Essays in Health and Development Economics

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To my parents for their endless love.

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Abstract

In the first chapter, I analyze the consequences of increased prenatal exposure to marijuana on infant health in the US. I combine individual-level information on birth outcomes with novel data on the location and opening dates of all cannabis dispensaries in the US. Results indicate that increased prenatal exposure to cannabis is unrelated to several infant health indicators. In the second chapter, we study the role of pharmaceutical promotion in the opioid epidemic in the US. Using data on visits made by sales representatives to physicians, we show that opioid promotion increases prescription of such drugs and also increases opioid overdose mortality. In the third chapter, we evaluate the impacts of an electrification program on student learning in rural Peru. Our findings suggest that improved access to electricity is, at best, weakly related to better student learning.

Resumen

En el primer capítulo, se analizan las consecuencias de una mayor exposición prenatal a la marihuana sobre la salud infantil en Estados Unidos. Utilizando datos sobre la ubicación y fecha de apertura de los dispensadores de cannabis, se muestra que mayor exposición prenatal a la marihuana no genera cambios significativos en la salud de los recién nacidos. En el segundo capítulo, se estudia el rol de la publicidad farmacutica en la epidemia de opiáceos en Estados Unidos. Se encuentra que los condados con mayor nivel de publicidad de opiáceos también tienen mayores tasas de mortalidad causadas por sobredosis de estas drogas. En el tercer capítulo, se evalúan los impactos de un programa de electrificación rural sobre el aprendizaje escolar en Perú. Los resultados indican que, en el mejor de los casos, un mejor acceso a electricidad tiene un efecto reducido en el desempeo estudiantil.

Preface

In the first chapter, I evaluate the consequences of increased marijuana exposure during pregnancy on infant health in the US. Unlike previous studies on the impacts of marijuana, which rely on state-level variation to identify their effects of interest, I exploit county-specific measures of cannabis prenatal exposure using data on the precise location and opening date of every cannabis dispensary (legal point of sale for marijuana) in the country. Estimations based on state-level measures of increased marijuana access suggest no adverse impact on infant health. In addition, the estimated effects exploiting county-level variation in the opening dates of cannabis dispensaries, suggest that higher prenatal exposure to cannabis is unrelated to changes in infant health, once I control for county fixed effects and state-specific trends. Additional evidence from an event-study analysis with similar controls, corroborates that increased availability of marijuana during pregnancy is not linked to changes in infant health.

In the second chapter, co-authored with Dijana Zejcirovic, 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 final chapter, co-authored with Hugo Nopo and Rosamaría Dasso, we evaluate the impact of improved access to electricity on student learning in Peru. During the past decade, the central government implemented an electrification program that rapidly increased electricity coverage in rural districts. Exploiting spatial and temporal variation in access to electricity induced by this large-scale intervention, we report heterogeneous effects on student learning. Using panel data on national standardized tests over 2007-2015, we find that the average impact of the intervention is not statistically different from zero. However, among treated schools, our results indicate that longer treatment exposure increases scores in Reading and Math for both male and female students. Based on these estimates, we speculate that in the long-run, the net impact of better access to electricity on student learning can be positive. Finally, we show that our results are not being driven by two confounding interventions that took place during the study period.

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Chapter 1

PRENATAL EXPOSURE TO MARIJUANA AND INFANT HEALTH IN THE US

1.1 Introduction

The number of state governments that legalized cannabis for medical purposes proliferated over the last decade. By the end of 2018, 33 states and Washington DC had approved medical marijuana laws (MMLs), which aim to provide patients with more treatment options for conditions such as chronic pain, mental health problems and cancer. This rapid expansion in marijuana availability has been accompanied by growing concerns from public health authorities. The public debate on this topic is fierce because both potential benefits and risks of marijuana legalization remain unclear.

In 2016, to inform this debate, the National Academy of Sciences, Engineering and Medicine gathered a panel of experts to start working on a comprehensive report to collect and analyze the available evidence on the health consequences of cannabis. After a year of work, the report was released in 2017 and its main conclusion was that there is insufficient evidence on the benefits and harms of this drug on a wide range of outcomes including cancer, respiratory diseases, cardiovascular risk, mental health, injury and death (National Academy of Sciences, 2017). Furthermore, the report identifies a research gap on the health impacts of cannabis use on pregnant women and infants. These vulnerable groups are of special interest because recent figures suggest that marijuana is the most common drug used during pregnancy (Volkow et al. 2017). To shed light on this issue, I explore whether increased access to marijuana can affect infant health through higher exposure to marijuana coming directly from maternal use or, indirectly, from other family members or close neighbors.

Pregnant women may use marijuana to reduce pain or nausea, two of its allegedly medical benefits. How can this use affect infants' health? It is a fact that the chemical components of marijuana (in particular tetrahydrocannabinol or THC) pass from the mother to the fetus through the placenta. Several observational studies in medicine show that prenatal exposure to cannabis diminishes fetal growth, and this reduction leads to lower birthweight and brain development problems. Based on the available evidence, current health guidelines (e.g. Center for Disease Control and Prevention, the American College of Obstetricians and Gynecologists) recommend pregnant women not to consume medical cannabis and to avoid being close to marijuana smoke. However, as pointed out in two recent systematic reviews (Conner et al. 2016 and Gunn et al. 2016), these correlations should be taken with caution because they could be capturing the effects of other related and harmful behaviors such as tobacco or alcohol use.

To further investigate this open question, I exploit geographic and temporal variation in increases of marijuana availability to estimate the consequences of higher prenatal exposure to cannabis on infant health. First, I combine data on the introduction of MMLs with a restricted version of the birth files from the National Vital Statistics System over 2004-2014. The differential timing of the enactment of MMLs across states allows me to link infant health indicators with increased availability of medical marijuana. The results from this specification suggest that prenatal exposure to legal marijuana has no significant impact on five health indicators (e.g. pre-term birth, respiratory problems at birth, admission to the intensive care unit). The only significant point estimate implies that increased access to marijuana during pregnancy rises the prevalence of low birth-weight (babies born with less than 2500 grams) by 5 percent.

Then, I construct county-specific measures of prenatal exposure to marijuana using novel data on the precise location and opening date of cannabis dispensaries in the US. These dispensaries are the legal point of sale of marijuana for final consumers. The estimated impacts exploiting county-level variation also indicate that increased prenatal exposure to cannabis has no significant effects on any of the six infant health indicators analyzed.

Finally, I estimate changes in infant health outcomes after the opening of cannabis dispensaries using an event-study framework. These estimates, using a restricted sample of births (only those occurring within a narrow window time around the opening date), also show that higher in-utero exposure to cannabis is unrelated to several adverse infant health outcomes. Taken together, these ITT effects suggest that increased marijuana availability during pregnancy is, at best, only weakly related to worse infant health. In nearly all cases, the coefficients are precisely estimated.

The main limitation of this analysis is the lack of a measure on actual (or even self-reported) marijuana use. For this reason, all these coefficients should be interpreted as Intent-To-Treat (ITT) effects. Treatment on the treated (TOT) effects could be quite larger (in absolute terms), depending on the prevalence of cannabis use. Although medical studies coincide on indicating that the prevalence of marijuana use has increased in recent years, there are important differences in the levels they report. National-level estimates show that the prevalence of marijuana use during pregnancy ranges from 2 % to 5% (Volkow et al 2017, but these figures can reach 15-20% among urban, young women (ACOG 2017, Mark et al 2015). Nonetheless, the ITT effects reported here are informative about the consequences of increased marijuana access on the residents of counties where dispensaries have opened.

This paper adds to the small but growing economic literature on the effects of MMLs. A number of recent studies have documented the effects of MMLs on consumption of marijuana, tobacco, alcohol and harder drugs. The emerging evidence suggests that MMLs are related to increases in marijuana and alcohol use, decreases in tobacco consumption, and have no impact on the use of harder drugs (Wen et al. 2014; Choi et al. 2016; Anderson et al. 2014). Powell et al. (2018) also find that MMLs reduce addiction and deaths related to prescription opioid drugs (painkillers). In addition to these health impacts, one of the potential economic benefits of these laws is that, as patients get better treatment for pain conditions, they are also more likely to work. Nicholas and Maclean (2016) show that MMLs increase the labor supply of older adults, suggesting that medical marijuana can be helpful to improve labor market participation among older adults. In short, this growing body of evidence shows both positive and negative impacts of MMLs on the health status of teenagers and adults. To the best of my knowledge, this is the first study analyzing the consequences of increased marijuana availability on infant health using county-level variation from dispensary data.

The rest of the paper proceeds as follows. Section 2 describes the data sources. Section 3 details the empirical framework. Section 4 presents our results and Section 5 offers concluding remarks.

1.2 Data

The primary data source for the empirical analysis is the National Vital Statistics System. In particular, I use the restricted version of the birth files for the period 2004-2014. These data include rich information on both maternal and infant characteristics for the universe of first-born births occurred in the US. In a given year, first-born babies account for 40 percent of all births in the country.

I consider six measures of adverse infant health. First, we have an indicator for pre-term birth, which is equal to one if the gestational period was less than 37 weeks, and zero otherwise. Second, an indicator for low birthweight, which is equal to one if birthweight is below 2500 grams, and zero otherwise. Third, a dummy variable indicating whether the child had seizures during birth or not. Fourth, an indicator of whether the newborn had a low APGAR score. This score goes from zero to ten and it summarizes the health status of infants based on five dimensions: appearance, pulse, grimace, activity and respiration. Higher scores mean better health at birth. This variable is equal to one if the APGAR score is below seven (as indicated by medical guidelines), and zero otherwise. Fifth, a dummy variable indicating whether the child needed assisted ventilation (due to respiratory problems during the first hours of life) or not. Sixth, we have an indicator variable if the infant was admitted to the neonatal intensive care unit.

As control variables, we have information on a range of socioeconomic and health characteristics. More specifically, I control for prenatal care, indicators for maternal race, educational level, US-born (native or immigrant), marital status, age groups, and delivery method (vaginal or cesarean section) and month of birth. Both outcome and control variables are taken to follow previous medical studies on the health effects of prenatal exposure to marijuana (Conner et al. 2016, Gunn et al. 2016).

On average, the number of observations with non-missing values for both outcome and control variables is 6 million, except for the indicator of admission to the neonatal intensive care unit, which has 2.7 million observations¹.

Marijuana availability steadily increased over the last decade because several states approved its use for medical purposes, as shown in Figure 1.1. Though this state-level variation is illustrative, the core of the empirical analysis exploits county-level variation in higher access to marijuana.

To construct a novel county-specific measure of marijuana access, I collected

¹This variable is not available for all states during the entire study period. Around half of the states began including this measure already in 2004, but some states started later.

data on the precise location and opening date of every cannabis dispensary in the US. These dispensaries, regulated by local governments, represent the primary legal point of sale of cannabis for final consumers². These data were extracted from www.marijuanadoctors.com and www.weedmaps.com between August and November 2018. I first gathered information on the address of every dispensary, and then looked, one-by-one, for the opening date (month and year). In total, 217 dispensaries opened between 2002 and 2018. From these, 128 opened during our study period (one in a different county, implying that there are 128 counties with one cannabis dispensary). The number of dispensaries opened by year is shown in Figure 1.2. We see that very few dispensaries opened before 2009, but in that year, and afterwards, a growing number of dispensaries opened.

1.3 Empirical Framework

The question of interest is whether prenatal exposure to increased marijuana access has an impact on infant health. As a starting point, I exploit the timing across states in the introduction of MMLs, linking changes in infant health to differences in the availability of medical marijuana induced by policy changes. More formally, I estimate the following equation:

$$AH_{ist} = \alpha_s + \alpha_t + \beta M M L_{st} + X'_i \Psi + \mu_{ist}$$
(1.1)

where AH_{ist} is a measure of adverse health of child *i*, in state *s* in period *t*. State and year fixed effects are denoted by α_s and α_t , respectively. The variable MML_{st} represents a state-level measure of increased prenatal exposure to cannabis. It indicates that access to medical marijuana was legalized in state *s* before children were born in year t^3 . The vector X_i includes individual characteristics. The error term is denoted by μ_{ist} , and it is allowed to be correlated within states.

In equation (1), the parameter of interest is β . The identification assump-

²Local governments can only authorize the opening of a dispensary after the state has already approved marijuana use.

³In practice, the variable is defined using the year of conception (calculated with the month and year of birth) but, for simplicity, I only refer to year t.

tion required for obtaining a consistent estimate of the impact of MMLs is that once I control for unobserved time-invariant characteristics at the state-level, yearspecific effects common across all individuals, and individual characteristics, the timing of MMLs across states unrelated to unobserved determinants of infant health. In this setup, the main threat to identification is that there could be statespecific factors that vary over time and correlate with MMLs and infant health. This would imply that β is also capturing the effects of these confounding factors. Another concern, pointed out in previous studies (Hunt et al 2018), is that statelevel measures of marijuana access may preclude identification of small effects (in magnitude) or impacts that are local in nature because the estimation ignores within-state variation.

To overcome this limitation, the core of the empirical analysis will be the following regression:

$$AH_{isct} = \lambda_c + \lambda_t + \lambda_s * t + \delta^{DD} dispensary_{ct} + X'_i \Phi + \varepsilon_{isct}$$
(1.2)

where λ_c denotes county fixed-effects, $\lambda_s * t$ represents state-specific time trends, dispensary_{ct} indicates that there is a cannabis dispensary in county c in period t, and the remaining terms are defined as before. In equation (2), the error term is allowed to be correlated within counties. Now, the parameter of interest is δ^{DD} , which captures the change in adverse health indicators following the opening of a cannabis dispensary, controlling for unobserved county-level confounders that are time-invariant, and state-level trends. The medical literature suggests that δ^{DD} should be positive. These adverse health impacts arise for two channels: maternal use of marijuana during pregnancy (direct effect) or prenatal exposure to marijuana smoke (indirect effect or externality).

The inclusion of both state-level trends and county fixed-effects implies that the coefficient δ^{DD} is estimated using only trend breaks that precisely coincide with the opening of cannabis dispensaries, after removing time-invariant county heterogeneity. This means that once I control for $\lambda_s * T$ and λ_c , the main threat to the estimation of δ^{DD} is that confounding factors, not captured by county fixedeffects, generate deviations from state-specific trends that occur on the opening dates of cannabis dispensaries. To complement the Differences-in-Differences estimation, I exploit variation in the opening dates of cannabis dispensaries in an event-study framework to estimate the change in infant health following increases in marijuana access. To fix ideas, let us consider the following equation:

$$AH_{isct} = \pi_c + \pi_t + \pi_s * time + \delta^{ES} 1(t > t_c^{OP}) + X_i' \Phi + \nu_{isct}$$
(1.3)

where t_c^{OP} is the date on which the cannabis dispensary opened in county c. In this framework, δ^{ES} captures the change in the outcome following the event (opening of the dispensary). The key assumption behind this specification is that the difference between birth dates and opening dates of cannabis dispensaries is exogenous to infant health, after controlling for county and year fixed effects, state-specific trends, and individual characteristics. To explore the stability of the event-study results, I restrict the sample of births using three different time intervals (in days): [t - 280; t + 280]; [t - 150; t + 150], and $[t - 90; t + 90]^4$. Because I only observe the month and year of the opening date, I assume that all dispensaries opened the first day of the month⁵.

