled by the Research and Documentation Center in Sarajevo (2008). The Book reports the number of war casualties (killed and missing individuals) by municipality, distinguishing between military members and civilians. Ball et al. (2007) provide an assessment of the database. We use civilian casualties by municipality of origin as this measures violence towards members of own communities who did not take an active part in the armed fighting. For the International Criminal Tribunal of former Yugoslavia (ICTY), victims are considered civilians unless it could be proven that the killed

person was militarily involved in the action. For each municipality (m), and for the subgroups $s = 1$ (Total), 2 (Civilian), (3) (Military), we compute the share of casualties as:

$$\text{Casualties}_{m,s} = \frac{\text{Total Casualties}_{m,s}}{\text{Pre-war Population}_{m}\ (1991)} \tag{3.1}$$

Figure 3.1 shows the geographic variation in civilian victims as a share of pre-war population by municipality of origin. Figure 3.2 displays the kernel density of war casualties in the municipality of origin, distinguishing between military and civilian fatalities. Table 3.1 reports the summary statistics for war casualties, the number and share of damaged houses (a measure of physical capital destruction), and war-related internal displacement in 1995, by municipality. Our final estimation sample excludes municipalities with missing information and excludes Mostar for its outlying post-war administrative partition pattern, the independent district of Brcko for its non-comparable administrative and political regime, and Srebrenica for its outlying number of civilian victims. The total number of victims is 97,207. Civilian casualties reach a maximum of 6.57% of the pre-war population, with a mean of 0.83% and a standard deviation of 1.24%. Military casualties were higher in absolute numbers but with less dispersion. The average number of houses damaged is 8 in 1000 inhabitants, of which an average of 49% get reconstructed by 2005.[8]

A second source of data, for voter turnout at the municipality level, is given by official electoral statistics from the Federal Office of Statistics of BiH (FOS-BiH) and the Central Election Commission of BiH for the local elections that took place in 1990, 2004, 2008, and 2012, and general elections of 2006, 2010, and 2014 in 149 municipalities.[9] We compute voter turnout as the share of people that voted over all registered voters by municipality.

An additional source is Bosnia and Herzegovina's 1991 census, conducted just before the war. The census provides pre-conflict information such as ethnic composition and economic indicators at the municipality level. To control for

[8]The maximum number of repaired houses exceeds 100% in some instances as more houses were built by the UNHCR than houses were reported to have been destroyed by 1995.

[9]We thank Borisa Mraovic for providing access to 1990 turnout data from Mraović (2014).

post-war ethnic homogenization, we use the 2013 census, released in June 2016 (Statistical Agency of Bosnia and Herzegovina, 2016). Following Montalvo and Reynal-Querol (2005), we compute the ethnic polarization index according to the following formula:

$$P = 4 \sum_{i=1}^{N} \pi_i^2 (1 - \pi). \tag{3.2}$$

The index illustrates the distance between the distribution of the ethnic groups from a situation of highest polarization, characterized by the bipolar distribution.

We collected additional post-war economic and socio-demographic indicators from the database compiled by the Center for Social Research Analitika, supported by the Open Society Institute and Swiss Agency for Development and Cooperation in Bosnia and Herzegovina.[10] Specifically, we use the estimated number of inhabitants per municipality (2007-2012) and the ethnic composition from the census of 2013 (Statistical Agency of Bosnia and Herzegovina, 2016), internal immigration net rates (2007-2012), average net wages, and the share of unemployed within the total active labor force (2005-2012).

Table 3.2 displays summary statistics and turnout levels for the 127 municipalities of our sample. Bosnia had the first free and democratic elections in 1990, the only ones that preceded the conflict. Turnout levels by municipality averaged around 80% in 1990. Our sample encompasses both general and local elections. We construct our outcome variables as the difference between turnout for each post-conflict election and turnout in 1990 ("Δ Turnout" in Table 3.2).

Another feature of the conflict is that ethnic polarization decreased between 1990 (0.77 index value) and 2013 (0.43 index value). Municipalities became more ethnically homogenous after the war. Swee (2015) finds that municipalities partitioned by the Inter-Entity Boundary Line provide a higher supply of schools and teacher (per-capita). He suggests that more ethnically homogeneous municipalities are conducive to higher public goods provision due to more convergent preferences. We, therefore, account for demographic and ethnic changes in our estimations and explore the role of ethnic homogenization as a potential determinant of post-war turnout.

[10] http://www.mojemjesto.ba/en/content/about-my-place-project

Geographic characteristics, such as mountainous terrain and surface area, are taken from Costalli and Moro (2012) and Jarvis et al. (2008). We take the measure of terrain ruggedness from the Digital Elevation Data Version 4 (Jarvis et al., 2008). For every municipality, we calculate the mean and standard deviation of the ruggedness index within each municipality. The ruggedness index value is given by the change in elevation within the 3x3 pixel grid.

Lastly, we complement the analysis with individual-level information on social capital and political participation from the EBRD Life in Transition Survey I (EBRD, 2006). The survey was conducted between August and October 2006 and covered 1000 respondents in 32 municipalities, in a nationally representative design. The survey includes information on individual socio-demographic characteristics and questions on social capital preferences and behavior. Despite the limitations given by its cross-section dimension, the survey has two advantages: (i) it provides the geocoded location of the respondents' primary sampling units, and (ii) it allows to control for pre-war (self-reported) levels of generalized trust and to rule out targeting of individuals with specific social preferences. The survey asks individuals to report their current generalized trust and to recall generalized trust in 1989.[11] We use the number of civilian casualties at the municipality level as a community-wide measure of violence. Table 3.3 provides summary statistics for our final estimation sample of 697 individuals from the LIT-I survey (EBRD, 2006), estimated using survey weights, accounting for the clustered design of the sampling. Our outcomes include (i) voting in elections in 2016, (ii) generalized trust (1-5 scale), and (iii) an index of trust in institutions, computed as the average scale (1-5) between: trust in the president, the parliament, political parties, and the ministers. We normalize each index such that their distribution has a mean of 0 and a standard deviation of 1. When estimating trust, we difference the index with respect to its pre-war measure. 43% of our estimation sample reports a decrease in trust (against 54% unvaried and 3% increasing).

[11] "Generally speaking, would you say that most people can be trusted, or that you cant be too careful in dealing with people? Please answer on a scale from 1 to 5, where 1 means that you have complete distrust in people, and 5 means that most people can be trusted. What would it be today? And before 1989?" See the LIT-I questionnaire from EBRD (2006).

3.4 Empirical Strategy

We start by estimating the impact of war on voter turnout at the municipality level shortly after the conflict and up to two decades after its resolution.[12] The potential endogeneity of the conflict with respect to political and social engagement of voters constitutes the main empirical challenge. For ordinary least square estimates to be unbiased, one must assume exogeneity of conflict intensity with respect to all observed and unobservable determinants of political participation. However, it is plausible that the occurrence and intensity of war correlate both with economic, political, and social characteristics of a municipality and with the ability of its inhabitants to respond, attack, and mobilize resources in the civil conflict. Additionally, war can directly influence socio-economic conditions, such as unemployment rates and human capital accumulation. These variables are therefore potential omitted determinants that could correlate with our outcome of interest.

To overcome endogeneity concerns, our main strategy relies on geographical variation in the intensity of war-related fatalities and missing people by municipality of origin. To estimate the relationship between civil conflict and political participation at the municipality level, we follow a difference in difference (DiD) approach, similarly to (Rohner et al., 2013). This strategy tackles the concern of endogeneity and omitted variables bias. By taking the first differences with respect to pre-war measures, we factor in every potential unobservable or non-measurable time-invariant characteristic that correlates both with voting and with conflict intensity, and would thus confound the estimates.

We believe that pre-war elections of 1990 constitute an appropriate baseline for the analysis, because the political landscape remained unchanged after the conflict: the same parties run for elections, and their core policies are still defined along the line of ethnic favoritism. For each post-conflict election (generic t), we estimate a reduced form linear probability model with the following econometric specification (Card and Krueger, 1994), obtained by differencing each post-war

[12] We focus on elections starting in 2004, when Bosnia and Herzegovina assumed full autonomy in the management and administration of the electoral process. As mentioned in Section 3.2 previous elections occurred under the OHR rule and were characterized by irregularities, vote buying, and voter intimidation (Pugh and Cobble, 2001).

election year with respect to pre-war elections of 1990:

$$\triangle Y_{m,t} = \beta_1 + \beta_2 \text{ War Casualties }_m + \delta B_{m,0} + \gamma C_{m,t} + \epsilon_{m,t}, \tag{3.3}$$

where $\triangle Y_{m,t} = Y_{m,t} - Y_{m,0}$ is the difference between turnout for municipality m in year t, with $t \in \{2004,'06,'08,'10,'12,'14\}$, and in 1990 ($t = 0$). War Casualties $_m$ represents the intensity of war in municipality m, measured by the number of fatalities and missing individuals. In the main specification, we consider the entirety of casualties occurred during the conflict (1992-1995) by municipality, and we then distinguish between civilian and military casualties. We express war casualties as the share of pre-war population by municipality of origin of the victims and we match it with the municipality of residence of the registered voters.

β_2 is the coefficient of interest, capturing the impact of conflict intensity on voter turnout. $C_{m,t}$ denotes municipality characteristics at time t. $B_{m,0}$ refers to baseline attributes (year 1990-91 or time-invariant). It includes ethnic shares, ethnic polarization (Montalvo and Reynal-Querol, 2005), Weidmann (2011) index of strategic importance,[13] pre-war population counts, the log of per-capita income, the share of cultivated land, geographic characteristics (surface area, rough terrain, distance to Croatia and Serbia), and the student/teacher ratio.