1.4 Results

1.4.1 Differences-in-Differences (DD) Results

Table 1.1 presents the DD estimates (ITT effects) of β of equation (1). The estimated coefficients are very small in magnitude and, for all outcomes but one, I cannot reject the null hypothesis of no impact (precisely estimated zero effects). These results suggest that MMLs are not associated with changes in the prevalence of pre-term births (less than 37 weeks), seizures, low APGAR scores, respiratory problems or admissions to the neonatal intensive care unit. I only find a statistically significant effect on the prevalence of low birthweight (below 2,500 gr). The point estimate in column (2) suggests that MML increases the prevalence of low birthweight by 0.39 percentage points. This impact represents an increase of

⁴These time intervals are chosen to roughly allow for one, two or three trimesters of prenatal exposure to cannabis dispensaries (in reverse order).

⁵Results do not change if I use alternative dates such as the fifteenth day of the month.

5 percent relative to the mean of the low birthweight rate (0.0735). As stated before, we may suspect that these estimates do not capture the full effect of prenatal exposure to cannabis because they ignore county-level variation (within the same state) in access to marijuana.

For this reason, we now turn our attention to the ITT estimates reported in Table 1.2, which exploit county-specific measures of prenatal exposure to marijuana. In column 1, I report the coefficients of the DD specification controlling for individual characteristics and year fixed effects. In column 2, state-specific trends are included in the model. Finally, in column 3, I control for unobserved county heterogeneity that is time-invariant. The mean of the dependent variable, and the number of observations are shown in the last two columns.

The estimated impacts of higher marijuana access on the occurrence of preterm births are positive and statistically significant in the first two columns but become insignificant once I include county fixed effects (in column 3). The same pattern (statistically significant in the first two columns but insignificant in the third) is found for the estimated effects on the prevalence of low birthweight and admission to the neonatal intensive care unit. We should note that this loss of statistical significance is not driven by larger standard errors (indeed they are smaller) but by a large reduction in the size of the coefficients. The point estimates associated with the impacts on low APGAR scores and assisted ventilation are insignificant across all specifications. The only effect that is statistically significant in the three models indicates that higher exposure to cannabis is related to increases in the likelihood of seizures. However, there is no clear biological mechanism nor prior evidence for such effect. Overall, it seems important to control for timeinvariant county-level characteristics because, after doing so, most ITT effects are precisely estimated zero coefficients.

1.4.2 Event-Study Results

To complement the DD results, I report the estimates from an event-study framework (equation 3) with three different windows time: 280 days, 150 days and 90 days. Table 1.3 presents the estimated coefficients for the broadest time window, which allows for 40 weeks of prenatal exposure to cannabis. We see that, again, all point estimates are not statistically different from zero. Thus, these results suggest that there is no discernible change in infant health after the opening of cannabis dispensaries.

In Tables 1.4 and 1.5 I further restrict the sample to narrower window times (150 and 90 days, respectively). In Table 1.4, we see that most estimated impacts are not statistically significant. Only the point estimates in columns 2 and 5 are significant, suggesting that higher prenatal exposure to cannabis is associated with higher prevalence of low birthweight and fewer newborns needing assisted ventilation. In Table 1.5, the only estimated coefficient that is significant (column 6) indicates that after the opening of cannabis dispensaries in a given county, there is an increase in the fraction of infants that are admitted to neonatal intensive care unit. In the rest of the columns, the estimated effects suggest that there are no changes in infant health after the opening of cannabis dispensaries.

1.5 Conclusion

In recent years, marijuana availability has rapidly increased throughout the US. This trend in cannabis legalization has also raised concerns among scholars and policy makers alike. The report made by the National Academy of Sciences, the most comprehensive study on this topic, concludes that much research remains to be done in order to fully understand the health impacts of cannabis.

In this paper, I focus on the consequences of increased prenatal exposure to cannabis on infant health. Similar to previous studies in the economic literature, I first use a differences-in-differences model exploiting state-level changes in prenatal exposure to cannabis generated by the enactment of medical marijuana laws. I find that these state-level policy changes are unrelated to several infant health indicators (precisely estimated zero coefficients).

Then, using the same differences-in-differences framework, I exploit variation in the opening dates of cannabis dispensaries, controlling for state-specific trends and time-invariant county heterogeneity. Without such controls, the estimated effects of increased marijuana availability are large and statistically significant. But after including county fixed effects, most coefficients become insignificant. This loss of significance is driven by smaller point estimates instead of less precision (in fact, standard errors are smaller). Additional evidence from an event-study framework (with three different window times) using only births occurring in counties with cannabis dispensaries also suggests that several infant health indicators are unaffected after the opening of such dispensaries.

Despite the lack of conclusive evidence, most health authorities (e.g. American Medical Association, National Institute of Health, American College of Obstetricians and Gynecologists) recommend against marijuana use during pregnancy pointing out that there is substantial theoretical work on the harmful consequences that cannabis can have on fetal growth and brain development.

Given that experimental studies are not feasible because of ethical reasons, this study represents a first step in providing more credible evidence on the impacts of prenatal cannabis exposure on infant health. In line with two systematic medical reviews, my findings suggest that increased marijuana availability during pregnancy is unrelated to several infant health indicators, once I control for county fixed-effects and state-specific trends. One important drawback in this analysis is that I do not observe maternal marijuana use directly and, therefore, all these estimates are ITT effects, not the impacts on actual cannabis users. As more data on marijuana use become available, future work should explore the health impacts on pregnant women who really consume cannabis.

Tables and Figures



Figure 1.1: Year of Medical Marijuana Legalization by state



Figure 1.2: Timing of the opening of cannabis dispensaries: 2004-2014

Denendent variahle.	Pre-term Rirth	I ow	Seizures	I ow	Assisted	Admission
	(less than 37 wks)	Birthweight		APGAR Score	Ventilation	to NICU
	(1)	(2)	(3)	(4)	(5)	(9)
Medical marijuana law	0.0019	0.0039^{***}	0.0001	0.0038	-0.0024	-0.0007
	(0.0034)	(0.0015)	(0.0002)	(0.0029)	(0.0044)	(0.0032)
	;	;	;	;	;	,
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
State FE	Yes	Yes	Yes	Yes	Yes	Yes
Observations	6,072,141	6,075,885	6,069,031	5,942,948	6,069,031	2,734,626
R-squared	0.0090	0.0107	0.0005	0.0064	0.0292	0.0172
Mean of dep. variable	0.181	0.0735	0.0002	0.0191	0.0177	0.0801

Table 1.1: DD Estimates of the Impacts of state-level MML on Adverse Infant Health

regressions control for individual characteristics, state and year fixed-effects. All dependent variables are discrete. Each outcome is equal to one if NOTE: Robust standard errors clustered at the state level are shown in parentheses. Each coefficient comes from a separate regression. All the stated adverse condition occurred and zero otherwise (NICU means: Neonatal Intensive Care Unit).

Variable: (1) (2) (3) Mean N Pre-term birth (less than 37 weeks) 0.0059*** 0.0067*** -0.0005 0.181 6,072,141 (less than 37 weeks) 0.0077 0.0078 0.0126 0.0171 0.0073 6,075,885 Low Birthweight (below 2500 gr) 0.0063** 0.0062** 0.0006 0.0735 6,075,885 Kelow 2500 gr) (0.0025) (0.0027) (0.0011) 0.0002* 0.0002* 0.0002 R-squared 0.0003** 0.0003** 0.0002* 0.0002 6,069,031 R-squared 0.0005 0.0006 0.0012 0.0191 5,942,948 Score (0.0017) 0.0006 0.0020 0.0191 5,942,948 score (0.0015) (0.0014) (0.0015) 0.0099 0.0177 6,069,031 Ventilation (0.0044) (0.0034) (0.0050) 0.0177 6,069,031 Ventilation 0.0186*** 0.0185*** 0.0019 0.2,734,626 to NICU (0.0059)	Dependent					
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	Variable:	(1)	(2)	(3)	Mean	Ν
Pre-term birth (less than 37 weeks) 0.0059^{***} 0.0067^{***} -0.0005 0.181 $6,072,141$ (less than 37 weeks) R-squared 0.0022) 0.0077 (0.0023) 0.0078 (0.0017) 0.0126 0.0017 Low Birthweight (below 2500 gr) 0.0063^{**} (0.0025) (0.0027) 						
(less than 37 weeks) (0.0022) (0.0023) (0.0017) R-squared 0.0077 0.0078 0.0126 Low Birthweight 0.0063^{**} 0.0062^{**} 0.0006 0.0735 $6,075,885$ (below 2500 gr) (0.0025) (0.0027) (0.0011) 0.0735 $6,075,885$ (below 2500 gr) (0.0025) (0.0027) (0.0011) 0.0735 $6,075,885$ Seizures 0.0003^{**} 0.0003^{**} 0.0002^{*} 0.0002 $6,069,031$ R-squared 0.0005 0.0006 0.0012 0.0012 0.0191 $5,942,948$ score (0.0015) (0.0014) (0.0015) 0.0191 $5,942,948$ score (0.0058) 0.0065 0.0099 0.0177 $6,069,031$ R-squared 0.0058 0.0051 0.0080 0.0177 $6,069,031$ Ventilation (0.0061) 0.0051 0.0080 0.0177 $6,069,031$ Ventilation (0.0044) (0.0034) (0.0050) 0.0177 $6,069,031$ R-squared 0.0290 0.0313 0.0432 $2,734,626$ to NICU (0.0059) (0.0060) (0.0014) 0.0348 Individual controlsYesYesYesYesYear FEYesYesYesYesYear FEYesYesYesYesState-specific trendsNoYesYes	Pre-term birth	0.0059***	0.0067***	-0.0005	0.181	6,072,141
R-squared 0.0077 0.0078 0.0126 Low Birthweight (below 2500 gr) 0.0063^{**} (0.0025) 0.0027 (0.0027) 0.00011 (0.0011) 0.0735 $6,075,885$ (0.0011)R-squared 0.0088 0.0089 0.0148 0.0002^* (0.0001) 0.0002^* (0.0001) 0.0002^* (0.0001) 0.0002^* (0.0001)R-squared 0.0003^{**} (0.0005) 0.0002^* (0.0001) 0.0002^* (0.0001) 0.0002^* (0.0001)R-squared 0.0005 (0.0005) 0.0006^* (0.0014) 0.0191^* (0.0015) $5,942,948$ score (0.0015)Low APGAR score 0.0017^* (0.0015) 0.0006^* (0.0014) 0.0191^* (0.0015) $5,942,948$ score (0.0015)R-squared 0.0061^* (0.0058) 0.0065^* (0.0099) 0.0177^* (0.0050) $6,069,031^*$ (0.0050)Assisted ventilation R-squared 0.0061^* (0.00290^* 0.0080^* ($0.0014)^*$ 0.0177^* (0.0080^*)Admission to NICU R-squared 0.0186^{***} (0.0159^*) 0.0160^* ($0.0014)^*$ 0.0801^* ($2,734,626^*$ to NICU R-squared $2,734,626^*$ (0.0159^*)Individual controls Year FE Year FE Year FE Year Yes Year Yes<	(less than 37 weeks)	(0.0022)	(0.0023)	(0.0017)		
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	R-squared	0.0077	0.0078	0.0126		
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	Low Birthweight	0.0063**	0.0062**	0.0006	0.0735	6,075,885
R-squared 0.0088 0.0089 0.0148 Seizures 0.0003^{**} 0.0003^{**} 0.0002^* 0.0002 $6,069,031$ R-squared 0.0005 0.0006 0.0012 0.0002 $6,069,031$ Low APGAR 0.0005 0.0006 0.0012 0.0112 0.0012 Low APGAR 0.0017 0.0006 0.0020 0.0191 $5,942,948$ score (0.0015) (0.0014) (0.0015) 0.0099 0.0191 $5,942,948$ Assisted 0.0061 0.0058 0.0065 0.0099 0.0177 $6,069,031$ Ventilation (0.0044) (0.0034) (0.0050) 0.0177 $6,069,031$ Ventilation 0.0186^{***} 0.0185^{***} 0.0019 0.0801 $2,734,626$ to NICU 0.0159 0.0160 0.0348 0.0014 0.0348 0.0801 $2,734,626$ Individual controlsYesYesYesYesYesYear FEYesYesYesYesState-specific trendsNoYesYes	(below 2500 gr)	(0.0025)	(0.0027)	(0.0011)		
Seizures 0.0003^{**} (0.0002) 0.0003^{**} (0.0001) 0.0002^{*} (0.0001) 0.0002 (0.0001) $6,069,031$ R-squared 0.0005 0.0006 0.0012 0.0002 $6,069,031$ Low APGAR score 0.0017 0.0006 0.0020 (0.0015) 0.0191 $5,942,948$ score R-squared 0.0058 0.0065 0.0099 0.0191 $5,942,948$ Assisted Ventilation R-squared 0.0061 (0.0044) 0.0051 (0.0034) 0.0177 $6,069,031$ Admission to NICU R-squared 0.0186^{***} 0.0159 0.0185^{***} 0.0160 0.0019 0.0348 0.0801 $2,734,626$ Individual controls Year FE State-specific trendsYes NoYes Yes YesYes YesYes Yes Yes	R-squared	0.0088	0.0089	0.0148		
R-squared (0.0002) $0.0005(0.0001)0.0006(0.0001)0.0012Low APGARscore0.0017(0.0015)R-squared0.00170.00580.00200.0014)0.00550.01910.00995,942,9480.0015)AssistedVentilationR-squared0.00610.00200.0044)0.02900.00510.003130.00800.004320.01776,069,031Admissionto NICUR-squared0.0186^{***}0.01590.00190.01600.00190.03480.08012,734,626Individual controlsYear FEState-specific trendsYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYes$	Seizures	0.0003**	0.0003**	0.0002*	0.0002	6,069,031
R-squared 0.0005 0.0006 0.0012 Low APGAR 0.0017 0.0006 0.0020 0.0191 $5,942,948$ score (0.0015) (0.0014) (0.0015) 0.0099 0.0191 $5,942,948$ R-squared 0.0058 0.0065 0.0099 0.0177 $6,069,031$ Assisted 0.0061 0.0051 0.0080 0.0177 $6,069,031$ Ventilation (0.0044) (0.0034) (0.0050) 0.0177 $6,069,031$ R-squared 0.0290 0.0313 0.0432 0.0801 $2,734,626$ to NICU (0.0059) (0.0060) (0.0014) 0.0348 0.0185 Individual controlsYesYesYesYesYear FEYesYesYesYesYear FEYesYesYesYesState-specific trendsNoYesYes		(0.0002)	(0.0001)	(0.0001)		, ,
Low APGAR score 0.0017 (0.0015) 0.0006 (0.0014) $0.00580.0020(0.0015)0.01915,942,948AssistedVentilationR-squared0.0061(0.0044)0.02900.00510.03130.00800.04320.01770.08016,069,0310.0177Admissionto NICUR-squared0.0186^{***}0.01590.0185^{***}0.01600.00140.03480.08012,734,626Individual controlsYear FEState-specific trendsYesYesYesYesYesYesYesYesYesYesYesYesYes$	R-squared	0.0005	0.0006	0.0012		
score (0.0015) (0.0014) (0.0015) R-squared 0.0058 0.0065 0.0099 Assisted 0.0061 0.0051 0.0080 0.0177 6,069,031 Ventilation (0.0044) (0.0034) (0.0050) 0.0177 6,069,031 Ventilation (0.0044) (0.0034) (0.0050) 0.0177 6,069,031 Admission 0.0186*** 0.0185*** 0.0019 0.0801 2,734,626 to NICU (0.0059) (0.0060) (0.0014) 2,734,626 Individual controls Yes Yes Yes Year FE Yes Yes Yes State-specific trends No Yes Yes	Low APGAR	0.0017	0.0006	0.0020	0.0191	5,942,948
R-squared 0.0058 0.0065 0.0099 Assisted 0.0061 0.0051 0.0080 0.0177 6,069,031 Ventilation (0.0044) (0.0034) (0.0050) 0.0177 6,069,031 Ventilation (0.0044) (0.0034) (0.0050) 0.0177 6,069,031 Admission 0.0186*** 0.0185*** 0.0019 0.0801 2,734,626 to NICU (0.0059) (0.0060) (0.0014) 0.0348 0.0348 Individual controls Yes Yes Yes Yes Year FE Yes Yes Yes Yes State-specific trends No Yes Yes Yes	score	(0.0015)	(0.0014)	(0.0015)		-,,-
Assisted 0.0061 0.0051 0.0080 0.0177 6,069,031 Ventilation (0.0044) (0.0034) (0.0050) 0.0432 0.0432 Admission 0.0186*** 0.0185*** 0.0019 0.0801 2,734,626 to NICU (0.0059) (0.0060) (0.0014) 0.0348 0.0348 Individual controls Yes Yes Yes Yes Year FE Yes Yes Yes Yes State-specific trends No Yes Yes Yes	R-squared	0.0058	0.0065	0.0099		
Ventilation (0.0044) (0.0034) (0.0050) R-squared 0.0290 0.0313 0.0432 Admission 0.0186*** 0.0185*** 0.0019 0.0801 2,734,626 to NICU (0.0059) (0.0060) (0.0014) 0.0348 Individual controls Yes Yes Yes Yes Year FE Yes Yes Yes Yes State-specific trends No Yes Yes Yes	Assisted	0.0061	0.0051	0.0080	0.0177	6.069.031
R-squared 0.0290 0.0313 0.0432 Admission 0.0186*** 0.0185*** 0.0019 0.0801 2,734,626 to NICU (0.0059) (0.0060) (0.0014) 2,734,626 rsquared 0.0159 0.0160 0.0348 Individual controls Yes Yes Yes Year FE Yes Yes Yes State-specific trends No Yes Yes	Ventilation	(0.0044)	(0.0034)	(0.0050)		-,,
Admission 0.0186*** 0.0185*** 0.0019 0.0801 2,734,626 to NICU (0.0059) (0.0060) (0.0014) 0.0348 2,734,626 R-squared 0.0159 0.0160 0.0348 2,734,626 Individual controls Yes Yes Yes Year FE Yes Yes Yes State-specific trends No Yes Yes	R-squared	0.0290	0.0313	0.0432		
Individual controlsYesYesYesYear FEYesYesYesState-specific trendsNoYesYes	Admission	0 0186***	0.0185***	0.0019	0.0801	2 734 626
R-squared0.01590.01600.0348Individual controlsYesYesYesYear FEYesYesYesState-specific trendsNoYesYes	to NICU	(0.0059)	(0,0060)	(0.001)	0.0001	2,75 1,620
Individual controlsYesYesYesYear FEYesYesYesState-specific trendsNoYesYes	R-squared	0.0159	0.0160	0.0348		
Year FEYesYesYesState-specific trendsNoYesYes	Individual controls	Ves	Ves	Ves		
State-specific trends No Yes Yes	Year FE	Yes	Yes	Yes		
	State-specific trends	No	Yes	Yes		
County FE No No Yes	County FE	No	No	Yes		