Next, to shed light on the potential mechanisms that shape the relationship between conflict exposure and voting, we include additional municipality characteristics measuring collateral effects of war intensity that could influence turnout levels. We discuss the theory and rationale behind each potential channel, together with the estimate results, in Section 3.5.2. We augment model 3.3 with a series of additional variables. We add dichotomous variables for the administrative Entity (FBiH or RS), whether the municipality was partitioned by the Internal Boundary Line established in the Dayton agreement of 1995, and the interaction of the two. Depending on data availability, we additionally include: (i) unemployment shares and average net wages (2006, 08, 10, 12), the student-teacher ratio (2014),

[13] Weidmann (2011) argues that the strategic importance of a municipality is determined not only by its own ethnic composition but also by the ethnic composition of the neighboring municipalities. If a municipality is ethnically polarized and neighbors ethnically homogenous municipalities, the strategic importance is high for the dominant group in the neighboring municipality.

population differences (2008, 10, 12), and net immigration rates. We also include the fraction of houses damaged during the war as a measure of physical capital destruction, and the share of damaged houses that were reconstructed by 2005 as part of the UN reconstruction housing program. We further include the change in ethnic polarization (from 1990 to 2013) to account for ethnic homogenization.

If voters were able to anticipate the dynamics of the conflict and the future political landscape, and adjusted their behavior accordingly, 1990 elections would be an inappropriate baseline for our estimations. This concern is, however, mitigated by the fact that the conflict and its brutality were far from foreseeable prior to their outbreak (Bieber, 2014). Not only the brutal escalation of the war was far from predictable, but its resolution and the subsequent administrative and institutional establishment were heavily influenced by external mediators such as the UN and the European Community. The institutional structure in Bosnia today was established within the Dayton Agreements in 1995 before any election took place. It is so unique (i.e. the presence of the OHR, asymmetric federal structure) that is hard to argue that power-sharing rules and municipality borders could have been easily anticipated. Swee (2015) argues that the precise locations of municipality borders were discussed and overthrown until the very last second of the signing of the Dayton agreement and uses the decision on the boundaries as exogenous variation of ethnic composition at the municipality level.

Because the conflict spread geographically, we repeat the estimations allowing the standard errors to be spatially correlated, calculating the distance of municipality centroids to each other. Section 3.5.4 presents some additional robustness checks.

3.4.1 Selective Targeting

If the aggressors selectively perpetrated higher violence in places where voter turnout was lower, the coefficient of civilian casualties would reflect selective targeting and reverse causation. However, the conflict literature suggests a different set of elements and goals that played a prime role in the outbreak and the spread of the Bosnian War, based on historical reconstructions and trial depositions. The major objectives of the Bosnian War were defined along the lines of secession,

territorial control, and ethnic homogenization (Weidmann, 2011; Novta, 2013; Costalli and Moro, 2012).[14]

For the validity of a difference-in-difference approach, pre-"treatment" parallel trends constitute a crucial assumption. In our setting we observe the same municipalities before and after the war. The assumption means that turnout trends in the elections that preceded the war outbreak should not have evolved in a way that systematically correlates with the subsequent conflict intensity. A common approach is to run placebo regressions of the diff-in-diff bringing the occurrence of the event forward in time, and interacting the treatment with a vector of pre-shock time dummy variables. Because 1990 elections were the only free and multi-party elections that preceded the conflict, we are not able to show a full parallel-trends test. To address this issue, we thus follow a strategy similar to Blattman (2009) and check whether municipalities were targeted in a non-random fashion. We check whether municipalities that were less politically active in 1990 displayed higher shares of war casualties. Table 3.6 shows the opposite scenario: civilian casualties and pre-war turnout rates correlate positively. Municipalities with higher turnout rates in 1990 experienced higher levels of civilian casualties during the war. In column (2) we add baseline characteristics, and in column (3) we add the share of military casualties, and the coefficient on civilian casualties does not change substantially. Two key aspects to notice are that voter turnout in highly affected municipalities has in fact *decreased* after the conflict resolution, and the results of our difference-in-differences estimations show a *negative*, rather than a positive, impact of casualties on voting. On one side, finding a positive correlation between pre-war turnout and casualties means that we should exert some caution when interpreting our main results. We are potentially overestimating the effect if voting in 1990 elections was higher in municipality most hit

[14]During the 16th Assembly of the Serbian People in Bosnia and Herzegovina, the Bosnian Serbs' leader Radovan Karadzic announced six strategic goals: separation from the other two ethnic communities, control of a corridor between the north-eastern region of Semberija and the north-western Krajina (self-proclaimed as a Serbian Republic from Croatian territories), establishment of a Serbian corridor along the Drina river, establishment of a border along the Una and Neretva rivers, the partition of Sarajevo, and access to the sea. Source: the minutes of the 16th Assembly of Serbian People in Bosnia and Herzegovina, 12 May 1992, Banja Luka. This was reported by the historian Robert Donia during his deposition as a witness for the ICTY (Sense Tribunal, The Hague, 01.06.2010).

by violence (or underestimating the effect in case of a downward pre-war voting trend). However, the "placebo" cross-sectional estimation can only be interpreted as a correlation. Locally based militias were crucial in facilitating the operation of paramilitary and volunteer armed units (Novta, 2013). We might thus expect more intense military action to originate from municipalities that were better able at organizing and mobilizing their local forces, and also had higher levels of social capital. The difference-in-differences precisely address the issue of unobserved heterogeneity. Additionally, the coefficient in column (3) is smaller than the effects of war exposure on voting that we find on average, in a regression where the outcome is expressed in levels. For a comparison of the effect, Table B-4 shows the results regressing Equation 3.3 on turnout expressed in levels rather than as a difference. This suggests that there is additional cross-sectional variation in the negative impact on voting that cannot be explained by mean-reversion.

As a final robustness check, we follow an instrumental variable approach. We instrument the number of civilian casualties with the ruggedness of the terrain of the municipality. Conflict theorists highlight the role that geographic characteristics play in wars. Controlling a mountain facilitates attacking the opposing group in the valley below (Novta, 2013), as the combating group can shell the valley. In Section 3.5.4 we discuss the results and the validity of the instrument.

3.5 Results

3.5.1 Civil Conflict and Voter Turnout

The average turnout in Bosnia and Herzegovina in the two decades of post-conflict general and local elections ranges between 49% and 62% (see Table 3.2). Voting varies considerably across municipalities, as graphically shown in Figure 3.4 for the latest elections of 2014. Before the conflict, average turnout was higher (80%) and had a lower dispersion across the administrative units (see Figure 3.3 and Table 3.2). This Section presents the estimates of the impact of civil conflict on the decrease in voter turnout, analyzing separately each local (2004, 2008, 2012) and central election (2006, 2010, 2014) that took place after the war. War-intensity is measured by the ratio of casualties over pre-war population by municipality of

origin. The outcome variable is given by the difference between turnout in each post-war election and turnout in 1990. Voter turnout is the share of total votes over total registered voters in each municipality.

Before presenting the results of our differencing strategy based on Equation 3.3 for each local and general election, in Table 3.4 we present the different results that we obtain by distinguishing war casualties based on their military status, using 2010 elections as an illustrative example. Column (1) shows the estimates for a measure of conflict given by the total share of casualties that occurred during the war, irrespective of their nature. The coefficient is small and not statistically significant at conventional levels. In column (2) we separate casualties by their status and obtain very different results: civilian casualties negatively impact turnout rates, while military casualties display a positive correlation. We then control for baseline characteristics (column 3) that represent elements of strategic targeting during the conflict (such as ethnic shares, distance from Serbia and Croatia, demographic, geographical and socioeconomic characteristics), and we see that the coefficient on military casualties loses its statistical significance. This suggests that the positive correlation between military victims and voters' turnout in fact reflects an omitted variable bias. In column (4) we control for additional post-conflict municipality characteristics, including measures of conflict intensity such as war-related displacement and physical capital damage. In column (5) we exclude military casualties from the regression. When we add municipality characteristics, the coefficient on civilian casualties maintains its magnitude and statistical significance. This finding supports the hypothesis that violence specifically addressed towards *civilians* can provoke profound ruptures in the social capital of a community (Kalyvas and Sambanis, 2005), and that the measure chosen as a proxy for war intensity can fundamentally matter for the results.

Once we have established the relevance of measuring civil conflict with violence towards *civilians*, we then repeat the estimations for every post-war election between 2004 and 2014. Graph 3.5 illustrates the point estimates with their 95% confidence intervals for the coefficient of civilian casualties in each election. The Graph reports the results based on Equation 3.3 for three different specifications: (i) without any additional regressor, (ii) with baseline municipality characteristics, and (iii) with baseline and post-war municipality characteristics (see Table

B-1 in Appendix for the coefficients of each regression, the mean, and the standard deviation of the dependent variables). Every regression includes military casualties. Pre-war characteristics (1991) include: the log of per-capita income, ethnic polarization (Montalvo and Reynal-Querol, 2005), Ethnic shares, population, a synthetic measure of geographic strategic importance (Weidmann, 2011), primary school students per teacher, distance from Croatia and Serbia, the share of land used for cultivation, surface area, and terrain ruggedness (standard deviation). Post war characteristics include: damaged houses (% of population) in 1995, repaired houses by 2005 (as a fraction of total damaged houses), net immigration shares in 1995, Entity (Rs or FBiH), a dichotomous indicator for municipalities partitioned by the Inter-Entity Boundary Line in 1996. For a subset of election years, depending on data availability, they also include: unemployment rates (2006, 2008, 2010), average net wage (2006, 2008, 2010), change in population (2008, 2010), and change in ethnic polarization (2012, 2014).