Table 1.2: DD Estimates of Cannabis Dispensaries on Adverse Infant Health

<u>NOTE</u>: Robust standard errors clustered at the county level are shown in parentheses. Each coefficient comes from a separate regression. All dependent variables are discrete. Each outcome is equal to one if the stated adverse condition occurred and zero otherwise (NICU means: Neonatal Intensive Care Unit). The last two columns display the mean of the dependent variable and the number of observations.

Dependent variable:	Pre-term Birth	Low	Seizures		Assisted	Admission
	(ICSS UIGHL 27 WKS) (1)	DILUIWEIGIII (2)	(3)	AFUAR Scole (4)	(5)	(9)
Change after dispensary	0.0033	0.0031	-0.0001	0.0006	-0.0025	0.0011
has opened	(0.0060)	(0.0027)	(0.0003)	(0.0018)	(0.0027)	(0.0047)
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
County FE	Yes	Yes	Yes	Yes	Yes	Yes
State-specific trends	Yes	Yes	Yes	Yes	Yes	Yes
Observations	142,673	143,001	142,504	130,710	142,504	111,781
R-squared	0.0098	0.0101	0.0023	0.0082	0.0257	0.0279
Mean of dep. variable	0.159	0.0665	0.0004	0.0200	0.0340	0.0897

Table 1.3: Event-study Estimates of Cannabis Dispensaries on Adverse Infant Health. Window time: +-280 days

discrete. Each outcome is equal to one if the stated adverse condition occurred and zero otherwise. NICU means: Neonatal Intensive Care Unit. The sample only includes counties with cannabis dispensaries and is restricted to births occurring between 280 days before or after the opening of the regressions control for individual characteristics, state and year fixed-effects, and state-specific linear time trends. All dependent variables are dispensary. NOTE:]

Danandant wariahla.	Dra_tarm Rirth	I ouv	Saizurae	Iow	Accietad	Admission
Dependent variante.	(less than 37 wks)	Birthweight	SUILEUL CS	APGAR Score	Ventilation	to NICU
	(1)	(2)	(3)	(4)	(5)	(9)
Change after dispensary	-0.0005	0.0069*	0.0001	0.0036	-0.0057**	0.0043
has opened	(0.0074)	(0.0040)	(0.0002)	(0.0038)	(0.0026)	(0.0045)
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
County FE	Yes	Yes	Yes	Yes	Yes	Yes
State-specific trends	Yes	Yes	Yes	Yes	Yes	Yes
Observations	69,134	60,309	69,019	61,718	69,019	52,514
R-squared	0.0105	0.0102	0.0033	0.0082	0.0284	0.0256
Mean of dep. variable	0.160	0.0659	0.0004	0.0206	0.0380	0.0898

Table 1.4: Event-study Estimates of Cannabis Dispensaries on Adverse Infant Health. Window time: +-150 days

The sample only includes counties with cannabis dispensaries and is restricted to births occurring between 150 days before or after the opening of discrete. Each outcome is equal to one if the stated adverse condition occurred and zero otherwise (NICU means: Neonatal Intensive Care Unit). regressions control for individual characteristics, state and year fixed-effects, and state-specific linear time trends. All dependent variables are NOTE: Robust standard errors clustered at the county level are shown in parentheses. Each coefficient comes from a separate regression. All the dispensary.

Dependent variable:	Pre-term Birth	Low	Seizures	Low	Assisted	Admission
	(less man 3/ wks) (1)	birtnweignt (2)	(3)	APUAK Score (4)	venulation (5)	10 NICU (6)
Change after dispensary	-0.0081	0.0093	0.0001	0.0041	-0.0072	0.0127*
has opened	(0.0121)	(0.0057)	(0.0004)	(0.0051)	(0.0061)	(0.0071)
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
County FE	Yes	Yes	Yes	Yes	Yes	Yes
State-specific trends	Yes	Yes	Yes	Yes	Yes	Yes
Observations	39,383	39,482	39,304	35,151	39,304	28,888
R-squared	0.0104	0.0114	0.0039	0.0103	0.0311	0.0289
Mean of dep. variable	0.160	0.0672	0.0003	0.0205	0.0382	0.0917

Table 1.5: Event-study Estimates of Cannabis Dispensaries on Adverse Infant Health. Window time: +-90 days

The sample only includes counties with cannabis dispensaries and is restricted to births occurring between 90 days before or after the opening of the discrete. Each outcome is equal to one if the stated adverse condition occurred and zero otherwise (NICU means: Neonatal Intensive Care Unit). dispensary. regressio NOTE:]

Chapter 2

PHARMACEUTICAL PROMOTION AND THE OPIOID EPIDEMIC IN THE US

Joint with Dijana Zejcirovic (University of Vienna)

2.1 Introduction

Pharmaceutical companies invest as much in developing new drugs as in advertising their existing ones. In most cases, firms' promotional activities not only include ads directed to consumers but also frequent visits to physicians made by sales representatives who have strong financial incentives to prompt the prescription of their employer's drugs. This latter practice, known as *detailing*, is crucial for determining firms' revenues and profits. However, these interactions between sales representatives and physicians leave room for potential risks to public health because firms' optimal level of promotion may not coincide with patient safety. In this paper, we analyze the impacts of pharmaceutical promotion on health outcomes through its influence on physicians' prescription behavior. Using new data on links between pharmaceutical representatives and physicians in the United States, we provide evidence of the adverse health effects of promotion of opioid drugs which, by now, have caused unprecedented levels of severe addiction and fatal overdoses across the country.

Recent figures indicate that every ten minutes one 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). This increase is so dramatic 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 from previous decades (Case and Deaton, 2015). The public costs of this epidemic are not limited to higher mortality rates. The misuse of opioids contributed to the increase in hospitalization rates¹ In addition to all these adverse conditions among adults, 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).

Though the number of opioid pain relievers prescribed in the US skyrocketed over the same period, there was no simultaneous increase in the pain reported by patients (Chang et al. 2014). This means that the available evidence suggests that

¹According to the CDC, more than 1,000 Americans are admitted to the emergency room every day because of abuse of opioid drugs (Crane, 2013). Similarly, recent figures suggest that one out of four patients who receive prescription opioids are struggling with addiction (SAMHSA, 2014).

the increase in opioid prescription was not demand-driven. Therefore, we must ask why did health care professionals in the US increase their opioid prescription rates so extensively over the last two decades?

Before the 1990s, the use of opioid drugs was limited to treat acute pain among terminally ill patients, who were suffering from cancer. Given the proven efficacy of these drugs to reduce severe pain among this sub-population, many health professionals began to prescribe them to treat any type of pain such as post-surgery pain and low back pain. This broadening of indication of opioid drugs arose for a number of factors that took place during the 1990s. First, health experts became increasingly concerned with the optimal management of pain, because it became the fifth vital sign, next to body temperature, pulse rate, respiration rate and blood pressure (note that pain is the only vital sign with no objective measure). Second, state medical boards began to relax restrictions on prescribing opioid drugs for the treatment of non-malignant chronic pain. Third, at that time, medical research on the effectiveness and side effects of opioid analgesics in combating chronic non-cancer pain was scant. Only recently, medical studies have concluded that there is no rigorous evidence for the effectiveness of long-term opioid therapy for improving non-malignant chronic pain, while there are serious risks such as dependency, misuse and overdose (Chou et al. 2015). Fourth, pharmaceutical companies launched aggressive marketing campaigns targeted to physicians to promote opioid medication as an effective treatment option for non-terminally ill pain patients. Some of these manufacturers downplayed the risk of addiction and other adverse health outcomes, partly relying on limited or faulty empirical evidence (Van Zee, 2009), despite receiving warning letters from the Food and Drug Administration (FDA). This misleading information originated knowledge deficits and wrongly perceived safety of opioid drugs which, in turn, led to excessive prescription patterns (Manchikanti et al. 2012). Moreover, it is estimated that 60% of all overdose deaths occur among patients who are following their physician's prescription (CDC, 2012). The remaining part of deaths occur because patients who are prescribed opioids can also acquire opioid painkillers illicitly or switch to illegal opioid drugs, such as heroin. Indeed, 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).

Our study examines the impact of pharmaceutical promotion of opioid analgesics targeted to health care professionals on opioid-related adverse health outcomes in the US. 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) for the years 2014 and 2015. We first document that opioid promotion and overdose death rates are positively correlated using OLS regressions. Then, we adopt a differencein-differences estimation to show that this positive association is not driven by unobservable time-invariant county characteristics. The identification problem remains because even after controlling for county fixed-effects, the level of promotion is unlikely to be randomly distributed across counties with respect to opioid overdose death rates. For example, the promotion of opioid painkillers could be higher in places with low demand for opioid drugs if pharmaceutical companies are trying to open new markets. To overcome this challenge, we adopt an instrumental variables (IV) approach, in which opioid promotion is instrumented with the distance of the counties to the pharmaceutical companies' headquarters and the presence of state laws that restrict pharmaceutical promotion to physicians.²

We find that higher promotional activities for opioid analgesics were associated with higher mortality rates from opioid overdoses in 2014 and 2015. Our most conservative IV point estimate indicates that increasing the number of doctors reached by sales representatives by 1% increases overdose deaths by 0.16% (the 95% confidence interval ranges from 0.03% to 0.3%). This figure implies that completely removing promotion in the average county would decrease opioidrelated overdose death rates by 1.9 per 100,000 inhabitants (0.2 standard deviations).

To shed light on the mechanism of increased overdose death rates, we show that doctors receiving promotion for opioid drugs have higher opioid prescription rates. The IV results indicate that promotion has a positive and statistically significant effect on the number of opioid prescriptions with an elasticity of 0.1. These estimates lie within the range of elasticity coefficients found in other work

²Engelberg 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.
analyzing the impact of pharmaceutical promotion on prescription behavior.³

The estimated 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. Most of the remaining headquarters opened in the 19th century, rendering the concern of endogenous sorting less likely.

Second, there is no significant relationship between closeness to the headquarters and the number of opioid-related overdose death rates before 1995. The negative correlation between distance to the headquarters and death rates starts at the beginning of the 2000s, after the onset of large-scale promotional efforts for opioid painkillers.

Third, as one of our instruments, 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.⁴ It is very convenient for our identification that these states banned or limited pharmaceutical promotion 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 evidence suggests that the three states did not introduce the state bans as a response to increasing overdose death rates.

In addition, we analyze the impact of opioid promotional activities on neonatal health outcomes. 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). Although conclusive empirical evidence on the long-run consequences of suffering from neonatal abstinence syndrome is

³Kremer et al. (2008) conduct a meta-analysis on the impact of pharmaceutical promotion and find elasticity estimates ranging from 0.05 to 0.15.

⁴Minnesota introduced the law in 1997, and 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".

missing, a steep rise in health care expenditures due to increasing hospitalization rates and associated charges has been documented (Patrick et al. 2012). Furthermore, several 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 use the CDC 2014 Natality Detail Data Set and aggregate promotion in the nine months prior to the birth in the county of birth. Our IV estimates indicate that increasing the number of doctors receiving opioid promotion in a county in the nine months prior to birth increases the likelihood for a baby to be born with a low birth weight, to be born prematurely, and to need assisted ventilation. 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 pregnancy displays the highest correlation with negative health outcomes. This helps us to rule out the concern that counties with high opioid promotion rates are simply 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 higher levels of promotion.

To study the link between opioid-related overdose mortality and opioid promotion, we show that pharmaceutical promotion of opioid painkillers increases opioid prescription rates. More specifically, we measure physician prescription behavior using Medicare Part D prescription data for 2013 and 2014. Following 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. We find that physicians write more opioid prescriptions if they receive opioid promotion in the corresponding year. We also show that their opioid prescription behavior, however, is unrelated to pharmaceutical promotion of other drugs. Taken together, these results lend support to the interpretation that it is the promotion of opioid medications in particular, and not promotion *per se*, what is driving the increase in opioid prescriptions.