The effect is large and statistically significant for all elections and all three specifications. The impact holds its statistical significance and magnitude up to twenty years after the conflict resolution. Considering the results obtained by estimating Equation 3.3 including baseline characteristics, a one percent increase in the share of civilian casualties leads to an average decrease in turnout that ranges between 1.7 and 2.5 percentage points. In other words, a one standard deviation increase in the share of civilian casualties (1.24% of the population) leads to a decrease in voter turnout of 3 (in 2006), 3.7 (2004, 2008, 2014), 4.2 (2010), and 4.5 (2012) percentage points. The coefficients translate roughly into one-third of a standard deviation change in turnout since 1990. Considering that civilian casualties reached up to 7% of the population in some municipalities, the effect is large in magnitude for every election. The impact is stable over time. Lastly, it is worth noting that the effect is similar between municipal and central elections. This suggests that the "supply side" of voting, which is different for local and general elections, does not determine our results. The experience of civil conflict violence towards civilians affects voter turnout up to twenty years after its resolution.

3.5.2 Mechanisms

We find that the severity of violence towards civilians, measured by the share of fatalities and missing individuals in the municipality of origin, decreases voter turnout with statistical significance and relevant magnitude. Our results contrast with the evidence of proactive social behavior found in Sierra Leone by Bellows and Miguel (2009), among former child soldiers in Uganda (Blattman, 2009), and in a series of micro-data based studies summarized in a meta-analysis by Bauer et al. (2016).

How does civil conflict affect the voting behavior of registered voters in post-conflict elections? The literature shows that war violence and civil conflict generate disrupting effects along an extensive range of dimensions in war-torn economies and societies. All such consequences potentially mediate the effect of civil conflict violence on political participation. This section summarizes which dimensions are likely to be affected through war experience and how we rule these alternative explanations out in our empirical analysis.

War and civil conflict violence induce changes in the demographic and social structure of the affected regions, through the direct effect of violence and through forced displacement. The Bosnian War caused almost 100,000 deaths and the displacement of almost 2 million refugees. Refugees and returned migrants potentially differ from non-displaced individuals in terms of their socio-economic characteristics and preferences. Kondylis (2010) finds evidence of higher unemployment rates among conflict-related displaced male migrants and higher drop out rates from the labor force among displaced women in BiH. We expect that communities with high shares of migrant members may differ from non-migrant communities in their voting behavior.

The ethnic composition of municipalities changed drastically between the census in 1990 and the census in 2013. Ethnic polarization decreased over time. The Bosnian conflict ended with the division of the territory into two main Entities. The Dayton Agreement redesigned the boundaries of some municipalities pertaining to the most contested areas along the internal front line. Swee (2015), relying on the partition of BiH municipalities, finds that the most ethnically homogeneous areas provide more schooling, measured by the number of primary schools and

the number of teachers per pupil. Changes in human capital are another potentially crucial channel through which war violence can affect voting. The literature documents large and persistent effects in education as a consequence of conflict (Akresh and de Walque, 2008; Leon, 2012). The ethnic composition of municipalities and public spending on education are likely to differ across more or less war-affected municipalities and correlate with political participation.

Variation in voter turnout across municipalities may also be influenced by local economic conditions. Because civil conflict can disrupt employment opportunities, we expand the analysis by including labor market characteristics, measured by unemployment rates and average net wages. An opposite consequence of conflict-related physical capital damage stems from post-war reconstruction programs, which bring about demand for labor. Reconstruction activity creates employment opportunities and large capital inflows. Reconstruction activity also exposes the local population to the presence of international actors, NGOs and volunteers, potentially influencing social capital and political preferences in the recipient areas.

To take into account the mechanisms discussed above, we replicate the estimation of Equation 3.3 including a series of post-war characteristics at the municipality level. We add the share of damaged houses (% of population) in 1995, the number of houses reconstructed by 2005 (as a fraction of total damaged houses), the number of war-related net emigrants in 1995 as a share of pre-war population, to which administrative entity the municipality belongs (RS or FBiH), and a dichotomous indicator for municipalities partitioned by the Inter-Entity Boundary Line. For a subset of elections, depending on the availability of the information, we also include: unemployment rates (2006, 2008, 2010), average net wages (2006, 2008, 2010), the change in pupil-teacher ratio in primary school (2014), changes in population (2008, 2010, 2012), net internal immigration (2010, 2012), and changes in ethnic polarization (2012, 2014). Because post-war characteristics of the municipalities may be endogenous towards war exposure, we additionally estimate the results by adding one post-war variable at a time, and we find that the coefficient estimates for war exposure are not sensitive to their inclusion.[15]

Table 3.5 displays the results of the most comprehensive model, which in-

[15]The results of the single-entry post-war variables regressions are available upon request.

cludes all the baseline and all the post-war municipality characteristics. They correspond to the diamond-shaped point estimates in Figure 3.5. The coefficients of civilian casualties are statistically significant at the 1% level in all elections (5% in 2004). A one standard deviation increase in civilian victims causes a reduction in voter turnout with respect to pre-war elections between 3 and 4.6 percentage points. The estimates do not differ significantly compared to our baseline specification.

Another possible explanation for the decreased turnout in war-affected areas is that is not necessarily the demand side of the political landscape that has changed due to war exposure, but the supply of political actors. We thus check whether political competition in the municipalities differs with the intensity of violence experienced. In Table 3.8, we investigate whether the number of parties running in the local elections of 2008 varies with the number of civilian casualties and see no differential patterns. We regress measures of violence separately on the number of parties running for the municipal council in the elections of 2008 and the number of parties proposing a mayor, adjusted by the population. Neither regressions shows a significant relationship with war intensity, suggesting that political competition does not differ as a result of the civil war.

Lastly, one can argue that turnout rates may be lower in affected areas not because voting has decreased but because registration rates have increased. In Table 3.9 we regress war intensity on the change in the number of people that registered to vote in the elections after the war from the number of registered in the elections in 1990. We do not see a statistically significant nor consistent relationship between war intensity and the number of registered voters in the post war elections.

The coefficients for war exposure on turnout rates are robust to the inclusion of all the discussed alternative explanations. It implies that voter turnout is affected by war exposure on top of the economic, socio-demographic, and ethnic consequences. This result supports the hypothesis that conflict changes the preferences of the most victimized individuals. We thus explore further this mechanism analyzing an individual-level survey on social capital and political preferences, in the following Section 3.5.3.

3.5.3 Social Preferences, Trust, and Voting

In our aggregate analysis, we find that war exposure decreases voter turnout. We hypothesize that fear and grievance generated by the civil conflict have eroded social relations and the sense of community. We thus explore whether our aggregate-level results mirror a similarly negative relation between exposure to violence and measures of social capital such as trust and political participation at the individual level. We use individual survey data on social capital, and voting from the LITS-I (2006) survey[16]. Because the survey does not provide information on war violence at the individual level, we use the information on municipality civilian casualties. For a series of social capital outcomes we estimate the following linear probability model:

$$SC_{i,m}^{`06} = \beta_0 + \beta_1 CivilianCas_m + \beta_2 SC_i^{`89} + \delta_i X_i^{'} + \gamma_m M_m^{'} + \epsilon_{i,m} \qquad (3.4)$$

$SC`06_{i,m}$ denotes the social capital measure of individual $i$ in municipality $m$ in 2006, $SC_{im}^{`89}$ in 1989. Social capital outcomes include: (i) generalized trust ("do you trust people in general?"), (ii) trust in institutions, and (iii) whether the individual voted in the most recent elections.[17] Trust is expressed as a categorical variable, ranging from 1 to 5, where 1 denotes "complete distrust" and 5 is "complete trust". The LITS-I survey includes the question "Did you trust people before 1989?". Despite the need for some caution due to the recalled nature of this information, it allows us to control for a pre-war measure of interpersonal trust. We do not have information on pre-war voting, but we include a variable indicating whether the respondent was a member of the Communist party before 1989, as a proxy for pre-conflict political engagement. We compute an index of trust in institutions as the average of trust in the president, the ministers, the parliament, and political parties. The survey does not report information on trust in institutions prior to the conflict. We control for individual (i) characteristics ($X_i^{'}$) and municipality (m) characteristics ($M_m^{'}$). Standard errors are clustered at the

[16] See Section 3.3 for a description.

[17] The survey was conducted between August and October 2006. It is not clear to which elections the question "Have you voted in the last presidential/parliamentary election?" refers to. General elections took place on October 1st, 2006 and local elections took place in 2004.

municipality level. For comparability purposes, we standardize all the dependent variables (voting, generalized trust, trust in institutions), pre-war trust, and the number of casualties by municipality, such that they have mean 0 and a standard deviation of 1.

Table 3.11 shows the regression results from Equation (3.4) for voting (columns 1 and 2), generalized trust (columns 3 and 4), and trust in institutions (columns 5 and 6). Our findings support the hypothesis that social capital and social participation are lower for individuals living in municipalities with higher exposure to civil conflict. One standard deviation (SD) increase in civilian casualties is associated with a decrease in the propensity to vote by 0.16 SD. The inclusion of individual and municipal characteristics does not alter the significance of the coefficient and increases the magnitude of the effect (column (2)). One SD increase in war intensity is associated with a decrease of generalized trust by around 0.2 SD (column (4)), and of trust in institutions by 0.2 SD (column (6)). These findings reveal a tight link between civil war and individuals' societal perceptions and preferences.