Since the data on promotional activities is only available from August 2013

onwards, we cannot attempt to explain the overall increase in drug poisoning mortality over time. Our approach, however, is useful to understand why some counties have much higher death rates of drug overdose than others. McDonald et al. (2012) document large geographic variations in opioid prescription rates in the US in 2008. For instance, the total amount of opioids dispensed in counties at the 75th percentile is four times larger than the amount dispensed in counties at the 25th percentile. They conclude that these large variations cannot be explained by differences in morbidity across the population. Our results contribute to this open question by showing that opioid promotion is related to opioid prescription patterns. The only evidence we have related to the increasing trend in overdose deaths comes from our reduced-form estimations (regressions of opioid-related death rates on our instruments over time) which reveal that for most years after 2000, an increasing number of people died in counties closer to opioid promoting headquarters.

More broadly, this paper adds to the growing literature on the opioid epidemic in the US. Previous studies 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). Reductions in overdose death rates have been found after the enactment of "Good Samaritan Laws" which provide immunity from prosecution for drug possession to anyone who is experiencing an opioid-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 (Buchmueller and Carey, 2018; Borgschulte et al. 2018; Kilby, 2015; Dave et al. 2017). Bachhuber et al. (2014) show that opioid-overdose related death rates decreased in states that legalized the use of medical marijuana (similarly, see Powell et al. 2018). 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 (Bradford et al. 2018), suggesting that these drugs are substitutes. Powell et al. (2015) show that an increase of opioid availability can have negative spill-over effects in the population. They find an increase of opioidrelated treatment admission and mortality rates among the Medicare-ineligible population following the introduction of the Medicare Prescription Drug Benefit Program (Part D), which increased the opioid utilization of the programs-eligible

population. Compared to this growing body of empirical work that comes from exploiting policy changes at the state-level, there is much less evidence on the relative importance of physicians' characteristics or firms' behavior.

As pointed out earlier, physician knowledge deficits appear to be one of the core causes of the opioid epidemic. To shed light on the factors that determine such deficit, Currie and Schnell (2018) analyze how opioid prescription rates depend on physician's medical school quality. They find that physicians who graduated from higher ranking medical schools prescribe significantly fewer opioids, suggesting that better medical training restrains opioid prescription. Our work complements these results by showing that opioid painkiller promotion to physicians plays a significant role in explaining the opioid epidemic through its effect on prescription behavior. By doing so, this is the first study to highlight the relevance of pharmaceutical promotion in the opioid epidemic, in addition to deficits in physician training or aggregate policy changes. Our findings corroborate previous work, unrelated to the opioid epidemic, that documents that pharmaceutical promotion to physicians influences their prescription behavior (Datta and Dave, 2017; Kremer et al. 2008). Moreover, 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.

The paper is structured as follows. Section 2.2 provides background information on the practice of pharmaceutical promotion to physicians in the US. Section 2.3 describes the data sources and provides basic descriptive statistics. Section 2.4 discusses the empirical strategy, followed by the estimation results (Section 2.5). Sections 2.5.1 to 2.5.2 report robustness checks. Section 2.5.3 explores the channel of increasing prescription rates and Section 2.6 concludes.

2.2 Background Information: Pharmaceutical Promotion to Physicians

Pharmaceutical promotion to physicians is a common practice in several 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. According to a nationally representative study, more than 80% of all physicians in the US received some form of gift by a pharmaceutical representative in 2004 (Campbell et al. 2007). One way that pharmaceutical companies promote directly to the physicians is through visits by sales representatives to the physician offices and hospitals. The sales representatives give details about the companies' drugs and in many instances leave a promotional gift, such as a lunch or pen or a drug sample.

In the economic literature, previous studies show that the 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, see Datta and Dave, 2017). Other work suggests that promotional activities reduce 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 distinguish 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, one cannot rule out the explanation of new information acquisition as sales representatives can, for example, emphasize that their drug have fewer side effects even when they are talking about pharmaceutical equivalents.

As the sales representatives are promoting directly to physicians, there is 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 stated 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 Purdue's promotional efforts and extended the marketing of their opioid pain relievers. The key message from these campaigns was that opioid drugs could 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 nor alternative therapies 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 (Medicare, Medicaid, and State Children's Health Insurance Program). These promotional payments are in the form of drug samples, meals, travel, research and consultancy fees, and related expenditures. These data on promotional activities are available from August 2013 until December 2015. In Figure 2.2 we split counties into high and low promotional activity 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

⁵The 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.

⁶For 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 2.2 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 B1a). For the years from 1999 on we have mortality data for all counties. In Figure B1b, we can see that mortality rates are statistically significantly higher in counties that receive high levels of promotion from 2005 on.

2.3 Data and Descriptive Statistics

We combine multiple sources of data to conduct our empirical analysis. An overview of all data sets used and the corresponding time periods can be found in Table B1.

After the enactment 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 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 are available from August 2013 to December 2016. Our main outcome of interest, opioidrelated overdose death rates, are only available for the years prior to 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.

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 data set can represent the amount directly paid to the physician for speaking fees or the dollar value of the lunch or other gifts provided, or the sum of both.

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 as an opioid analgesic following the Anatomical Therapeutic Chemical (ATC) Classification System of the WHO (ATC code N02A). We exclude opioids 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.

⁷See Table B2 in the Appendix for a list of keywords used.

Table 2.1 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 indicate that physicians and teaching hospitals receive on average visits by one opioid manufacturer a year. This suggests that the different 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 data set 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 abstain from making welfare statements about terminally-ill patients who instead of dying from their fatal disease, die from an overdose of opioid medication. We subtract the count of deaths by neoplasms only from the count of the fatalities caused not only by overdose but also by neoplasms (ICD-10 Code: C00-D48), to obtain the count of fatalities due to opioid overdose only. Table 2.1 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 B3 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 1997⁹, while Vermont¹⁰ and Massachusetts¹¹ 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.

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

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

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

¹¹Commonwealth 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).

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 B5. 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 2500 grams).

Another data source used is the Medicare Provider Utilization Data 2013 and 2014 collected by the CMS. These files contain information on Medicare Physicians, 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 adopt a difference in difference estimation because of lack of data on payments made before August 2013.

Table 2.2 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 data set 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 gather more information on the characteristics of physicians, 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 2.2. 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 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 use multiple data sources to collect socio-economic county characteristics that could correlate with opioid overdose mortality rates. 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 B4 summarizes county characteristics for 2014 and 2015.

 $^{^{12}}$ 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 the school has a formal curriculum on conflict of interests iv) how well the policies are enforced and sanctioned and v) other domains.

2.4 Empirical Analysis

2.4.1 Pharmaceutical Promotion and Opioid Overdose Deaths

The purpose 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 opioid drugs. Physicians decide whether to prescribe these 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:

$$OD_c = \alpha_s + \beta^{OLS} Prom_c + X'_c \Gamma + \varepsilon_c$$
(2.1)

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 and 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:

$$OD_{c,t} = \theta_1 CountyFE_c + \theta_2 TimeFE_t + \beta^{FE} Prom_{c,t} + X'_{c,t}\Gamma + \varepsilon_{c,t}$$
(2.2)

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 analgesics over alternative treatment options. Besides the omitted variable bias, OLS regression results may suffer from measurement error. Pharmaceutical companies have, as argued earlier, an incentive to underreport 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 2.3 three states (Minnesota, Vermont, Massachusetts) have introduced some form of state bans on pharmaceutical promotion. These three states have introduced state bans for all kinds of pharmaceutical promotion, not opioid medication in particular. We show in Section 2.5.1) that the introduction of these bans 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:

$$Prom_{c} = \phi + \rho_{1}Dist_{c} + \rho_{2}Ban_{c} + X_{c}^{'}\Psi + \mu_{c}$$

$$(2.3)$$

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 socioeconomic characteristics, which also determine opioid overdose rates.

The second-stage equation is:

$$OD_c = \alpha + \beta^{IV} \widehat{Prom}_c + X'_c \Gamma + \varepsilon_c$$
(2.4)

where \widehat{Prom}_c is the prediction from the first-stage (Equation 2.3). 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 Sections 2.5.1 to 2.5.2.

2.4.2 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 2.4.1 using physician-level information. We estimate the following first and second stage equations:

$$Prom_{i,t} = \pi + \gamma_1 Dist_i + \gamma_2 Ban_{is} + \theta Spec_i + \zeta Pres_{i,t-1} + \nu_{iz}$$
(2.5)

$$Pres_{i,t} = \lambda + \delta^{IV} \widehat{Prom}_{i,t} + \kappa Spec_i + \eta Pres_{i,t-1} + \epsilon_{iz}$$
(2.6)

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.

2.5 Results

2.5.1 **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 2.3, we report the estimated coefficients of Equation 2.1. 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 2.3 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 2.4 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 2.5. 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 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. Excluding these 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 2.5. 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

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

¹⁴Table B3 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.

to the OLS estimates, these coefficients are larger, suggesting that the latter were potentially downward biased. One reason why the OLS results may be downward biased is that pharmaceutical companies target counties with low initial demand for opioids to open new markets. 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 further control for county characteristics. These characteristics 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 control for these variables. The robustness to the inclusion of county characteristics limits the concern that we are simply 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 as the number of doctors receiving promotion because, as in previous studies, we do not distinguish between the informative or persuasive nature of promotion. In Section 2.5.2, 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 larger elasticities than spending more money on the same physicians.

Exogeneity Assumption and Over-identification Test

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 2.3a 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 2.3b shows the differences in opioid overdose rates in 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.¹⁵ It is important to note that the ban holds for all types of drugs, not only

¹⁵In our empirical analysis, we include a dummy for states that have any form 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

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 teaching hospitals is uncorrelated with the error term in the equation of interest. We, therefore, restrict our set of pharmaceutical companies to the ones whose headquarters location in 2014/2015 was already determined before 1995.¹⁶ All companies that started operations after 1995 or moved their headquarters after 1995 are dropped from our sample in the main analysis.¹⁷ We also show that opioid overdose rates before 1996 are independent from the distance to headquarters in 2015 in Figure 2.4. 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 between promotion and death rates for the West Coast, as distance to headquarters in kilometers is not relevant for these counties.¹⁸

Our instrumental variables model depicted in Equations (2.3) and (2.4) is overidentified. This allows us to look at the regression results using the instruments

only Minnesota and Vermont are coded as states with bans. In this specification, 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.

¹⁶Before 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.

¹⁷The different results including and excluding all companies are depicted in Table 2.5.

¹⁸Results are available upon request.

separately and to test whether the instrument exogeneity condition is valid for one of the two instruments. Table A1 shows the estimation results of Equations (2.3) and (2.4) 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 and physicians within a reasonable distance from 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.¹⁹ 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 over-identification 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 over-identified 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 enhances the credibility that our instruments are valid.

¹⁹Excluding the possibility of air travel, for which physical distances are less relevant.

Heterogeneity Analyses

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 sub-samples of counties with more and less than 100,000 inhabitants. Table A2 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.

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.²⁰ Table A3 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 overdoses. It is claimed that many patients who were prescribed opioid medications and became addicted, substituted with 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).

2.5.2 Alternative Outcome Variable: 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 of detrimental health outcomes for unborn babies. For this reason, we investigate whether the negative health impact we document 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

 $^{^{20}\}mbox{Overdose}$ death due to heroin intake is classified as T40.1 in the CDC multiple cause of death mortality database.

delivery is negatively related to health outcome measures. More specifically, we regress the number of doctors that received opioid promotion on neonatal health outcomes following the same empirical approach as depicted in Section 2.4.1. 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. In all regressions, we include mother characteristics at birth, such as demographics and health measures, delivery information (prenatal care, the method 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. The positive relationship we have documented between opioid promotion and death rates can also be found in terms of negative neonatal health outcomes. Table A4 displays the OLS regression results described in 2.5.2. 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 a low birth weight if its weight is below 2500 grams. Prematurity is defined by neonates born at less than 37 weeks' gestation. Panel A of Table A4 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 A4 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 A5 depicts the results of the first and second stage regressions of the 2SLS equation described in Section 2.5.2 and 2.4.1. 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 co-

efficient estimates and the partial F-Values of the first stage are displayed in Panel B of Table A5. 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 40.3.²¹

Robustness Checks

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 drugs and that the location of the headquarters is related to previous levels of overdose rates. We, therefore, repeat our analysis of Equation (2.3) and (2.4) but additionally control for overdose death rates in the previous years. As seen in Table A6 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 A6 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 A6, 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

²¹When 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.

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.

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 A7). As in the OLS regressions, our estimates are half the size compared to the regressions in which promotion is measured by the number of doctors receiving any value of promotion.

As we are normalizing the number of doctors receiving promotion for opioid painkillers by the population, readers may be worried that promotion for opioid drugs may mechanically be high in counties with a large number of doctors. We repeat our analysis adjusting the number of doctors receiving promotion by the total number of doctors in the corresponding county and find qualitatively similar results with larger elasticities (results are available upon request). The magnitudes of our elasticities from the main specification do not change if instead we additionally control for how many doctors were active in the corresponding county.

2.5.3 **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 2.6 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.²² 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 2.6 also displays the regression results of the first and second stage of Equations (2.5) and (2.6). 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 2.6, 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 2.3. 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 A9). These estimates are in line with elasticity coefficients found in other work. Kremer et al. (2008) conduct a metaanalysis on the impact of pharmaceutical promotion and find elasticity estimates between 0.05 and 0.15.

In Table 2.7, 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 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 2.6, where we do not control for promotion of 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 A10 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

²²Table A9 in the Appendix displays the results for alternative empirical specifications.

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 first to be less likely to receive opioid-related promotion and second to adjust their opioid prescription behavior less after engaging with sales representatives. Unfortunately, these data are 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 A8 displays the heterogeneous effects of receiving opioid promotion on opioid prescription rates. Column (1) shows that physicians affiliated to a hospital with conflict of interest policies 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 A8). We do not see 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 with 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.

2.6 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).

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 receive 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 prescription behavior of opioid drugs varies substantially, especially among general practitioners. The more opioid drugs are prescribed, the more people die of opioid-related overdoses (Currie and Schnell, 2017). Currie and Schnell (2017) find that a fraction of this variation 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 on average. 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.²³ In overseeing the 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 on 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.²⁴

²³According to the Code of Federal Regulations Title 21 (Food and Drug 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).

²⁴A 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." (National Academies of Sciences , 2017).

Tables and Figures

Figure 2.1: 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 2.2: 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



Figure 2.3: 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 2.4: Reduced form estimates: distance to headquarters and overdose death rates over time.



Coefficent 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 2.1: Summary statistics US counties pharmaceutical promotion & opioid-related death rates 2014-2015

	Observations	Mean	Median	Std. Dev	Min	Max
• •••						
2014						
County Aggregates	2142	11.25	1.00	22.06	0	620
Doctor receiving Optoid Promotion	2142	10.04	1.00	55.90	0	1520
Shere of Payments with no Drug ID	2058	10.04	1.00	01.99	0 00	1339
Total Payments for Opioids in \$	2930	1161	0.20	10673	0.00	1067246
Total Payments for Opioids in $\$$ (> 0)	1708	2127	7.15	26648	1 50	1067246
Total Payments for Dainkillers in (> 0)	3142	2137	16.78	20048	0	1523830
Total Payments for Dainkillers in $\$$ (> 0)	1815	2390 4137	10.76	29990	1 1 8	1523839
Total Fayments for Fankmers III \mathfrak{s} (> 0)	1815	4137	130.11	39360	1.10	1525659
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	Õ	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)	2015	11.12	2.00	21.07	0	517
Iotal Deaths	2915	11.13	2.00	31.96	0	517
Adjusted by Population (by 100,000)	2915	9.00	0.39	10.4	0	131

Source: CMS Open Payment Data 2014 and 2015, CDC Wonder Multiple Cause of Death Data.
	Ν	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
Closest HO Distance & State Ban					
Min. Distance HO 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

Table 2.2: Summary statistics Medicare prescribers 2014

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.