If war violence specifically hit with higher intensity individuals with lower levels of social capital, our results would be upward biased. To rule specific targeting, we follow two strategies. First, we check for heterogeneous effects by age. As Cassar et al. (2013) highlight, it is less likely that militias target young individuals on the basis of their social preferences. In addition, children and adolescents were not able to take active part and self-select into the conflict. Table 3.12 shows that the coefficient of the interaction between young age (< 14 years) and war intensity is not significant. As a second approach, we follow the empirical strategy of Blattman (2009) and show that there is no statistically significant correlation between war exposure and pre-conflict trust, political participation, and socioeconomic characteristics of individuals. Table 3.13 reports the results, supporting the claim that the results are not biased by specific targeting of individuals based on their levels of social capital.

Lastly, readers may be concerned that victims of the civil war are less likely to respond to questions regarding social preferences in the survey. This could confound our estimates as it would make survey responses between war victims and others incomparable. For the considered measures of social capital (voting, trust in others, trust in institutions), around 10% of survey respondents do not answer

the questions. In Table B-5 we analyze whether individuals from high-violence regions are less likely to answer to the survey questions regarding their social preferences. The coefficient estimates on civilian casualties on the likelihood to respond show no statistically significant relationship and are small in magnitude.

These findings support the hypothesis that ethnic conflict led to lower political participation through changes in preferences and behavior of the affected individuals.

3.5.4 Robustness Checks

In order to assess the robustness of our findings, we perform a series of checks and placebo tests. It is reasonable to assume that the dynamic and intensity of the war generated spatial spillovers. The probability of a municipality to have been attacked during the war is likely positively correlated with the strategic importance of the neighboring municipalities (Weidmann, 2011). The standard errors of our main results may be biased by omitting to allow for spatial correlation across observations. We thus repeat the main analysis allowing for correlation of standard errors between bordering municipalities, taking the distance of municipality centroids to each other.[18] If we allow for spatial correlation, standard errors are generally smaller (see Table B-2 and compare to standard errors in Panel B of Table B-1). We, therefore, prefer to report standard errors using White's heteroskedasticity-consistent estimator in our main analysis, as these results appear to be more conservative and do not rely on assumptions about how close municipalities must be for the standard errors to be spatially correlated.

More than 90% of war casualties were men (BBD, 2008). This may raise the concern of a gender composition bias in the electorate. If men vote disproportionately more than women, lower turnout rates may be a mechanic consequence of the change in gender composition resulting from the conflict. Unfortunately, we do not have information on gender differences in turnout before the war. We can, however, use the turnout results of the general election of 2006 to calculate the difference in turnout between men and women. We observe that male turnout ex-

[18] We use Hsiang's (2010) Stata code for the adjustment of spatially correlated standard errors, whose approach follows Conley (1999).

ceeds female turnout by 2.7 percentage points (58.6% vs. 55.9%). The difference is not large enough to explain the decreased turnout rates by 2-3 percentage points we observe after the war. When we check for heterogeneous effects between men and women, we find a similar effect of conflict exposure on female turnout compared to male turnout (see Table B-3 in Appendix for heterogeneous effects by gender).[19] This suggests that the effect is not gender-specific. Lastly, we find that there is no statistically significant impact of conflict on the ratio between male and female registered voters.

Another approach to rule out the sorting into victimization of low participation individuals is to check whether voters who were too young to have been directly targeted in the war respond differently to war exposure than older cohorts. For this, we repeat our main analysis (Equation (3.3)) but looking at turnout rates of voters below and above 30 years old in the general election of 2006. We can see in Table 3.7 that the effect of war exposure is very similar for the voters that were at most 19 years old at the end of the conflict compared to older voters.

As a final robustness check, we adopt an instrumental variable approach. The conflict literature makes use of geographic characteristics of villages or municipalities as sources of exogenous variation in violence intensity, such as distances to capital cities or neighboring regions, or terrain characteristics (Voors et al., 2012; Cassar et al., 2013). The idea is that these villages only experience violence due to the geographic characteristics and would have been shielded if they were, for example, further away from the capital or less mountainous. The assumption is that these characteristics do not influence social capital measures directly, but only through the intensity of violence. We use the standard deviation of terrain ruggedness to instrument conflict intensity. As discussed in Section 3.4.1, the literature shows that holding a mountain top in a municipality can be strategically important in warfare because it facilitates hiding and attacks towards the valley. This logic also applies to the Bosnian context (Beger, 2012; Novta, 2013). Holding a mountain top is only beneficial if there is a valley to be attacked. We, therefore, use the variation in ruggedness to instrument civilian casualties. In the first and the second stage of our Instrumental Variable (IV) estimation, we control for military casual-

[19] In column (4) of Table B-3 we can observe that there is no significant relation between the intensity of war casualties and the difference in turnout between genders in 2006.

ties and the usual baseline characteristics. In order to increase precision, we pool all the elections and control for year fixed effects. Table 3.10 displays the regression results of both the OLS and the 2SLS IV estimations (columns (1) and (2)). The first stage results depicted in column (3) show that the instrument works in the expected direction: the higher the variability in ruggedness in a municipality, the more civilians died during the civil war. The partial F-Value of the first stage regression (Table 1.6, column 3) shows that we have a strong instrument, with a partial F-Value of 42.57. The coefficient estimates of the OLS and 2SLS IV regressions are similar, but the IV estimates are slightly smaller in magnitude (-2.9 and -2.2 percentage points) and less precisely estimated. The exclusion restriction is not testable, but we find that variation in ruggedness does not significantly correlate with turnout rates in 1990, before the outbreak of the War. The instrument is static in nature, and our identification strategy relies on the assumption that they exerted an influence only while the conflict took place, similarly to Rohner et al. (2013).[20]

3.6 Conclusion

There is growing evidence that exposure to violence and conflict can affect individual behavior and ultimately reshape preferences (Voors et al., 2012). Existing studies based on micro-level data show diverging effects. Traumatic experiences can adversely affect trust (Alesina and La Ferrara, 2002), but war violence may also generate the opposite consequence of fostering social capital and collective action (Bauer et al., 2016; Bellows and Miguel, 2009; Blattman, 2009). We estimate the impact of civil conflict on voter turnout in Bosnia and Herzegovina, following a difference in differences strategy based on pre-war and post-war official electoral statistics at the municipality level. We find that voting decreases in response to intense violence towards civilians, up to two decades after its resolution, by approximately 4 percentage points per one standard deviation increase in civilian casualties. To shed light on the mechanisms through which civil war

[20] As in the OLS specification, there is no systematic relationship between the number of military casualties and voter turnout if we instrument military casualties with terrain ruggedness. Results are available upon request.

violence affects voting, and to conciliate our findings with the existing literature, we integrate the analysis studying individual-level data on social capital and civic engagement. We show that the negative impact on voting pairs with lower levels of trust and social capital. War victims exhibit social and political apathy and report mistrust in institutions, confirming a trend largely lamented in anecdotal evidence and in the press (Wiendel Rasmussen, 2017).

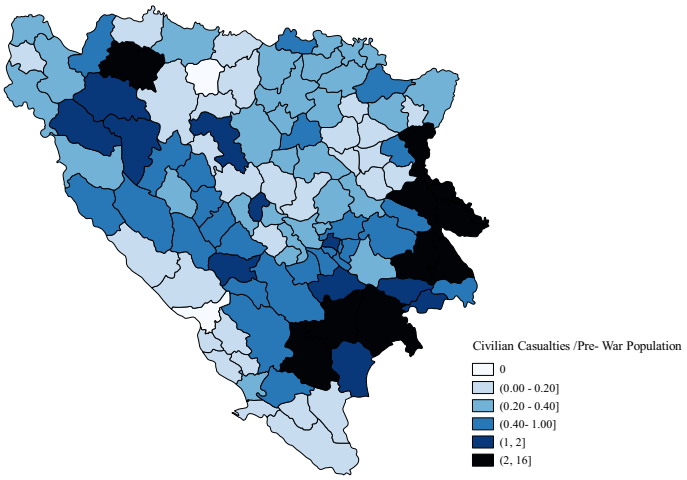
Our study contributes to the existing literature by focusing on the context of an ethnic-grounded civil war, fought within previously tied communities, and characterized by a static and comparable political scenario. Political parties still define their core agenda along ethnic divisions and nationalism. In this light, our results are consistent with the evidence of lower trust toward out-group community members (Cassar et al., 2013). Because in the BiH context "generalized" trust encompasses "out-group" trust, our results also conciliate with opposite findings of increased in-group "parochial" trust and civic engagement, found in other recent micro-level studies (Bauer et al., 2016; Blattman, 2009; Bellows and Miguel, 2009). Bauer et al. (2016) note that many studies do not clearly determine where the boundary between in- and out-group members lies. Our results underline the importance of defining competing groups, how they interacted before the war, and how they define their identity after the conflict.

By separating civilian casualties from military victims, we further show that the different nature of victims as a measure of conflict can imply opposite conclusions.

Many studies established a tight link between the development of solid institutions and economic activity with social capital and political participation. Our results of a negative relation between violence towards civilians, voting, generalized trust, and trust in institutions may, therefore, be part of a broader picture explaining the difficult recovery and transition of war-torn economies and societies. Institutional actors should be concerned if a part of the society that suffered the most during the civil war does not vote. The under-representation of the victimized population in politics and institutions may ultimately distort public goods provision, redistribution, and economic activity. Ignoring the legacies of conflict in terms of societal distrust and apathy toward institutions can lead to a dangerous underestimation of the true costs of civil conflict.

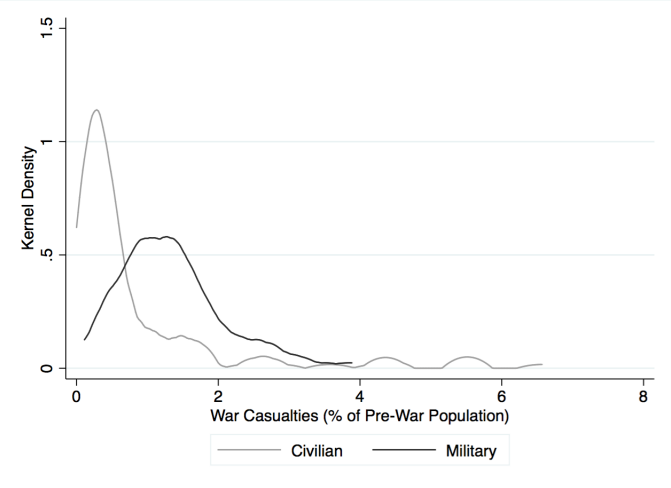
Tables and Figures

Figure 3.1: Civilian casualties by municipality of origin



Source: Authors' estimation from the "Bosnian Book of Dead". Percentages refer to the war-related number of dead or missing civilians computed as a fraction of pre-war populations by municipality, based on the 1991 BiH Census. Full sample of municipalities.

Figure 3.2: War casualties by municipality of origin, by military or civilian status



Source: Authors' estimation from the "Bosnian Book of Dead". The graph reports kernel densities of war-related number of dead or missing civilians and militaries by municipality of origin, computed as a fraction of pre-war populations by municipality (based on the 1991 BiH Census), for the main estimation's sample of municipalities. Srebrenica and Mostar are excluded.

Table 3.1: Descriptive statistics: war intensity, reconstruction, and internal displacement

Variable	Observations	Mean	Std Dev	Min	Max
Civilian Casualties (Total)	127	320.72	572.60	0	4026
Military Casualties (Total)	127	517.52	436.83	5	2056
Civilian Casualties (% of Pre-war Population)	127	0.83	1.24	0.00	6.57
Military Casualties (% of Pre-war Population)	127	1.33	0.75	0.11	3.88
Houses Damaged / Pre-war Population ('000)	127	8.08	7.16	0.00	31.70
Houses Repaired / Damaged (by 2005)	127	0.49	0.42	0.00	2.66
Internally Displaced (1995)	127	1172.34	1410.29	1	7604
Internally Displaced (1995, % of Pre-war Pop.)	127	0.04	0.04	0.00	0.21

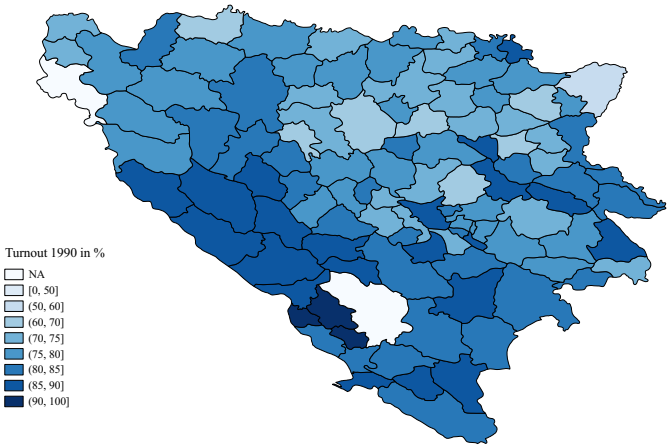
Source: Authors' estimation from the "Bosnian Book of Dead", 1991 BiH Census, and the Federal Office of Statistics of Bosnia and Herzegovina. War casualties refer to victims' municipality of origin.

Table 3.2: Descriptive statistics: municipality characteristics and voter turnout

Variable	Observations	Mean	Std Dev	Min	Max
Baseline Characteristics					
Log (Per-capita Income) in 1991	127	8.49	0.19	8.15	8.95
Ethnic Polarization	127	0.77	0.21	0.04	0.98
Population ('000) in 1991	127	40.24	30.93	4.17	195.69
Strategic Importance Municipality	127	0.03	0.02	0.00	0.06
Students/Teacher in 1991	127	24.13	22.30	12.52	265.29
Distance to Croatia in km	127	52.73	35.26	4.92	137.52
Distance to Serbia in km	127	86.59	63.45	4.74	243.52
Surface Area	127	363.86	266.37	9.06	1224.74
% of Cultivated Land	127	0.18	0.16	0.00	0.67
Ruggedness St. Dev. (3x3 pixel) ('000)	127	0.10	0.04	0.00	0.24
Ethnic Shares in 1991 (omitted: Yugoslavian and Other Ethnicity)					
Muslim	127	0.41	0.24	0.00	0.97
Serb	127	0.36	0.26	0.00	0.97
Croatian	127	0.18	0.25	0.00	0.99
Voter Turnout (Voters/Registered)					
Turnout in 1990	127	79.65	6.42	60.00	96.00
Turnout 2004 Local Elections	127	48.37	10.19	22.85	84.30
Turnout 2008 Local Elections	127	60.56	8.97	39.97	92.27
Turnout 2012 Local Elections	127	61.39	9.81	36.21	91.59
Turnout 2006 General Elections	127	57.27	7.52	36.52	75.48
Turnout 2010 General Elections	127	58.44	8.16	33.75	82.68
Turnout 2014 General Elections	127	56.02	9.38	32.77	80.40
Δ Local Turnout 2004 (2004-1990)	127	-31.28	12.72	-61.33	1.30
Δ Local Turnout 2008	127	-19.09	10.45	-47.03	7.27
Δ Local Turnout 2012	127	-18.26	11.38	-44.22	8.70
Δ General Turnout 2006	127	-22.37	9.25	-53.89	-1.52
Δ General Turnout 2010	127	-21.21	9.51	-49.22	-1.32
Δ General Turnout 2014	127	-23.63	10.61	-52.61	2.40
Post-war Characteristics					
Municipality partitioned	127	0.42	0.50	0	1
Republika Srpska (D=1)	127	0.42	0.50	0	1
Ethnic Polarization in 2013	127	0.43	0.30	0.01	0.99
Unemployment Rate 2006	127	48.55	14.22	18.02	80.56
Unemployment Rate 2008	127	45.93	14.05	14.46	86.49
Unemployment Rate 2010	127	48.82	14.62	5.32	97.78
Unemployment Rate 2012	127	50.67	13.67	16.58	86.18
Average Net Wage 2006	127	506.31	93.08	278.00	851.00
Average Net Wage 2008	127	680.72	109.51	476.00	1069.00
Average Net Wage 2010	127	726.99	111.22	531.93	1135.81
Average Net Wage 2012	127	749.91	120.67	524.00	1195.00
Population ('000) in 2008	127	27.11	30.35	0.26	223.64
Population ('000) in 2010	127	27.16	30.64	0.41	226.46
Population ('000) in 2012	127	27.12	30.79	0.53	228.64
Net Immigration (% of Pop.) 2010	127	0.00	0.02	-0.02	0.19
Net Immigration (% of Pop.) 2012	127	0.00	0.03	-0.01	0.27

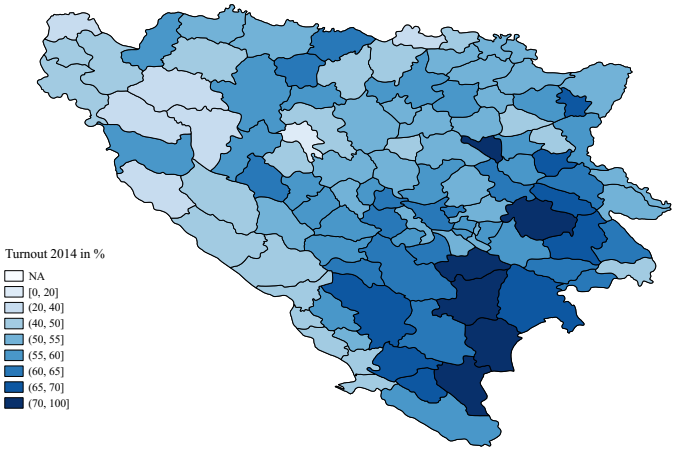
Source: Authors' estimations from the 1991 Bosnia and Herzegovina's Census, Official Electoral Statistics, and FOS-BiH.

Figure 3.3: Voter turnout in 1990 by municipality



Source: Mraović (2014)

Figure 3.4: Voter turnout in 2014 by municipality



Source: Central Election Commission of Bosnia and Herzegovina

Table 3.3: Summary statistics, LITS-I (2006)

Variable	N	Mean	Std Dev	Min	Max
Demographic Characteristics					
Age	697	49.39	16.63	18	87
Female	697	0.57	0.49	0	1
Married	697	0.23	0.42	0	1
Divorced	697	0.04	0.20	0	1
Employed	697	0.38	0.49	0	1
Inactive	697	0.51	0.50	0	1
No Education	697	0.17	0.37	0	1
Primary Education	697	0.18	0.38	0	1
Secondary Education	697	0.55	0.50	0	1
Tertiary Education	697	0.11	0.31	0	1
Income Rank [1-10]	697	4.35	1.97	1	10
Income Rank in 1989 [1-10]	697	6.64	2.10	1	10
Subj. Health Assessment (1: Bad, 0: Not Bad)	697	0.26	0.44	0	1
Household number of Children	697	0.37	0.71	0	4
Urban Settlement	697	0.45	0.50	0	1
Minority	697	0.09	0.29	0	1
Bosnian	697	0.50	0.50	0	1
Croat	697	0.10	0.30	0	1
Serb	697	0.33	0.47	0	1
Other Ethnicity	697	0.06	0.25	0	1
War Exposure & Displacement					
Ever Fought as a Soldier	697	0.11	0.31	0	1
Internal Migrant Before 1996	697	0.11	0.32	0	1
Civilian Casualties	697	0.46	0.66	0.06	4.41
Civilian Casualties (*Municipality Measure*)	32	0.45	0.75	0.06	4.41
Voting & Social Capital					
Voted in Previous Elections (2006)	697	0.65	0.48	0	1
Party Member	697	0.14	0.35	0	1
Party Member in 1989	697	0.09	0.28	0	1
Generalized Trust (1: complete distrust, 5: complete trust)	697	0.19	0.39	0	1
Generalized Trust in 1989	697	0.58	0.49	0	1
Change in Trust (pre to post-war): Yes to No	697	0.43	0.49	0	1
Change in Trust: No change	697	0.54	0.50	0	1
Change in Trust: No to Yes	697	0.03	0.18	0	1
Trust in Institutions	697	2.16	1.13	1	5