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				0 055 4***		0.00002
log USD 2014				0.0334		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

Table 2.3: OLS: opioid overdose deaths and opioid promotion

Estimation result of Equation (2.1). 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.

Dependent Variable:	(1)	(2)
log Opioid Overdose Deaths		
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

Table 2.4: Fixed effect regression: opioid overdose deaths and opioid promotion

Estimation result of Equation (2.2). 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 2.3. 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.

	Panel A: First	t Stage	
Dep. Var.:	(1)	(2)	(3)
log Receiving Doctors			
Dist. calculated to:	All Headquarters	Opened be	efore 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	Ν	Ν	Y
Year F. E.	Y	Y	Y
	Panel B: Secon	d Stage	
Dep. Var.:	(1)	(2)	(3)
log Opioid Overdose Deaths	~ /		
Instruments: State ban			
and Distance to	All Headquarters	Opened be	efore 1995
		1	
log Receiving Doctors	0.687***	0.337***	0.317***
0	(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	Ν	Ν	Y
Year F E	Y	Y	Y

Table 2.5: 2SLS: opioid overdoses and opioid promotion

Estimation results of Equations (2.3) and (2.4). Partial F-value of first stage Equation (2.3) 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).

Method:	O	LS	28	LS
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***
I	(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	Ν	Ν
Physician Specialty	Y	Y	Y	Y
First Stage Results				
β Dist. HO 2014			-0.00318***	-0.00970***
p 2134 11 2 2 011			(0.000288)	(0.000981)
β State Ban			-0.0155***	-0.0477***
			(0.000878)	(0.00310)
Partial F-Value			187.6	145.6

Table 2.6: Opioid prescriptions and opioid promotion: OLS & 2SLS

Number of opioid claims of Medicare Physicians and opioid-related promotion OLS and 2SLS estimates. 2SLS estimation results of Equations (2.5) and (2.6). 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.

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***
opioia i tomotion	(0.824)
	(0.024)
# Opioid Pres. 2013	0.972***
	(0.00722)
County FF	V
Physician Specialty	I V
Maan Dan, Var	114.0
SD Dara Var	114.9
SD Dep. var.	322.3
Observations	633306
<u>R2</u>	0.889

Table 2.7: Placebo: opioid prescriptions and non-opioid promotion

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 (2.5). 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.

Appendix A

		All counties			<500km to HQs			
Dep. Var.:	(1)	(2)	(3)	(4)	(5)	(6)		
log Opioid Overdose Deaths								
log opioid overdose Deaths								
Instruments used	Both	Distance only	Ban only	Both	Distance only	Ban only		
instruments used.	Dom	Distance only	Dunionity	Dom	Distance only	Duil Only		
	0.007***	0.7/5***	0.150	0.007***	0.070*	0.250***		
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				

Table A	.1:	2SL	<i>S</i> :	sing	le	instruments	and	Sargan ³	's	over-id	entif	ficatior	i test	i
				~~~~~					_					•

2SLS regression results (see Eq. (2.3) and (2.4)) using i) both instruments, ii) the instruments separately for a) all counties and for b) all counties within 500km distance to the closest head-quarters. Partial F-value of first stage Equation (2.3) and P-Value of Sargan over-identification test displayed in last two rows. Standard errors in parentheses adjusted for heteroscedasticity, * (p<0.10), ** (p<0.05), *** (p<0.01).

Dependent Variable:	(1)	(2)
log Overdose Death in Counties with:	< 100,000 inh.	$\geq$ 100,000 in h.
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

Table A2: 2SLS overdoses and promotion: small vs. large counties

2SLS regression results (see Eq. (2.3) and (2.4)), 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).

	(1)	(2)	(3)	(4)	
Method	0	LS	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	

Table A3: Illicit vs. all opioid overdose deaths

OLS and IV estimates for overdoses only including Heroin (T40.1) compared to all opioid overdoses. OLS estimate from Equation (2.1) and IV following Equation (2.3) and (2.4). 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 2.5). Standard errors in parentheses adjusted for heteroscedasticity, * (p<0.10), ** (p<0.05), *** (p<0.01).

	(1) Admission NICU	(2) Ventilation Immediately	(3) Ventilation > 6hr	(4) APGAR 5	(5) Birth Weight	(6) Low BW < 2500g	(7) Premature Born
		<u>y</u>					
		Par	nel A: Promotion	During Pregna	ncy		
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
	0.00.100**	0.00001.50	Panel B: Promoti	on By Trimeste	r 5.010444	0.0000 (****	0.001.12
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***
Sid fillioster	(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

### Table A4: OLS: neonatal health and opioid promotion

Estimation result of Equation (2.1). 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.

			Panel A: Secon	d Stage Result	ts		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Admission	Ventilation	Ventilation	APGAR 5	Birth Weight	Low BW	Premature
	NICU	Immediately	> 6hr			< 2500g	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
			Panal R. First	t Stage Results			
B Dist HO 2014	-0 385***	-0 385***	-0 385***	-0 385***	-0 385***	-0 385***	-0 385***
p Dist. 11Q 2014	(0.0560)	(0.0560)	(0.0560)	(0.0560)	(0.0560)	(0.0560)	(0.0560)
B State Ban	1 360***	1 360***	1 360***	1 360***	1 360***	1 360***	1 360***
	-1.500	-1.500	-1.500	-1.500	-1.500	-1.500	-1.500
Dortial E Value	(0.132)	(0.132)	(0.132)	(0.132)	(0.132)	(0.132)	(0.132)
Paruai F-value	40.64	40.04	40.64	40.67	40.69	40.69	40.68

#### Table A5: 2SLS: neonatal health and opioid promotion

Estimation result of Equations (2.3) and (2.4). 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 (2.3) 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 2.1. 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.

Dep. Var.:	(1)	(2)	(3)
log Opioid Overdose Deaths			
log Opioid Promotion Receiving Doctors	0.317*** (0.0782)	0.166** (0.0718)	0.359* (0.198)
log Opioid Overdose Deaths in t-1		0.418***	
		(0.0180)	
log Non-Opioid Promotion Receiving Doctors			-0.0388
			(0.0829)
Mean Den Var	1 664	1 682	1 664
SD Den Var	1 190	1.002	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

Table A6: IV 2SLS overdoses and promotion: pre-year level of overdose deaths and non-opioid promotion

2SLS regression results (see Eq. (2.3) and (2.4)). 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).

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

#### Table A7: Overdose & promotion: 2SLS and OLS promotion in USD

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 (2.3) and (2.4). 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 2.5 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.

	(1)	(2)	(3)
	# Opioid	# Opioid	<b>Received</b> Opioid
	Prescriptions	Prescriptions	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 Ren Ban * D	-127 8*	-144 5**	
Sales Rep. Dali D	(72.52)	(72.60)	
	(12.32)	(72.09)	
Male		24.67***	0.0154***
		(2.245)	(0.00141)
Male * D		178.9***	
		(32.92)	
Graduated before 1995		51 08***	0 0203***
Graduated before 1775		(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

Table A8: Opioid prescriptions and opioid promotion: heterogeneity by physician characteristics

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.

Functional Form	Linear	Log	Elasticity	Deciles
Dep. Var.: Opioid Proscriptions	(1) # Proc. 2014	(2) # <b>P</b> roc. 2014	(3) # Pros. 2014 (log)	(4) # <b>P</b> ros 2014
Oploid Frescriptions	# 1168. 2014	# 1108. 2014	# Fles. 2014 (log)	# FICS. 2014
Opioid Promotion (USD)	0.00526** (0.00238)			
Opioid Promotion (log USD)		15.55*** (0.895)	0.114*** (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

Table A9: OLS estimates promotion & prescriptions: different functional specifications

Number of opioid claims of Medicare Physicians and opioid-related promotion. Estimation result of Equation (2.5). 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.

	(1)	(2)
	% Opioid Claims	% Opioid Claims
Opioid Promotion	0.00239***	0.00400***
	(0.000200)	(0.000215)
Non-Onioid Non-Painkiller Promotion		-0.00156***
Non Opiola Non Famkiner Fromotion		(0.00100)
		(0.0000092)
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
<u>R2</u>	0.688	0.689

Table A10: Promotion and share of opioid claims over all claims

Outcome variable: share of opioid claims over all claims by Medicare Physicians and pharmaceutical promotion. Estimation result of Equation (2.5), 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.

Appendix B

#### Figure B1: 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.

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Data			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

Table B1: Data availability

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Table R7	Nubetance	namee	ncedta	o identity	INT	101d	anal	GAC1C	1n 1	navn	1ent	data.
$1000 D_{2}$ .	Substance	names	uscu u	0 Iuciui	νυι	JIUIU	anai		111	υανπ	IUIII	uata
								<b>O</b>		· · · . /		

Opium	Hydromorphone
Oxycodone	Papaveretum
Pethidine	Fentanyl
Piritramide	Dextropropoxyphene
Methadone	Pentazocine
Butorphanol	Nalbuphine
Tramadol	Dezocine
Tapentadol	
	Opium Oxycodone Pethidine Piritramide Methadone Butorphanol Tramadol Tapentadol

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

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

Table B3: List of opioid promoting manufacturers

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 B4: 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				
2015				
Promotion (adjusted by population)	5 5 5	0.25	0.00	125 /1
Doctors receiving other Painkiller Promotion	13.66	9.25	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
Unel services	0.03	0.02	0.00	0.28
Unclassificu	0.00	0.00	0.00	0.08

	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 Ph	ysicians					
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

### Table B5: Summary statistics neonatal health

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

## **Chapter 3**

# IMPROVED ACCESS TO ELECTRICITY AND STUDENT LEARNING IN PERU

Joint with Hugo Ñopo (ILO) and Rosamaría Dasso (IFAD)

### **3.1 Introduction**

Recent figures indicate that around 1.3 billion people lack access to electricity in developing countries (International Energy Access, 2016). International agencies and national governments are allocating increasing amounts of resources to close the energy gap between urban and rural areas, where electricity coverage is significantly lower. This global policy issue has also caught the attention of a growing number of empirical studies which aim to estimate the welfare impacts of providing access to electric energy (Barron and Torero 2017; Dasso and Fernandez 2015; Khandker et al. 2013; Lee et al. 2016; Lenz et al. 2017; Van de Walle et al. 2017).

In this paper, we ask whether improved access to electricity in rural areas can boost student learning using data from Peru. The Peruvian case is relevant for two reasons. On the one hand, student learning remains at dismal levels despite fast macroeconomic growth and improvements in social indicators (e.g. poverty reduction). Low learning levels are reflected on international assessments (e.g. PISA 2009, 2012) in which Peru ranks among the worst performing countries in numeracy and literacy. On the other hand, electricity access in rural areas nearly doubled over the past decade, increasing from 40% of households with electricity in 2006 to almost 80% by the end of 2012. This unusual improvement in access to electricity occurred due to the rapid expansion of the rural electrification program known as PER - *Programa de Electrificación Rural*. Since other developing countries face similar challenges in improving both education and electricity access, our study can shed light on how these two variables are related to each other.

Better access to electricity can affect educational outcomes through different mechanisms. First, electricity could change patterns of time use among children towards longer study hours (Barron and Torero 2014) and improve learning. Second, as shown by Barron and Torero (2017), household electrification reduces indoor pollution which improves child health, and could indirectly translate into learning gains. Third, access to electricity at school can impact learning through better school infrastructure (e.g. amenities, information and learning technologies) which may be useful for both students and teachers. More indirectly, electrification could influence educational outcomes through changes in parental re-

sources. Dasso and Fernandez (2015) show that better access to electricity in rural Peru, increases hours of work among men, and labor earnings among women. We hypothesize that these changes in paternal time and maternal income can also impact student learning.

We build on this body of work to provide novel evidence on the impacts on learning of a large-scale intervention that improved energy access in rural areas. Our main analysis relies on administrative data on electrification projects, national standardized tests, and school infrastructure. The identification strategy exploits spatial and temporal variation in access to electricity to estimate the impact of the program on student learning. First, we document that the intervention increases access to electricity by 8 percentage points. Second, we find that the estimated average impacts on student learning are not statistically different from zero. Third, we present evidence indicating that among treated schools, longer treatment exposure slowly improves both Reading and Math skills for boys and girls. If these heterogeneous impacts are persistent, the effects of PER on learning could be positive in the long-run.

To complement the analysis on student learning, we use household-level panel data to explore whether electrification changes school enrollment and expenditures on education. In most cases, we cannot reject the null hypothesis of no impact on these two educational outcomes. The null impacts on these indicators could suggest that improvements in learning are being driven by longer study hours, instead of changes in enrollment or higher household expenditures on children. Unfortunately, we cannot directly test this mechanism due to lack of data.

Using supplementary data sources, we provide additional evidence to support the validity of our results. First, we show that, before the expansion of the program in 2007, there were no differential trends in educational outcomes between treatment and control districts. Second, we consider two other programs (Conditional Cash Transfers and "One Laptop Per Child") that were taking place in rural districts during the study period. These confounding interventions may introduce biases to our estimates if they were correlated with the implementation of PER. Our coefficients remain unchanged after controlling for these interventions.

The rest of the paper proceeds as follows. Section 2 reviews the related literature. Section 3 describes the rural electrification program. Section 4 details our data sets. Section 5 outlines the empirical strategy. Section 6 presents results and robustness checks. Section 7 offers concluding remarks.

## 3.2 Related Literature

The literature on the impacts of school interventions on educational outcomes has been reviewed by Kremer and Holla (2009), and Glewwe et al. (2011). The former focuses on randomized evaluations in developing countries, and the the latter reviews studies with both experimental and non-experimental designs. We begin this section by discussing the latter review and then briefly summarize other studies related to our paper, but *not* covered in such reviews.

From 1990 to 2010, 6 out of 79 reviewed studies in Glewwe et al. (2011) provide estimates for the effects of electrification on test scores. Based on the empirical methods of each study, the authors divide their 79 papers into: i) 36 regular studies (3 on electrification), and ii) 43 high-quality studies (3 on electrification). Among the 3 regular studies, the estimated coefficients on electricity access are mostly positive and significant (3 coefficients are positive but insignificant, and 6 are positive and significant). Among the 3 high-quality studies, only insignificant (3 negative and 3 positive) effects are found¹. These reviewed studies, however, are not especially interested in estimating the educational impacts of access to electricity but rather include this variable on their regressions along with other control variables.

Aside from these studies on school interventions, there are four empirical papers not included in the previous reviews, closely related to ours. Dinkelman (2011) is the first paper using a clean identification strategy to quantify the consequences of electrification on labor outcomes in rural South Africa. Using land gradient as an instrument for electrification, and complementing her IV estimates with fixed-effect models, her results indicate that female employment increases in treated areas. In a similar spirit, Rud (2012) studies the effects of electrification on industrial production in India, using river water flow as an instrument for access to electricity. He documents positive impacts of electrification on manufacturing

¹There are more estimates than studies because some papers provide more than one estimated effect (e.g. rural versus urban).

output.

Khandker et al. (2013) use household panel data from rural Vietnam and compare households with and without electricity connection to show that electrification has positive and significant effects on household income and expenditures, and school enrollment. They also find that these benefits level off after 9 years of electricity use.

Libscomb et al. (2013) use county-level data from urban and rural Brazil to examine the impacts of electrification on the Human Development Index (HDI). Their strategy consists of simulating electricity grid expansion taking only into account topographic considerations (water flow and river gradient). Then, the authors use these predictions as instrumental variables for actual program placement. They document large positive effects on the income and education (literacy and enrollment rates) components of the index but not on health (life expectancy). These large estimated effects, however, may be driven by the fact that the IV approach uses variation on compliers which, by construction, are counties with the most cost-effective electrification projects.

More recently, Barron and Torero (2014, 2017) randomly assigned incentives to households to connect to electricity in El Salvador. In their studies, they provide clean evidence of positive impacts of electrification on hours of study and child health. Access to electricity allows children to allocate more time to schoolrelated activities and also reduces indoor pollution, improving health among children. Both channels - longer study hours and better health - could lead to improvements in student learning.

Our work adds to this recent and growing literature in three ways. First, we provide direct evidence on the effects of electrification on learning, which are a better proxy for human capital given the low levels of school quality in developing countries (Hanushek and Woessmann 2008). Second, we evaluate a large-scale governmental intervention rather than a pilot program in a small number of places. Third, we pay attention to household responses (expenditures on child's education) which are crucial to correctly interpret the estimates of electrification on educational outcomes.

## 3.3 The Intervention: Programa de Electrificación Rural (PER)

In 1992, the Peruvian electricity sector (generation and distribution) was privatized and the government created OSINERGMIN, the energy regulator. At that time, 60 percent of rural households in Peru were below the poverty line and only 7 percent had access to electricity. To improve this situation, the Ministry of Energy and Mining launched an electrification program named PER - *Programa de Electrificación Rural*- to foster social and economic development in rural areas.

The Ministry worked jointly with electricity distribution companies (EDC) to expand energy access in rural areas. Electrification projects were implemented following these criteria:

- Projects located in districts with lower electricity coverage (percentage of households with electricity)
- Projects located in districts with higher poverty rates (percentage of households whose consumption is below the national poverty line)
- Projects with lower proportion of the estimated subsidy per connection
- Projects with lower cost per (new) connection
- Projects with higher use of renewable energy

The Ministry partially funded electrification projects using these variables and distribution firms covered the rest of the investments. Most of these projects consisted of transmission lines (grid expansion). In areas where grid expansion was not feasible, hydro-power plants and photovoltaic solar systems were used. Once projects were concluded, no incentives (subsidies) were provided to schools nor households to get electricity connection.

Throughout the paper, urban districts are excluded from the analysis. From the universe of rural districts in Peru, we define two groups. The treatment group (PER districts) includes all rural districts that had an electrification project funded by the program and concluded between 2007 and 2010. The comparison group (non-PER districts) consists of the remaining rural districts. Therefore, districts in the comparison group do not necessarily lack access to electricity. This means that our treatment is best thought as an increase in access to electricity. We should not interpret this intervention as going from a state of no electricity to universal access to it.

Figure 3.1 presents graphical evidence of the rapid increase in electricity coverage over the study period. In particular, we compare access to electricity at home across treated and non-treated districts. As expected by the priority criteria, PER districts (places where electrification projects funded by the Ministry took place) had an electricity coverage 10 percentage points lower than non-PER districts (rural districts not targeted by PER) at the beginning of the study period. Four years later, this gap in access to electricity had been reversed and PER districts had higher coverage than non-PER districts (comparison group). This figure shows that access to electricity in PER districts increased by 20 percentage points in only four years. Similarly, in Figure 3.2, we present the rapid increase in access to electricity among schools in PER districts. The program's current goal is to increase electricity coverage up to 95 percent in all rural areas before 2023.

We focus on projects that were concluded in the period 2007-2010 for three reasons. First, 90 percent of projects concluded before 2013 were finished between 2007-2010. This increase was related to an executive order signed by the government in May 2007 which provided greater fiscal autonomy to the program to fund electrification projects. In Table 3.1, we see that previous to 2007, annual investment in electrification projects was around US\$ 40 million. This amount was doubled in 2007, and continued to increase in the next few years, reaching US\$190 in 2010. Second, the National Office of Statistics (INEI for its name in Spanish) collected a unique household panel data set from 2007 to 2010. Third, the Ministry of Education administered national standardized tests to all 2nd-graders in the country during 2007-2015.

From 2007 to 2010, 554 electrification projects were concluded throughout rural Peru (628 were finished between 1994-2012) with a total investment of US\$ 517 million. Figure 3.3 depicts the roll-out of the program during the study period. First, we notice the large-scale of PER. Second, we see that the implementation of the program was evenly distributed across space, rather than being concentrated in any particular geographical area.

### 3.4 Data

We use three data sources in the empirical analysis. First, we use administrative records of the Ministry of Energy and Mining, containing the list of electrification projects that were concluded between 2007 and 2010. In our study period, 554 projects - out of 628 since 1994- were concluded in 412 rural districts. For each project, we observe the year of conclusion and the treated districts (101 projects include more than one district). This information is used to define treatment and comparison groups.

Second, we use nine rounds of the national standardized test, named ECE -*Evaluación Censal de Estudiantes*-, administered by the Ministry of Education, and taken by all 2nd-graders (who are, on average, 7 years old) in Peru between  $2007-2015^2$ . At the end of each school year (around early December), students are evaluated in Math and Reading skills, using a three-level scale grading system (from highest to lowest): proficient, partially proficient, and not proficient. For each school, year and subject, we define three outcome variables based on this grading system: the percentage of students who reached the highest proficiency level of the exam, the percentage of students in the intermediate level, and the percentage of students in the lowest level. To ensure that these school-level outcomes are meaningful, we restrict our sample to rural schools with at least 5 students in both 2007 and 2008, and that took the test at least 7 times (out of 9). We complement these data with information on school infrastructure and teaching staff from the school census, which is conducted on a yearly basis by the Ministry of Education.

Third, we use a household panel data set known as ENAHO - *Encuesta Nacional de Hogares*- conducted by INEI between 2007-2010. This survey includes comprehensive information (education, health, employment, dwelling characteristics) at both the household and individual level³. For each individual between

²The Ministry only has budget to perform the national test in 2nd grade. For this reason, we only look at the impact of electrification on learning in this grade. Though it is possible that the impact of electrification is not constant across different grades, this analysis is beyond the scope of our study.

³Originally, ENAHO is a cross-sectional sample, representative at the national level, that is drawn every year since 2004. The panel sample used in this analysis is, indeed, a random sub-sample of the original (larger) sample taken in 2007

ages 3 and 18 in 2007, we construct two outcomes using ENAHO data: enrollment and the log of household education-related expenditures per child⁴. Enrollment is a discrete variable, which is equal to one if the child is enrolled in school and zero otherwise. Educational expenditures per child include: uniform and shoes, textbooks, materials, fees, and parent's associations. Although primary and secondary education are compulsory in Peru, enrollment rates are nearly universal (i.e. close to 100 percent) only in primary schools. Pre-primary schools and secondary schools are far from these levels, and therefore, it is reasonable to expect some impacts on these educational levels.

For the school-level analysis, we match ECE and PER data sets using district identifiers, exam year, and project's year of conclusion. For the household-level analysis, we match ENAHO and PER data sets using district identifiers, ENAHO survey year, and year of conclusion of each project. After doing so, the ECE-PER and ENAHO-PER samples include 4,246 schools and 1,602 individuals, respectively.

Table 3.2 presents descriptive statistics of control and treatment groups in the year 2007 for both panel data sets. In the ECE data set, we see that schools in control districts have more 2nd grade students than schools in treated areas, though this difference is very small in magnitude (less than one student). The proportion of male students is 51 percent in both groups. Then, we see that 19 percent of schools in control districts have complete staff teaching⁵ and this figure is 16 percent in treated districts. Relative to treated schools, the average share of teaching staff with tenure (permanent contracts) is slightly higher in control schools. Although some of these differences are statistically significant at the 5 percent level, they are quite small in magnitudes and, if anything, they show that treatment schools were worse off than control schools at baseline, which makes it harder for us to detect positive impacts on student learning.

At the bottom of the table, we report summary statistics using the ENAHO

⁴In practice, we add 1 to each amount of expenditures (expressed in *Nuevos Soles*) because 20% of the observations have zero expenditures. Therefore, our dependent variable is log (expenditures+1). The average expenditure before and after such modification is 101 and 102 *Nuevos Soles*, respectively.

⁵Complete staff teaching is equal to one if there is one teacher per grade, and zero otherwise (the same teacher is responsible for several grades.

household data. In control districts, around 80 percent of households have a male head and 85 percent in treated districts but this difference is not statistically significant. Household heads are between 48 and 50 years old in the sample, and this small difference between groups is only significant at the 10 percent level. In control areas, 11 percent of household heads do not have formal education while in treated areas this figure goes up 14 percent, however, this difference is not statistically significant. In both groups, around half of the household heads in both groups have a monthly income between 240 and 260 *Nuevos Soles*, which is equivalent to 72 and 79 US current dollars. In Section 6.3 we present evidence suggesting that there were not differential trends in educational outcomes before 2007.

Before discussing our empirical methodology, we present evidence on the impacts of the intervention on *actual* access to electricity at school and at home. To do so, we use the ECE (school-level) panel data and the ENAHO household panel sample to separately regress an indicator variable for access to electricity on the treatment indicator and a measure of exposure to the program. This measure is the number of months between the conclusion of the first electrification project in the district and the time of the exam (in the ECE data) or the time of the interview (in the ENAHO data). We include school fixed-effects with the ECE data and household fixed-effects with the ENAHO data⁶, along with year fixed effects in each specification.

Table C1 in the Appendix presents the point estimates from each regression. As we can see, our independent variables of interest are strongly and positively correlated with access to electricity. In column 1, we show that the program is associated with a 7 percentage point increase in electricity coverage in schools. Moreover, in column 2, we see that longer program exposure increases access to electricity even further. The estimated coefficients in columns 3 and 4 tell a similar story but for access to electricity at home. In short, these results show that the program actually leads to higher electricity coverage among treated schools/households.

⁶The ENAHO household panel sample is simply the individual panel data set we use in the regression analysis but collapsed at the household level.

## **3.5 Empirical Strategy**

Cross-sectional comparisons between schools with and without electricity are likely to deliver inconsistent estimates of the effects of electrification on student learning because these schools may also differ in other (unobserved) dimensions. We avoid such comparisons by taking advantage of the rapid increase in electricity coverage induced by the roll-out of PER, and using school panel data to control for unobserved time-invariant characteristics that may determine both PER placement and outcomes. In particular, we adopt a Fixed-Effects (FE) approach to estimate the impact of PER on student learning by using within-school variation over time in access to electricity (Imbens and Wooldridge 2009).

For simplicity, we only refer to schools throughout this section though we later use individual-level panel data. To fix ideas, let s = 1, 2, ..., N, j = 1, 2, ..., M, and t = 1, 2, ..., T denote, schools, districts and years, respectively. This setup lead us to estimate the following equation:

$$y_{sjt} = \alpha_s + \alpha_t + \beta P E R_{jt} + X'_{sjt} \Gamma + \mu_{sjt}$$
(3.1)

where  $y_{sjt}$  is student learning (as measured by tests scores in Math and Reading) of school s located in district j in period t. Unobserved school heterogeneity is captured by  $\alpha_s$ . Year-specific effects are denoted by  $\alpha_t$ .  $PER_{jt}$  indicates that, in district j, at least one electrification project had been concluded by year t. The vector  $X_{sjt}$  includes time-varying measures of the school's teaching staff. The error term is denoted by  $\mu_{sjt}$  and is clustered at the school level to allow for correlation within schools over time.

The first question we seek to answer with our analysis is whether PER has an impact on student learning, after controlling for school unobserved heterogeneity, and other control variables. Thus, in equation (1),  $\beta$  is the parameter of interest. The inclusion of  $alpha_s$  in the equation implies that  $\beta$  is estimated using within-school variation over time (Angrist and Pischke 2009). As noted by Duflo et al. (2008), this coefficient should be interpreted as the overall effect (total derivative) of providing electricity at the district level rather than seeing it as the effect of

having electricity, holding everything else constant (partial derivative)⁷. In this sense,  $\beta$  is not a structural parameter, but it is informative to policy makers because it measures the difference in outcomes between schools which receive PER and those which do not.

A second related question is whether schools exposed to electricity for longer periods experience differential effects on learning. To test whether this is the case, we construct a measure of temporal exposure to PER and estimate a second equation:

$$y_{sjt} = \lambda_s + \lambda_t + \rho EXPOSURE_{jt} + X'_{sit}\Psi + \varepsilon_{sjt}$$
(3.2)

where  $EXPOSURE_{jt}$  measures the temporal difference between the learning assessment (i.e. the exam) and the year of conclusion of the electrification project. For instance, if a given project was concluded in January of 2008, and the test was administered in December of 2009, then we would say that this school had been exposed to PER for 24 months⁸. The effect of one additional year of exposure to PER is denoted by  $\rho$ . This coefficient is relevant to check whether schools with longer periods of exposure to PER experience larger impacts.

In both equations, we split the sample by gender (learning of male and female students), motivated by the heterogeneous labor impacts of PER on adult men and women documented by Dasso and Fernandez (2015). In addition, all our regressions include region-by-year dummies (interactions of dummies for regions with dummies for years) to control for region-specific time changes that could affect outcomes⁹. Finally, in all our regressions with these data, we weight each observation (school) by the number of test takers in each school at baseline (in year 2007).

Before presenting the results, we discuss the main threat to our empirical strategy. The key assumption behind our estimations is the absence of differential trends among control and treated districts in the pre-intervention period. This

⁷As shown in other contexts (Das et al. 2013), this distinction matters for policy discussions or external validity concerns because the treatment can have indirect effects that could offset each other.

⁸If there were two (or more) projects in a given district, we use the date of the project that was concluded first.

⁹Peru has 25 regions, which are divided in provinces. Each province is subdivided in districts.
means that, even after controlling for unobservable time-invariant characteristics, our estimates could still be capturing pre-treatment differences in educational outcomes. More specifically, the roll-out of PER could be correlated with prevailing time-varying differences in school enrollment, or educational expenditures, and these differences would, in turn, mechanically translate into post-program differences.

To address this concern, we need data on educational outcomes from the preintervention period (before 2007). However, both ENAHO panel data and ECE school-level data are only available from 2007 onwards. Given this data limitation, we use cross-sectional data from two different data sets. For the individual-level data, we use multiple ENAHO cross-sectional samples, and consider the same educational outcomes (enrollment and education-related expenditures) described in the previous section. For the school-level data, we use information on student population from the school census, which is conducted by the Ministry of Education. In particular, we consider the overall student population (number of students in each school, across all grades), the number of students passing their grade (not failing), and the number of students in second grade (when they take the national standardized test). In both data sets, we have information for three years of the pre-intervention period: 2004, 2005 and 2006.