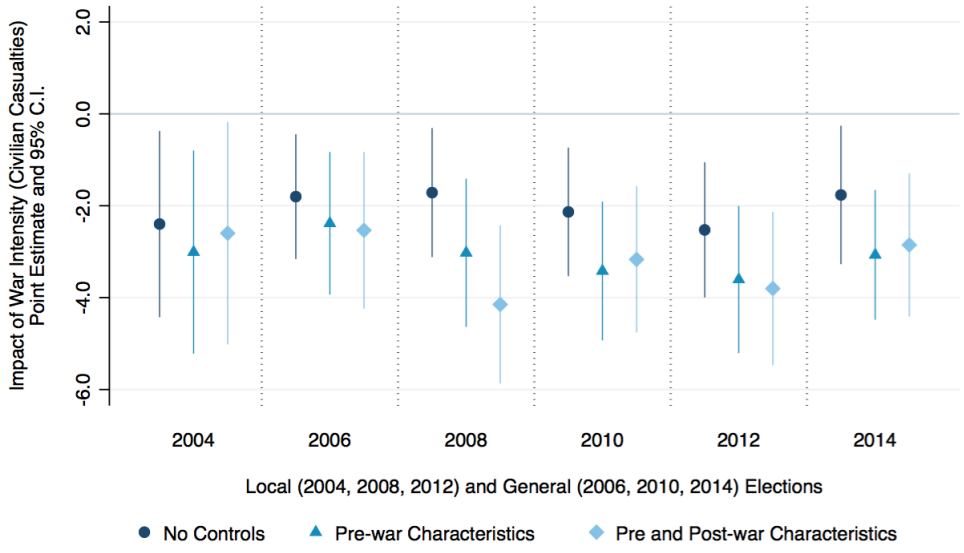
Source: Authors' estimations from the EBRD Life in Transition Survey I, 2006 (LITS-I). The data report information at the individual level in a nationally representative survey that covers 32 municipalities.

Table 3.4: War casualties and general elections in 2010

	(1)	(2)	(3)	(4)	(5)
Dependent Variable: $Turnout_{2010} - Turnout_{1990}$					
Casualties	0.373				
	(0.435)				
Civilian Casualties		-2.135***	-3.420***	-3.168***	-2.856***
		(0.714)	(0.770)	(0.811)	(0.768)
Military Casualties		4.962***	0.573	1.600	
		(1.557)	(1.595)	(1.567)	
Pre-War Characteristics	No	No	Yes	Yes	Yes
Post-War Characteristics	No	No	No	Yes	Yes
R^2	0.00504	0.0926	0.424	0.494	0.489
Observations	127	127	127	127	127
Mean Dep. Variable	-21.21	-21.21	-21.21	-21.21	-21.21
Std Dev. Dep. Variable	9.507	9.507	9.507	9.507	9.507

Source: Authors' estimation from the "Bosnian Book of Dead" (BBD 2008), 1991 BiH Census, official electoral statistics from FOS-BiH, and geographic data at the municipality level (see Section 3.3). Casualties are in % of pre-war population by municipality. Pre-war characteristics: log (per capita income), ethnic polarization, ethnic shares, population, strategic importance (Weidmann, 2011), student-teacher ratio, and share of cultivated land in 1991, area, surface ruggedness, and distance to Croatia and Serbia. Post war characteristics: fraction of damaged houses, fraction of repaired houses by 2005, 1995 emigration ratio, municipality partition, Entity, unemployment rate, average net wage, population difference (2010-1991), net emigration in 2010. Standard errors in parentheses and adjusted for heteroskedasticity. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Figure 3.5: Impact of civil conflict on voter turnout in local and general elections



Source: Authors' estimation from the "Bosnian Book of Dead", BiH Census (1991), FOS-BiH, and geographic data at the municipality level (see Section 3.3). Sample size: 127 municipalities. See Table B-1 for coefficients and standard errors. The graph reports point estimates and 95% confidence intervals for the coefficients of civilian casualties (% of pre-war population) by municipality of origin from regressions of model 3.3, (i) without controls, (ii) with baseline characteristics, and (iii) with pre and post-war characteristics. Coefficients represent the impact of war exposure on the difference between voter turnout in local (2004, 2008, 2012) and general (2006, 2010, 2014) elections and pre-war turnout (1990). Every regression includes military casualties (not displayed). Pre-war characteristics (1991): log of per-capita income, Ethnic polarization, pre-war ethnic shares, population, strategic importance (Weidmann, 2011), student/teacher ratio, distance from Croatia and Serbia, and share of land used for cultivation, surface area, terrain ruggedness. Post war characteristics: damaged houses (% of population) in 1995, repaired/damaged houses by 2005, emigration share in 1995, to which administrative entity the municipality belongs, municipality partition, unemployment rates (2006, 2008, 2010), average net wage (2006, 2008, 2010), change in population (2008, 2010), and change in ethnic polarization (2012, 2014).

Table 3.5: Voter turnout and war exposure: alternative mechanisms

Dependent Variable:	(1)	(2)	(3)	(4)	(5)	(6)
$Turnout_t - Turnout_{1990}$		Local Elections			General Elections	
$t =$	2004	2008	2012	2006	2010	2014
Civilian Casualties	-2.598**	-4.148***	-3.803***	-2.533***	-3.168***	-2.853***
	(1.235)	(0.878)	(0.851)	(0.870)	(0.811)	(0.794)
Military Casualties	-2.989	2.261	2.859*	0.721	1.600	2.214
	(2.050)	(1.607)	(1.671)	(1.451)	(1.567)	(1.964)
Houses Damaged (% Pop.) in 1995	-0.00970	-0.174	-0.393***	-0.0391	-0.187	-0.255*
	(0.141)	(0.133)	(0.115)	(0.131)	(0.130)	(0.132)
Emigrants in 1995 (% 1991 Pop.)	-32.74	29.75	-11.97	-13.98	-2.913	-47.79*
	(33.93)	(25.81)	(25.74)	(26.09)	(24.39)	(27.04)
Houses Repaired / Damaged 1995	3.762	-2.879	-5.885**	-1.824	-3.674*	-4.880***
	(2.691)	(2.357)	(2.635)	(1.861)	(1.859)	(1.802)
Municipality Partition	-5.972**	0.0743	1.197	-0.956	-1.531	-0.719
	(2.584)	(2.807)	(2.509)	(2.546)	(2.407)	(2.805)
Entity: Republika Srpska	-1.876	12.22***	10.13***	6.764**	1.646	5.609*
	(4.105)	(3.154)	(2.872)	(3.332)	(2.881)	(3.208)
Entity x Partition	5.092	-3.455	-6.437*	-3.964	-2.467	-4.076
	(4.590)	(4.023)	(3.684)	(4.108)	(3.632)	(3.997)
Unemployment Rate		0.212***	0.209***	0.200***	0.130*	
		(0.066)	(0.070)	(0.068)	(0.069)	
Average Net Wage		-0.023**	-0.027***	-0.001	-0.012	
		(0.009)	(0.008)	(0.009)	(0.008)	
Δ Population		-0.036	0.006		0.054	
		(0.068)	(0.065)		(0.057)	
Minority Representation		2.452				
		(2.149)				
Δ Ethnic Polarization			3.689			2.902
			(2.761)			(3.164)
Net Immigration			62.61***		-53.23*	
			(20.53)		(27.49)	
ΔStudents/Teacher						0.302
						(0.375)
R^2	0.481	0.525	0.642	0.417	0.494	0.543
Observations	127	127	127	127	127	127
Mean Dep. Variable	-31.28	-19.09	-18.26	-22.37	-21.21	-23.63
Std Dev. Dep. Variable	12.72	10.45	11.38	9.254	9.507	10.61

Source: Authors' estimation from BBD (2008), 1991 BiH Census, official electoral statistics (FOS-BiH), and geographic data (see Section 3.3). Casualties are in % of pre-war population by municipality. Pre-war characteristics always included: log (per capita income), ethnic polarization, ethnic shares, population, strategic importance (Weidmann, 2011), student-teacher ratio, distance to Croatia and Serbia, and cultivated land (%) in 1991. Inclusion of Post-War Characteristics depends on data availability. Standard errors in parentheses and adjusted for heteroskedasticity. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table 3.6: Conflict intensity: non-random targeting

	(1)	(2)	(3)
Dependent Variable (in 1990)		Turnout 1990	
Civilian Casualties	1.052***	1.830***	1.759***
	(0.306)	(0.461)	(0.429)
Military Casualties			0.316
			(0.895)
R^2	0.0410	0.365	0.365
Observations	127	127	127
Mean Dep. Variable	79.65	79.65	79.65
Std Dev. Dep. Variable	6.424	6.424	6.424
Baseline Characteristics	No	Yes	Yes

Source: Authors' estimation from the "Bosnian Book of Dead", 1991 BiH Census, the Federal Office of Statistics of Bosnia and Herzegovina, and additional geographic data at the municipality level (see Section 3.3). Standard errors in parentheses and adjusted for heteroskedasticity. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table 3.7: War and turnout: heterogeneous effects by age group ($\leq$30 years old), 2006