To formally test whether program placement between 2007-2010 is correlated with pre-intervention educational outcomes, we regress each outcome variable on a future treatment indicator for each pre-intervention year. Finding significant differences between districts that have not been treated yet, should raise concerns on the validity of our empirical strategy. Tables 3.3 and 3.4 present the point estimates from these regressions. In both tables, we find that all coefficients are close to zero (and small relative to the mean) and statistically insignificant. This evidence is reassuring because it suggests that pre-treatment time-varying differences are not driving our results.

## **3.6 Results**

#### **3.6.1** Electrification and Test Scores

We start by discussing whether the program affects school learning. The national standardized test has three scales: high (proficient), intermediate (partially proficient) and low (not proficient). For each subject (Math and Reading), we look at the fraction of students in each level.

Table 3.5 shows the effects of PER on learning for male students. In Panel A, we report the difference in outcomes between schools in treated and control districts. In all columns, the point estimates are small in magnitude and not statistically significant, suggesting that the average impact of the intervention on learning among male students is close to zero. In Panel B, we report the differential impact of longer exposure to the program, conditional on being treated. The point estimates in columns 1 and 3, show that schools with longer exposure to improved electricity have less students in the lowest level and more students in the highest level of the Math assessment than schools with less exposure. The estimated effects imply that, after 12.5 months of improved access to electricity, the share of male students in the highest level of Math increases by 1 percentage point. This improvement represents a 12 percent increase of the average fraction of proficient students of Math in control schools. In column 6, we also find a positive impact of longer exposure on the fraction of students achieving the highest grade in the Reading assessment. This coefficient is slightly larger than the estimated impact on Math learning.

Results for female students are shown in Table 3.6. In Panel A, we find that in most cases, we cannot reject the null hypothesis of zero impact, although one point estimate (in column 5) is significant at the 10 percent level. This coefficient indicates that the program is associated with a decline in the percentage of students achieving the intermediate level in the Reading exam. In Panel B, the point estimates are statistically significant in columns 3 and 6. These coefficients show that both Math and Reading learning improve with longer exposure to the program. These figures imply that, after 16 and 14 months of treatment exposure, respectively, proficiency rates in Math and Reading increase by 1 percentage point.

Taken together, our results suggest that schools with better access to electricity do not experience immediate learning gains. Interestingly, there are small positive effects as treatment exposure increases. In the long run, the net impact of electrification on student learning would depend on whether the effect of temporal exposure is monotonic and persistent.

#### **3.6.2** Enrollment and Educational-related Expenditures

In order to get a better understanding of the impacts of PER, we now turn to explore the effects of improved access to electricity on enrollment and educational expenditures using household panel data. We do so because household behavior may change with the intervention, and these responses could indirectly affect student learning. For instance, Dasso and Fernandez (2015) document an increase in labor earnings among adult women living in treated districts. Thus, we can expect that treated families increase human capital investments in children.

Table 3.7 shows our results using the male sample of children (3-18 years old in year 2007). For each outcome variable, we split the sample in four age groups: 1) all ages; 2) between 3 and 5 (pre-primary level); 3) between 6 and 12 (primary level); and 4) between 13 and 18 (secondary level). We present the coefficients on PER and EXPOSURE in Panels A and B, respectively. Consistent with previous studies (see Glewwe et al. 2012), the point estimates in Panel A suggest that living in a PER district does not affect enrollment (columns 1-4) nor educational expenditures (columns 5-8). In Panel B, we see that, conditional on being treated, temporal exposure to PER does not affect enrollment nor educational expenditures (columns 1 and 5).

We present analogous estimates for the female sample in Table 3.8. In Panel A, most estimates suggest that improved access to electricity does not have an impact on enrollment (column 1) or expenditures (column 5). In Panel B, we cannot reject the null hypotheses of zero impact among girls of all ages (columns 1 and 5). Although a few estimates are statistically significant (e.g. positive impact on enrollment among girls aged 3-5), the overall picture that emerges from our analysis is that better access to electricity is not enough to change enrollment

rates or household educational expenses.

### 3.6.3 Robustness Checks

In this sub-section, we explore whether the estimated impacts of PER are robust to the inclusion of additional control variables. In particular, we consider two confounding interventions. During our study period (2007-2010), we are aware of two programs that also arrived to some rural districts from our sample, and could have affected educational outcomes.

First, the Peruvian conditional cash transfer (CCT) program, named "*Juntos*", rapidly reached more than 600 rural districts between 2005 to 2010. As it is common in these programs, the female head of the household receives a monthly stipend conditional on sending her children to school and taking them to health centers on a regular basis. To the extent that the presence of Juntos could increase school enrollment, and educational expenditures (through its cash payments), it is important to rule out that our results are not driven by Juntos' conditions nor its cash benefits even though PER and Juntos were rolled-out without any coordination¹⁰.

Second, the Ministry of Education launched the "One Laptop per Child (OLPC)" program in 2008, and within that year, it delivered around 40,000 laptops to children from 500 primary schools in rural areas. As of today, the Peruvian case is one of the largest interventions in the world made by the "One Laptop per Child" initiative (Cristia et al. 2017). OLPC targeted around 1,900 small primary schools, located in the poorest regions of Peru. Its implementation had two waves. In the first one, districts with both access to electricity and internet connection were prioritized. In the second wave, the former requirement was kept but the latter was abandoned because most poor districts lacked internet connectivity. Therefore, OLPC implementation was, by construction, correlated with PER placement even though both programs were run independently.

The presence of these two interventions in PER districts could introduce bias in our main estimates. To address these concerns, we redo the analysis but now

¹⁰During 2005-2012, Juntos was affiliated to the Office of the Council of Ministers. The program pursued different objectives from PER and used different criteria for its roll-out (e.g., children chronic malnutrition, exposure to violence during the 1980's armed conflict).

controlling for the presence of Juntos and OLPC. Administrative data from Juntos roll-out from 2005 to 2011 was provided by Juntos officials upon request from the authors. These data indicates the year when the program arrived at a given district. Administrative data from OLPC roll-out (including both waves) was provided by the General Office of Technology for Education, at the Ministry of Education. These data indicates the year when OLPC arrived at a given school.

We match these two data sets on programs' roll-outs with our ECE and ENAHO samples. In the emprical analysis using ECE data, we also control for Juntos at the district level, but we are able to control for OLPC at the school level. That is, we include a dummy equal to 1 if Juntos is in the district and 0 otherwise, and a dummy that takes the value of 1 when the school is treated by OLPC and 0 otherwise. In the analysis using ENAHO data, we control for the presence of these programs at the district level. That is, we include a dummy for each program that is equal to 1 if the program arrived to the district and 0 otherwise.

In Tables C2 and C3 in the Appendix, we report the estimated effects of PER on learning for male and female students, respectively. The fact that results remain unchanged after controlling for both programs suggest that these interventions are not biasing our main estimates. Tables C4 and C5 present the estimated impacts of improved access to electricity on school enrollment and educational expenditures, after controlling for Juntos and OLPC. Once again, the point estimates are virtually equal to those in Tables 3.7 and 3.8.

Therefore, our main results are robust to the inclusion of two interventions that could affect educational outcomes in rural districts. The stability of our estimates is in line with previous studies that have separately estimated the effects of Juntos on enrollment and attendance (Perova and Vakis 2012), and the effects of OLPC on tests scores (Cristia et al. 2017) and have found little evidence of positive impacts.

## 3.7 Conclusion

Improving student learning and providing better access to basic services such as electricity are top policy priorities in developing countries. At the same time, there is an open academic debate on whether school physical resources are relevant for

learning. This study aims to provide new evidence on this issue by analyzing the impact of a nation-wide electrification program on educational outcomes in rural Peru.

To do so, we take advantage of an unusually rapid expansion of a public intervention that increased access to electricity in rural Peru. Between 2007-2010, 554 electrification projects (mostly transmission lines, and to a much lesser extent hydropower plants and photovoltaic solar systems) reached several rural districts in the country, leading to substantial improvements in electricity coverage in only a few years.

We use school-level panel data on Math and Reading test scores administered by the Ministry of Education between 2007-2015 to report heterogeneous effects of improved access to electricity. In most cases, we cannot reject the null hypothesis of no impact on student learning. However, among treated schools, longer treatment exposure improves scores in Reading and Math for boys and girls. We speculate that, if such positive effects are persistent, electricity provision can slowly produce gains in student learning over the long run.

Therefore, from a policy perspective, efforts to increase access to electricity should not be abandoned. Our study presents empirical evidence showing that electrification programs can generate positive impacts on learning though these effects are not immediate. In addition, other studies have shown that electrification improves other economic outcomes (e.g. employment) that are favorable for rural households.

## **Tables and Figures**





<u>Source</u>: ENAHO panel sample 2007-2010. Only includes rural areas. Treatment districts include rural areas with electrification projects funded by PER. Control districts consist of rural areas without any electrification project funded by PER.



Figure 3.2: Access to electricity in rural primary schools. Peru 2007-2015

<u>Source</u>: ECE school-level panel 2007-2015. Only includes rural areas. Treatment districts include rural areas with electrification projects funded by PER. Control districts consist of rural areas without any electrification project funded by PER.



Figure 3.3: Electrification projects by year of conclusion at the district level, 2007-2010

Year	Investment
	(in million US\$)
2004	39,67
2005	47,03
2006	35,30
2007	79,64
2008	86,91
2009	160,12
2010	190,55
Total	639,21

Table 3.1: Public investment in electrification projects, 2004-2010

Source: National Plan of Rural Electrification. Ministry of Energy and Mining (2015).

	Control	Treatment	p-value for t-test
Variables in year 2007	(1)	(2)	on: (1)=(2)
ECE panel data:			
Number of students	13.36	13.96	0.018
(in 2nd grade)	(0.193)	(0.158)	
% of male students	0.518	0.514	0.422
(in 2nd grade)	(0.004)	(0.003)	
Complete teaching staff	0.192	0.163	0.015
(one teacher per grade)	(0.009)	(0.007)	
% of teaching staff with tenure	0.232	0.209	0.024
	(0.008)	(0.006)	
ENAHO panel data:			
HH head is male	0.81	0.85	0.114
	(0.018)	(0.015)	
HH head's age	50.47	48.74	0.078
	(0.738)	(0.651)	
HH head has no formal education	0.11	0.14	0.277
	(0.014)	(0.014)	
HH head's maternal language is Spanish	0.54	0.56	0.556
	(0.022)	(0.020)	
HH head's monthly income (in Soles)	245.50	227.20	0.478
	(18.94)	(17.31)	

Table 3.2: Mean comparisons between control and treatment groups in 2007

<u>NOTE</u>: Standard errors are shown in parentheses.

Dep. variable:		Student			Students passing			N. of students	
		Population			their grade			in 2nd grade	
Year:	2004	2005	2006	2004	2005	2006	2004	2005	2006
	(1)	(2)	(3)	(4)	(5)	(9)	(2)	(8)	(6)
PER	0,985	0,815	0,107	0,299	0,238	0,373	0,183	0,158	0,176
	(0.989)	(0.992)	(0.103)	(0.410)	(0.415)	(0.433)	(0.108)	(0.112)	(0.113)
R-squared	0,0361	0,0363	0,0353	0,0347	0,0345	0,0337	0,0361	0,0357	0,0351
N. of schools	12.285	12.580	12.691	12.285	12.580	12.691	12.285	12.580	12.691
Mean of dep. variable	166,371	163,848	162,744	70,375	69,876	69,844	18,235	17,722	17,324

Table 3.3: Placebo effects on school-level characteristics during the pre-treatment period

NOTE: Robust standard errors clustered at the district level are shown in parentheses. Each coefficient comes from a separate regression. Dependent variables: i); ii), All

regressions use school census data, and restrict the sample to schools in rural districts (PER and non-PER).

Dep. variable:		Enrollment		Log of	educ. exper	nditures
Year:	2004	2005	2006	2004	2005	2006
	(1)	(2)	(3)	(4)	(5)	(6)
PER	0.0019 (0.0275)	-0.0107 (0.0277)	-0.0213 (0.0274)	-0.0244 (0.0596)	0.0028 (0.0628)	-0.0052 (0.0577)
Observations	15,030	15,752	15,684	15,030	15,752	15,684
R-squared	0.0094	0.0074	0.0127	0.0434	0.0470	0.0347
Mean of dep. variable	0.632	0.639	0.634	3.153	3.210	3.248

Table 3.4: Placebo effects on individual-level outcomes during the pre-treatment period.

<u>NOTE</u>: Robust standard errors clustered at the district level are shown in parentheses. Each coefficient comes from a separate regression. Dependent variables: i) enrollment is equal to one if the individual is enrolled in school and zero otherwise; ii) educational expenditures per child include: uniform and shoes, textbooks, materials, fees, and parent's associations. All regressions use ENAHO annual cross-sections, and restrict the sample to individuals below 18 years old, living in rural districts (PER and non-PER) at the time of the survey.

Subject:		Math			Reading	
% of students in the:	Lowest level (1)	Int. Level (2)	Highest level (3)	Lowest level (4)	Int. Level (5)	Highest level (6)
Panel A:						
PER	0.002	-0.005	0.003	0.009	-0.010	0.001
	(0.010)	(0.008)	(0.006)	(0000)	(00.0)	(0.005)
R-squared	0.059	0.031	0.059	0.156	0.057	0.103
Schools	4,246	4,246	4,246	4,246	4,246	4,246
Mean of dep. variable	0.616	0.285	0.0988	0.421	0.469	0.110
Panel B:						
Exposure (in months)	-0.0011*	0.0003	$0.0008^{**}$	-0.0008	-0.0003	$0.0010^{***}$
	(0.0006)	(0.0005)	(0.0003)	(0.0005)	(0.0005)	(0.0004)
R-squared	0.0638	0.0369	0.0673	0.1540	0.0594	0.1099
Schools	2,674	2,674	2,674	2,674	2,674	2,674

Table 3.5: Effects of electrification on 2nd-graders learning. Boys' sample

(test takers) in each school at baseline (year 2007). Dependent variables in columns (1), (2) and (3): percentage of male students in the lowest, intermediate and highest proficiency level fixed-effects, the number of students, an indicator for complete teaching staff, and the share of teaching staff with tenure. Each observation is weighted by the number of male students exam, respectively. Panel A includes schools from both control and treated districts. Panel B only includes schools which received, at least, one electrification project in their district. in the Math exam, respectively. Dependent variables in columns (4), (5) and (6): percentage of male students in the lowest, intermediate and highest proficiency level in the Reading chool and year NOTE: Robust stand

$ \begin{tabular}{ c c c c c c c c c c c c c c c c c c c$		Math			Reading	
Panel A:         0.010         -0.010         0.000         0.01           PER $(0.010)$ $(0.009)$ $(0.006)$ $(0.01)$ R-squared $(0.010)$ $(0.009)$ $(0.006)$ $(0.01)$ R-squared $0.064$ $0.030$ $0.068$ $0.14$ Schools $4,079$ $4,079$ $4,079$ $4,079$ Mean of dep. variable $0.620$ $0.281$ $0.0994$ $0.41$ Panel B: $0.0012^{**}$ $0.0006$ $0.0006^{*}$ $-0.00$ R-squared $0.0006$ $0.0006$ $0.0006^{*}$ $-0.00$	the: Lowest level (1)	Int. Level (2)	Highest level (3)	Lowest level (4)	Int. Level (5)	Highest level (6)
PER $0.010$ $-0.010$ $0.000$ $0.01$ R-squared $(0.010)$ $(0.009)$ $(0.006)$ $(0.01)$ R-squared $0.064$ $0.030$ $0.068$ $0.1^2$ Schools $4.079$ $4.079$ $4.079$ $4.07$ Mean of dep. variable $0.620$ $0.281$ $0.0994$ $0.41$ Panel B: $0.0006$ $0.0006$ $0.0006^*$ $-0.00$ Panel B: $0.0006$ $0.0006$ $0.0006^*$ $-0.00$ R-squared $0.0706$ $0.0382$ $0.0006^*$ $-0.00$						
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	0.010	-0.010	0.000	0.014	-0.017*	0.003
R-squared     0.064     0.030     0.068     0.14       Schools     4.079     4.079     4.079     4.07       Mean of dep. variable     0.620     0.281     0.0994     0.41       Panel B:     0.0006     0.0006     0.0006*     -0.00       R-squared     0.0006     0.0005     0.0003     0.014	(0.010)	(0.00)	(0.006)	(0.010)	(0.009)	(0.006)
Schools     4,079     4,079     4,079     4,079       Mean of dep. variable     0.620     0.281     0.0994     0.41       Panel B:     0.0006     0.0006     0.0006*     -0.00       Exposure (in months)     -0.0012**     0.0005     (0.0003)     (0.000       R-squared     0.0706     0.0382     0.0681     0.14	0.064	0.030	0.068	0.140	0.056	0.089
Mean of dep. variable         0.620         0.281         0.0994         0.41           Panel B:         -0.0012**         0.0006         -0.00         -0.00           Exposure (in months)         -0.0012**         0.0006         -0.00         -0.00           R*squared         0.0706         0.0382         0.0681         0.14	4,079	4,079	4,079	4,079	4,079	4,079
Panel B:       Exposure (in months)       -0.0012**       0.0006       -0.00         R-squared       0.0006)       (0.0005)       (0.0003)       (0.00         R-squared       0.0706       0.0382       0.0681       0.14	iable 0.620	0.281	0.0994	0.416	0.473	0.111
Exposure (in months) -0.0012** 0.0006 0.0006* -0.00 (0.0005) (0.0003) (0.000 R-squared 0.0706 0.0382 0.0681 0.14						
(0.006)  (0.0005)  (0.0003)  (0.003)  (0.0081)  0.14	nths) -0.0012**	0.0006	$0.0006^{*}$	-0.0009	0.0002	0.0007*
R-squared 0.0706 0.0382 0.0681 0.14	(0.0006)	(0.0005)	(0.0003)	(0.0006)	(0.0006)	(0.0004)
	0.0706	0.0382	0.0681	0.1434	0.0627	0.0895
Schools 2,500 2,500 2,500 2,500 2,500 2,500 2,50	2,556	2,556	2,556	2,556	2,556	2,556

Table 3.6: Effects of electrification on 2nd-graders learning. Girls' sample

fixed-effects, the number of students, an indicator for complete teaching staff, and the share of teaching staff with tenure. Each observation is weighted by the number of female students Reading exam, respectively. Panel A includes schools from both control and treated districts. Panel B only includes schools which received, at least, one electrification project in their (test takers) in each school at baseline (year 2007). Dependent variables in columns (1), (2) and (3): percentage of female students in the lowest, intermediate and highest proficiency NOTE: Robust standard errors clustered at the district level are shown in parentheses. Each coefficient comes from a separate regression. All regressions control for school and year level in the Math exam, respectively. Dependent variables in columns (4), (5) and (6): percentage of female students in the lowest, intermediate and highest proficiency level in the district.

Dependent variable:		Enro	llment		Log	of educatio	nal expend	tures
Age group in 2007:	All	3-5	6-12	13-18	All	3-5	6-12	13-18
	(1)	(2)	(3)	(4)	(5)	(9)	6	(8)
Panel A:								
PER	0.004	-0.024	-0.005	0.009	-0.091	-0.183	-0.100	-0.176
	(0.020)	(0.062)	(0.014)	(0.047)	(0.00)	(0.238)	(0.083)	(0.202)
Observations	3,283	545	1,663	1,075	3,283	545	1,663	1,075
R-squared	0.045	0.347	0.067	0.208	0.076	0.534	0.146	0.177
N. of individuals	829	138	419	272	829	138	419	272
Mean of dep. variable in control districts	0.708	0.615	0.819	0.585	3.859	2.994	4.274	3.629
Panel B:								
Exposure (in months)	0.003	-0.002	0.001	$0.008^{**}$	0.000	-0.027	-0.010*	0.025
	(0.002)	(0.007)	(0.002)	(0.004)	(0.007)	(0.024)	(0.006)	(0.016)
Observations	1,989	369	1,018	602	1,989	369	1,018	602
R-squared	0.062	0.380	0.084	0.295	0.096	0.533	0.180	0.265
N. of individuals	503	94	257	152	503	94	257	152

Table 3.7: Effects of electrification on enrollment and educational expenditures. Boys' sample

NOTE: Robust standard errors clustered at the district level are shown in parentheses. Each coefficient comes from a separate regression. All regressions control for individual and year fixed-effects. Dependent variables: i) enrollment is equal to

one if the individual is encolled in school and zero otherwise; ii) educational expenditures per child include: uniform and shoes, textbooks, materials, fees, and parent's associations. Panel A includes male individuals from both control and treated

districts. Panel B only includes male individuals who received, at least, one electrification project in their district.

Dependent variable:		Enroll	lment		Log	g of educati	onal expend	itures
Age group in 2007:	All	3-5	6-12	13-18	Αll	3-5	6-12	13-18
1	(1)	(2)	(3)	(4)	(2)	(9)	(2)	(8)
Panel A:								
PER	0.034	$0.106^{*}$	0.039	-0.065	0.010	0.030	0.162	-0.422**
	(0.024)	(0.063)	(0.025)	(0.043)	(0.110)	(0.269)	(0.09)	(0.191)
Observations	3,052	590	1,637	825	3,052	590	1,637	825
R-squared	0.057	0.371	0.083	0.233	0.101	0.500	0.176	0.226
N. of individuals	773	149	414	210	773	149	414	210
Mean of dep. variable in control districts	0.718	0.654	0.815	0.578	3.724	2.962	4.204	3.344
Panel B:								
Exposure (in months)	0.002	0.002	-0.002	0.004	0.006	0.001	-0.015**	$0.038^{**}$
	(0.002)	(0.005)	(0.002)	(0.004)	(0.007)	(0.023)	(0.007)	(0.016)
Observations	1,747	339	942	466	1,747	339	942	466
R-squared	0.117	0.458	0.148	0.316	0.155	0.568	0.251	0.344
N. of individuals	444	86	239	119	444	86	239	119

Table 3.8: Effects of electrification on enrollment and educational expenditures. Girls' sample

one if the individual is enrolled in school and zero otherwise; ii) educational expenditures per child include: uniform and shoes, textbooks, materials, fees, and parent's associations. Panel A includes female individuals from both control and treated NOTE: Robust standard errors clustered at the district level are shown in parentheses. Each coefficient comes from a separate regression. All regressions control for individual and year fixed-effects. Dependent variables: i) enrollment is equal to

districts. Panel B only includes female individuals who received, at least, one electrification project in their district.

# Appendix C

Table C1: Effects of P	ER on acces	ss to electric	ity at schoo	l/home
Dependent variable:		Access to	electricity	
	Access a	at school	Access	at home
	(1)	(2)	(3)	(4)
PER	0.074***		0.062***	
	(0.010)		(0.020)	
Exposure (in months)		0.002***		0.005***
		(0.0007)		(0.002)
R-squared N. of schools/households	0.124 4,246	0.162 2,674	0.086 698	0.146 401

<u>NOTE</u>: Robust standard errors clustered at the school/household level are shown in parentheses. Each coefficient comes from a separate regression. All regressions control for school/household fixed effects and year dummies. The dependent variable is equal to 1 if the school/household has electricity and 0 otherwise. In Columns 1 and 2, each observation is weighted by the number of students in each school at baseline (year 2007). Column 1 includes schools from both control and treated districts. Column 2 only includes schools which received, at least, one electrification project in their district. Column 3 includes households from both control and treated districts. Column 4 only includes which received, at least, one electrification project in their district.

1 able CZ: Effects of F	EK on test score	ss using ECE	data, atter contr	othing for Junto	s and ULPC.	Boys' sample
Subject:		Math			Reading	
% of students in the:	Lowest level	Int. Level	Highest level	Lowest level	Int. Level	Highest level
	(1)	(2)	(3)	(4)	(5)	(9)
Panel A:						
PER	0.002	-0.005	0.003	0.009	-0.010	0.001
	(0.010)	(0.008)	(0.005)	(0.00)	(0.009)	(0.005)
R-squared	0.059	0.031	0.059	0.156	0.057	0.103
Schools	4,246	4,246	4,246	4,246	4,246	4,246
Mean of dep. variable	0.616	0.285	0.0988	0.421	0.469	0.110
Panel B:						
Exposure (in months)	-0.0011*	0.0003	$0.0008^{**}$	-0.0008	-0.0002	$0.0010^{***}$
	(0.0006)	(0.0005)	(0.0003)	(0.0005)	(0.0005)	(0.0004)
R-squared	0.0639	0.0369	0.0674	0.1540	0.0595	0.1099
Schools	2,674	2,674	2,674	2,674	2,674	2,674

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fixed-effects, the number of students, an indicator for complete teaching staff, and the share of teaching staff with tenure, the presence of Juntos in the district, and the presence of OLPC percentage of male students in the lowest, intermediate and highest proficiency level in the Math exam, respectively. Dependent variables in columns (4), (5) and (6): percentage of male NOTE: Robust standard errors clustered at the school level are shown in parentheses. Each coefficient comes from a separate regression. All regressions control for school and year students in the lowest, intermediate and highest proficiency level in the Reading exam, respectively. Panel A includes schools from both control and treated districts. Panel B only in the school. Each observation is weighted by the number of male students (test takers) in each school at baseline (year 2007). Dependent variables in columns (1), (2) and (3): includes schools which received, at least, one electrification project in their district.

Table C3: Effects of P	ER on test score	is using ECE	data, after contr	olling for Juntos	s and OLPC.	Girls' sample
Subject:		Math			Reading	
% of students in the:	Lowest level	Int. Level	Highest level	Lowest level	Int. Level	Highest level
	(1)	(2)	(3)	(4)	(5)	(9)
Panel A:						
PER	0.010	-0.010	0.000	0.014	-0.017*	0.003
	(0.010)	(0.00)	(0.006)	(0.010)	(0.009)	(0.006)
R-squared	0.064	0.030	0.068	0.140	0.056	0.089
Schools	4,079	4,079	4,079	4,079	4,079	4,079
Mean of dep. variable	0.620	0.281	0.0994	0.416	0.473	0.111
,						
Panel B:						
Exposure (in months)	-0.0013**	0.0006	0.0006*	-0.0009	0.0002	0.0007*
	(0.0006)	(0.0005)	(0.0003)	(0.0006)	(0.0006)	(0.0004)
R-squared	0.0707	0.0384	0.0682	0.1435	0.0627	0.0896
Schools	2,556	2,556	2,556	2,556	2,556	2,556

NOTE: Robust standard errors clustered at the school level are shown in parentheses. Each coefficient comes from a separate regression. All regressions control for school and year
fixed-effects, the number of students, an indicator for complete teaching staff, and the share of teaching staff with tenure. Each observation is weighted by the number of female students
(test takers) in each school at baseline (year 2007). Dependent variables in columns (1), (2) and (3): percentage of female students in the lowest and highest proficiency level in the Math
exam, respectively. Dependent variables in columns (4), (5) and (6): percentage of female students in the lowest and highest proficiency level in the Reading exam, respectively. Panel A
ncludes schools from both control and treated districts. Panel B only includes schools which received, at least, one electrification project in their district.

Table C4: Effects of PER on enrollment an	id education	nal expendi	tures using	ENAHO da	ita, after coi	ntrolling fo	r Juntos and O	LPC. Boys' sample
Dependent variable:		Enrol	lment			Log of ed	ucational expe	enditures
Age group in 2007:	All	3-5	6-12	13-18	All	3-5	6-12	13-18
1	(1)	(2)	(3)	(4)	(5)	(9)	(2)	(8)
Panel A:								
PER	0.003	-0.024	-0.006	0.007	-0.077	-0.148	-0.092	-0.153
	(0.020)	(0.064)	(0.014)	(0.047)	(060.0)	(0.241)	(0.082)	(0.198)
Observations	3,283	545	1,663	1,075	3,283	545	1,663	1,075
R-squared	0.047	0.350	0.068	0.211	0.078	0.538	0.148	0.180
N. of individuals	829	138	419	272	829	138	419	272
Mean of dep. variable in control districts	0.708	0.615	0.819	0.585	3.859	2.994	4.274	3.629
Panel B:								
Exposure (in months)	0.003	-0.003	0.001	$0.008^{**}$	-0.001	-0.029	-0.011*	0.024
1	(0.002)	(0.006)	(0.002)	(0.004)	(0.007)	(0.022)	(0.006)	(0.015)
Observations	1,989	369	1,018	602	1,989	369	1,018	602
R-squared	0.063	0.388	0.086	0.295	0.100	0.542	0.183	0.268
N. of individuals	503	94	257	152	503	94	257	152

NOTE: Robust standard errors clustered at the district level are shown in parentheses. Each coefficient comes from a separate regression. All regressions control for individual and year fixed-effects, the presence of Juntos and the presence of OLPC

in the district. Dependent variables: i) enrollment is equal to one if the individual is enrolled in school and zero otherwise; ii) educational expenditures per child include: uniform and shoes, textbooks, materials, fees, and parent's associations.

Panel A includes male individuals from both control and treated districts. Panel B only includes male individuals who received, at least, one electrification project in their district.

nt variable.		Fnrol	ment			I og of er	hicational evne	puditures
2007:	ШЧ	3-5	6-12	13-18	All	3-5	ацеанона сърс 6-12	13-18 13-18
	(1)	(2)	(3)	(4)	(5)	(9)	(2)	(8)
	0.033	0.108*	0.039	-0.066	0.011	0.021	$0.166^{*}$	-0.421**
	(0.024)	(0.063)	(0.025)	(0.044)	(0.110)	(0.273)	(0.098)	(0.187)
	3,052	590	1,637	825	3,052	590	1,637	825
	0.058	0.372	0.083	0.234	0.101	0.501	0.177	0.228
uals	773	149	414	210	773	149	414	210
variable in control districts	0.718	0.654	0.815	0.578	3.724	2.962	4.204	3.344
months)	0.002	0.002	-0.002	0.004	0.005	0.001	-0.015**	$0.037^{**}$
	(0.002)	(0.005)	(0.002)	(0.004)	(0.007)	(0.023)	(0.007)	(0.016)
S	1,747	339	942	466	1,747	339	942	466
	0.117	0.459	0.148	0.319	0.155	0.569	0.252	0.349
uals	444 4	86	239	119	444	86	239	119

NOTE: Robust standard errors clustered at the district level are shown in parentheses. Each coefficient comes from a separate regression. All regressions control for individual and year fixed-effects, the presence of Juntos and the presence of OLPC

in the district. Dependent variables: i) enrollment is equal to one if the individual is enrolled in school and zero otherwise; ii) educational expenditures per child include: uniform and shoes, textbooks, materials, fees, and parent's associations.

Panel A includes female individuals from both control and treated districts. Panel B only includes female individuals who received, at least, one electrification project in their district.

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