	$>$ 30 years old	$<$ 30 years old	All
Civilian Casualties	-2.399***	-2.824***	-2.384***
	(0.765)	(0.995)	(0.793)
R^2	0.320	0.337	0.331
Observations	127	127	127
Mean Dep. Variable	-20.02	-30.46	-22.37
Std Dev. Dep. Variable	9.103	10.61	9.254

Source: Authors' estimation from BBD (2008), 1991 BiH Census, official electoral statistics (FOC-BiH), and additional pre-war control variables at the municipality level (see footnote of Table B-1). Casualties indicate dead or missing individuals by municipality of origin, as a share of pre-war population. All regressions include military casualties as a separate regressor. The outcome variable is the difference in turnout from 1990 (for all voters) to 2006 (specific to the age group). column (1) shows results for voters above the age of 30 in 2006, column (2) for voters below 30 years, and column (3) for all voters. We do not display results for other elections because this information is only available for 2006. Standard errors in parentheses and adjusted for heteroskedasticity. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table 3.8: War and party competition: number of parties in local elections (2008)

	(1) # Parties (% of Pop. '000)	(2) # Parties (% of Pop. '000) (Mayor Elections)
Civilian Casualties	0.0329	0.0416
	(0.125)	(0.0726)
R^2	0.296	0.231
Observations	127	127
Mean Dep. Variable	1.001	0.421
Std Dev. Dep. Variable	1.352	0.792

Source: Authors' estimation from BBD (2008), 1991 BiH Census, official electoral statistics (FOS-BiH), and additional pre-war control variables at the municipality level (see footnote of Table B-1). The dependent variable in column (1) is the number of parties running for office at the municipality level in the local elections of 2008, as a share of the municipality's population (in thousands). The dependent variable in column (2) refers to the number of parties running for mayor in the same election (share of the municipality's population). Taking absolute values instead of shares does not alter the results. Standard errors in parentheses and adjusted for heteroskedasticity. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table 3.9: DiD: war and number of registered to vote

	(1)	(2)	(3)	(4)	(5)	(6)
Dependent Variable:						
Δ Registered Voters (t-1990)	2004	2008	2012	2006	2010	2014
Civilian Casualties	405.3	-274.7	-255.9	149.5	-166.6	-745.0
	(928.0)	(559.9)	(547.3)	(1027.8)	(544.4)	(770.3)
Military Casualties	-1990.4	545.3	281.0	-2993.7	577.0	-1264.6
	(1996.7)	(1023.1)	(1188.2)	(2321.3)	(1092.0)	(1641.8)
R^2	0.591	0.907	0.911	0.481	0.908	0.695
Observations	127	127	127	127	127	127
Mean Dep. Variable	-11396.0	-6503.8	-4932.0	-8388.2	-5508.8	-15229.7
Std Dev. Dep. Variable	14113.2	15387.1	16366.9	14496.4	15994.3	13857.2
Baseline Characteristics	Yes	Yes	Yes	Yes	Yes	Yes
Post-war Characteristics	Yes	Yes	Yes	Yes	Yes	Yes

Source: Authors' estimation from BBD (2008), 1991 BiH Census, official electoral statistics (FOS-BiH), with pre and post-war municipality characteristics (see footnote of Table B-1). Casualties indicate total dead or missing individuals by municipality of origin, as a share of pre-war population. The dependent variables are the difference between the total number of registered voters at time t and in 1990. Estimates based on registered voters as a share of municipality's population (available for years 2008, '10, and '12) are available upon request (coefficients for casualties are statistically non-significant nor economically large). Standard errors in parentheses and adjusted for heteroskedasticity. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table 3.10: War and turnout: 2SLS IV (ruggedness)

	(1) (OLS) Turnout	(2) (IV: Second Stage) Turnout	(3) (IV: First Stage) Civilian Casualties
Civilian Casualties	-2.917***	-2.197*	
	(0.357)	(1.331)	
Ruggedness Std Dev (3x3 pixel, '000)	6.004		8.335***
	(11.468)		(1.181)
Military Casualties	0.738	0.227	0.709***
	(0.778)	(1.249)	(0.073)
Observations	762	762	762
R^2	0.421	0.419	0.563
Partial F-Stat (First Stage)	.	.	42.566
Mean Dep. Variable	-22.638	-22.638	0.832
Std Dev. Dep. Variable	11.505	11.505	1.233
Year FE	Yes	Yes	Yes
Pre-war Characteristics	Yes	Yes	Yes

Source: Authors' estimation from the 1991 BiH Census, official electoral statistics and municipality characteristics from the Federal Office of Statistics of Bosnia and Herzegovina. Additional pre-war control variables: per-capita income, student/teacher ratio, population, ethnic shares, surface area, cultivated land (%), strategic importance, ethnic polarization, and distance from Croatia and Serbia. We instrument civilian casualties with the standard deviation of terrain ruggedness. The ruggedness index is calculated as the average change in elevation within a 3x3 pixel grid in each municipality (source: Jarvis et al. (2008)). Standard errors in parentheses are adjusted for heteroskedasticity. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table 3.11: Individual-level results: voting, trust, income (LITS-I, 2006)

	(1)	(2)	(3)	(4)	(5)	(6)
	Vote		Generalized Trust		Trust in Institutions	
Civilian Casualties	-0.157***	-0.250***	-0.139***	-0.210**	-0.154***	-0.230**
	(0.0136)	(0.0665)	(0.0454)	(0.0863)	(0.0381)	(0.0888)
Income (pre-war)		-0.0197		-0.0612		-0.135**
		(0.0481)		(0.0366)		(0.0508)
Generalized Trust (pre-war)			0.00619	0.0624		
			(0.136)	(0.105)		
Observations	697	697	697	697	697	697
R^2	0.025	0.160	0.020	0.337	0.024	0.259
Indiv. Char.	N	Y	N	Y	N	Y
Munic. Char.	N	Y	N	Y	N	Y

Source: Authors' estimations from LITS-I (2006), using survey weights. Trust, voting, and casualties variables are standardized with mean 0 and standard deviation 1. Voting refers to whether individual has voted in the last presidential or parliamentary election. Generalized trust ranges between 1 and 5 (1 "complete distrust" 2 "some distrust" 3 "neither trust nor distrust" 4 "some trust" 5"complete trust"). Trust in institutions is an average of: trust in the president, the parliament, the political parties and the ministers. Income is self-reported, ranking between 0 and 10. Individual characteristics include: age, gender, educational attainment, employment, ethnic group, marital status, number of children in the household, urban/rural, self-reported health, current party membership, communist party membership before 1990, whether respondent if of an ethnic minority, ever fought in military, internal displacement before 1996. Baseline municipality characteristics (1991) include: log (per-capita income), ethnic polarization, ethnic shares, population, strategic importance (Weidmann, 2011), student/teacher ratio, distance to Croatia and Serbia, share of land used for cultivation. All regressions include military casualties and houses damaged (% of pre-war population). Standard errors in parentheses are clustered at the municipality level. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table 3.12: Individual-level results, heterogeneity by age (LITS-I, 2006)

	(1) Vote	(2) Trust People	(3) Trust Institutions
Civilian Casualties	-0.235***	-0.237***	-0.248***
	(0.0678)	(0.0844)	(0.0862)
Young Age (< 17 during war)	-0.432***	0.0633	-0.0127
	(0.139)	(0.0995)	(0.130)
Young Age * Civilian Casualties	0.0934	-0.00387	0.0224
	(0.174)	(0.0758)	(0.108)
Observations	697	697	697
R^2	0.159	0.326	0.256
Indiv. Char	Y	Y	Y
Munic. Char.	Y	Y	Y

Source: Authors' estimations from LITS-I (2006). Outcome and treatment variables are standardized. Individual and pre-war municipality characteristics included (see footnote of Table 3.11 for details on outcome and controls). Standard errors in parentheses are clustered at the municipality level. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table 3.13: Non-selective targeting of individuals (LITS-I, 2006)

	Civilian Casualties			
Generalized Trust (pre-war)	-0.0813			-0.0725
	(0.0785)			(0.0596)
Member of Communist Party (pre-war)		-0.0775		-0.0220
		(0.127)		(0.0820)
Income Rank (pre-war)			-0.0701	-0.0606
			(0.0648)	(0.0531)
Observations	697	697	697	697
R^2	0.019	0.001	0.050	0.170
Age and Gender	N	N	N	Y

Source: Authors' estimates from LITS-I (2006) and BBD (2008). Outcome variable civilian casualties 1992-1995 at the municipality level over pre-war population. Standard errors clustered at the municipality level in parentheses. All estimates include survey weights. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Appendix B

Table B-1: DiD: turnout all elections

Dependent Variable:	(1)	(2)	(3)	(4)	(5)	(6)
$Turnout_t - Turnout_{1990}$	Local Elections			General Elections		
$t =$	2004	2008	2012	2006	2010	2014
	Panel A: No Controls					
Civilian Casualties	-2.398**	-1.714**	-2.525***	-1.802**	-2.135***	-1.765**
	(1.034)	(0.717)	(0.750)	(0.693)	(0.714)	(0.769)
Observations	127	127	127	127	127	127
R^2	0.0438	0.0553	0.152	0.0748	0.0926	0.112
	Panel B: Pre-War Characteristics					
Civilian Casualties	-3.008***	-3.025***	-3.606***	-2.384***	-3.420***	-3.070***
	(1.129)	(0.823)	(0.817)	(0.793)	(0.770)	(0.720)
Observations	127	127	127	127	127	127
R^2	0.450	0.360	0.408	0.331	0.424	0.426
	Panel C: Pre and Post-War Characteristics					
Civilian Casualties	-2.598**	-4.148***	-3.803***	-2.533***	-3.168***	-2.853***
	(1.235)	(0.878)	(0.851)	(0.870)	(0.811)	(0.794)
Observations	127	127	127	127	127	127
R^2	0.481	0.525	0.642	0.417	0.494	0.543
Mean Dep. Variable	-31.28	-19.09	-18.26	-22.37	-21.21	-23.63
Std Dev. Dep. Variable	12.72	10.45	11.38	9.254	9.507	10.61

Source: Authors' estimation from BBD (2008), 1991 BiH Census, official electoral statistics (FOS-BiH), and geographic data (see Section 3.3). Casualties are in % of pre-war population by municipality. Military casualties included in every regression but not displayed. Pre-war characteristics: log (per capita income), ethnic polarization, ethnic shares, population, strategic importance (Weidmann, 2011), student-teacher ratio, distance to Croatia and Serbia, and share of cultivated land in 1991. Post war characteristics, for the available years: % of damaged houses, % repaired houses by 2005, war displacement (total emigration in 1995), Entity, municipality partition, unemployment rate (2006, 2008, 2010), average net wage (2006, 2008, 2010), population difference $t - 1991$ (2008, 2010, 2012), Ethnic polarization difference (2012, 2014), student-teacher ratio difference (2014), net emigration (2010, 2012). Standard errors in parentheses and adjusted for heteroskedasticity. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table B-2: War and turnout: spatially correlated standard errors

	(1) 2004	(2) 2008	(3) 2012	(4) 2006	(5) 2010	(6) 2014
Civilian Casualties	-3.008**	-3.025***	-3.606***	-2.384***	-3.420***	-3.070***
	(1.222)	(0.561)	(0.627)	(0.703)	(0.680)	(0.737)
Observations	127	127	127	127	127	127
Mean Dep. Variable	-31.28	-19.09	-18.26	-22.37	-21.21	-23.63
Std Dev. Dep. Variable	12.72	10.45	11.38	9.254	9.507	10.61

Source: Authors' estimation from BBD (2008), 1991 BiH Census, official electoral statistics (FOS-BiH), and geographic data (see Section 3.3). Casualties are in % of pre-war population by municipality. Military casualties included in every regression but not displayed. Pre-war characteristics included in the regressions for all years: log (per capita income), ethnic polarization, ethnic shares, population, strategic importance (Weidmann, 2011), student-teacher ratio, distance to Croatia and Serbia, and share of cultivated land in 1991. Standard errors are adjusted using Hsiang's (2010) Stata code following Conley (1999) to allow for spatial correlation across municipalities. Distances are calculated from centroids of municipalities. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table B-3: War and turnout: heterogeneous effects by sex, 2006

	(1) Turnout Male	(2) Turnout Female	(3) Turnout All	(4) Turnout Male-Female	(5) Registered Male/Female
Civilian Casualties	-2.774***	-2.132***	-2.384***	-0.642	-0.0121
	(0.853)	(0.788)	(0.793)	(0.398)	(0.0102)
R^2	0.339	0.316	0.331	0.155	0.330
Observations	127	127	127	127	127
Mean Dep. Variable	-21.04	-23.77	-22.37	2.726	1.071
Std Dev. Dep. Variable	9.127	9.783	9.254	3.688	0.115

Source: Authors' estimation from BBD (2008), 1991 BiH Census, official electoral statistics (FOC-BiH), and additional sources of baseline controls (see Section 3.3). Civilian Casualties indicate dead or missing individuals by municipality of origin, as a share of pre-war population. All regressions include military casualties as a separate regressor. In columns (1) to (3), the outcome variable is the difference in turnout between 2006 (group specific) and 1990 (for all voters). column (1) shows results for male voters, column (2) for female voters, and column (3) for all voters. The dependent variable in column (4) is the difference between male and female voter turnout. The dependent variable in column (5) is the ratio between male and female registered voters. We do not display the same estimates for other elections because this information is only available for 2006. Standard errors in parentheses and adjusted for heteroskedasticity. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table B-4: Civil conflict and turnout: OLS (outcome variable in levels)

	Local Elections			General Elections		
	(1)	(2)	(3)	(4)	(5)	(6)
Dependent Variable: Turnout						
	2004	2008	2012	2006	2010	2014
Panel A: OLS						
Civilian Casualties	-1.250	-1.266*	-1.847**	-0.625	-1.662**	-1.312*
	(1.072)	(0.757)	(0.737)	(0.681)	(0.705)	(0.693)
R^2	0.402	0.482	0.442	0.300	0.405	0.410
Observations	127	127	127	127	127	127
Panel B: OLS, with Turnout 1990 (Pre-war Characteristics)						
Civilian Casualties	-1.228	-1.199	-1.956**	-0.911	-2.125***	-1.763**
	(1.109)	(0.778)	(0.760)	(0.722)	(0.725)	(0.681)
Turnout in 1990	-0.0122	-0.0382	0.0619	0.163	0.263***	0.257**
	(0.118)	(0.108)	(0.114)	(0.104)	(0.0990)	(0.115)
R^2	0.402	0.482	0.443	0.312	0.432	0.429
Observations	127	127	127	127	127	127
Panel C: OLS, with Turnout 1990 (Pre and Post-war Characteristics)						
Civilian Casualties	-0.608	-2.144**	-2.265***	-0.950	-1.794**	-1.477**
	(1.146)	(0.927)	(0.797)	(0.857)	(0.768)	(0.734)
Turnout in 1990	-0.0650	0.0741	0.246**	0.192	0.300***	0.315***
	(0.126)	(0.112)	(0.119)	(0.119)	(0.109)	(0.109)
R^2	0.469	0.597	0.642	0.383	0.478	0.544
Observations	127	127	127	127	127	127
Mean Dep. Variable	48.37	60.56	61.39	57.27	58.44	56.02
Std Dev Dep. Variable	10.19	8.973	9.808	7.523	8.162	9.377

Source: Authors' estimation from BBD (2008), 1991 BiH Census, official electoral statistics (FOC-BiH), and additional sources of geographic data (see Section 3.3). The dependent variables are levels of turnout in each post-war year. Casualties are in % of pre-war population by municipality. Pre-war controls include for all years: log income per capita in 1991, ethnic polarization 1991, pre-war ethnic shares, population, strategic importance 1991 (Weidmann, 2011), student-teacher ratio 1991, distance to Croatia and Serbia, share of land used for cultivation. Standard errors in parentheses and adjusted for heteroskedasticity. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

In the following Table, we show that the probability of not answering survey questions on social capital (vote, trust, income) is not significantly related to war intensity experienced at the municipality level. We run the regression on the full sample of LITS-I (2006) respondents. Including survey weights and clustering at the primary sampling unit does not alter the results (available upon request).

Table B-5: Individual level (LITS 2006): non-response and conflict intensity

	(1)	(2)	(3)	(4)
Outcome: Missing Answer on	Vote	Trust Institutions	Tust Post-war	Trust Pre-war
Civilian Casualties	-0.00110	-0.000790	-0.00322	-0.0180
	(0.000937)	(0.00381)	(0.00730)	(0.0153)
Constant	0.00700	0.0211	0.0250	0.185***
	(0.00634)	(0.0148)	(0.0261)	(0.0588)
Observations	1000	1000	1000	1000
R^2	0.006	0.022	0.048	0.049
% of missing answers:	9.27	10.18	12.82	9.73

Source: Authors' estimated from LITS-I (2006) and BBD (2008). Additional controls: age group, gender, marital status, employment status, self-reported Health Status, number of children in the household, urban-rural-metropolitan residency, rural-urban migration (and viceversa, before 1996). Standard errors clustered at the municipality level in parentheses. Asterisks denote statistical significance at the 1(***), 5(**) or 10(*) percent level.

Table B-6: Party landscape evolution: 1990, 1996, 2006

1990	1996	2006
Panel A: Parties continuously represented		
Party of Democratic Action (SDA)	→ Party of Democratic Action (SDA)	→ Party of Democratic Action (SDA)
	Party for BiH (SBiH)	→ Party for BiH (SBiH)
Serb Democratic Party (SDS)	→ Serb Democratic Party (SDS)	→ Serb Democratic Party (SDS)
Croatian Democratic Union (HDZ)	→ Croatian Democratic Community BiH (HDZ-HNZ)	→ Croatian Democratic Community BiH (HDZ-HNZ)
		↘ Croatians Together (HDZ 1990)
League of Communists-Social Democratic Party (SK-SDP)	→ Joint List (joint of SDP, UBSD, Croatian Peasant Party, MBO and the Republican Party)	→ Social Democratic Party of Bosnia and Herzegovina (SDP)
Democratic Socialist Alliance (DSS)		→ Democratic People's Alliance (DNS)
Panel B: Parties entering/exiting over time		
Alliance of Reformist Forces of Yugoslavia (SRSJ)	People's Alliance for Free Peace (NSSM)	Party of Independent Social Democrats (SNSD)
Alliance of Socialist Youth-Democratic Alliance (SSO-DS)		Party of Democratic Progress (PDP)
Muslim Bosniak Organization (MBO)		Patriotic Party (BPS Sefer Halilovic)
		People's Party Working for Prosperity (NS)
		Democratic People's Community (DNZ)
		Democratic People's Alliance (DNS)

Data sources: elections 1990 Karic (2011), 1996 Kasapović (1997), 2006 Central Election Commission BiH (izbori.ba). Panel A displays parties continuously represented in the House of Representatives or formed from previously running parties. Panel B shows parties entering and exiting House of Representatives over time.

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