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Essays on Health Economics and Public Policy

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Dissertation

Presented to the Faculty of the Graduate School of

The University of Texas at Austin

in Partial Fulfillment

of the Requirements

for the Degree of

Doctor of Philosophy

The University of Texas at Austin

May 2023

Acknowledgments

I would like to express my deepest gratitude to my advisor, Marika Cabral, for her tireless dedication, thorough feedback, and encouragement throughout my doctoral studies. This dissertation would not have been possible without her advice and support. I would also like to express my sincere thanks to my dissertation committee members, Michael Geruso, Cody Tuttle, and Todd Olmstead, for their insightful comments and support throughout my dissertation.

I thank my co-authors—David Beheshti, Marika Cabral, Maya Rossin-Slater, Molly Schnell, and Hannes Schwandt—for providing me with invaluable opportunities to work with them on research projects that are included, as well as not included, in this dissertation. Collaborating with them has been a valuable and joyful experience, and I have learned a lot from these experiences. Many faculty members at the University of Texas provided helpful feedback and suggestions for my research during my graduate studies. I would like to thank Leigh Linden, Richard Murphy, Gerald Oettinger, Dean Spears, Steve Trejo, and Manuela Angelucci for their comments.

I wish to acknowledge all my friends at the University of Texas for making my PhD life a joyful experience. I would especially like to thank Andrew, Changseok, Dongha, Jinyeong, Jiwon, Narae, Sadhika, Qingsong, Vova, and many others who have made my six years of PhD study all the more memorable and enjoyable.

I am sincerely grateful to my parents, Eunjoo Cha and Jong Yeol Kim, and my brother, Donghyun Kim, for their unconditional love and support. Lastly, I would like to express my profound gratitude to Minseog for bringing so much joy and happiness into my life. His support and unwavering belief in me have sustained me throughout my journey.

Essays on Health Economics and Public Policy

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The University of Texas at Austin, 2023

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Substance use disorders (SUDs) are a major public health concern both in the United States and worldwide. The three chapters of this dissertation examine the intended and unintended consequences of public policies designed to tackle SUDs.

Chapter 1 explores the short- and long-run impacts of SUD treatment on human capital accumulation and labor market outcomes among at-risk adolescents. Specifically, I study the effect of treatment center schools, which provide residential SUD treatment and have a school on site. Using administrative data that link individual-level records across multiple government agencies in Texas, I examine within-individual changes in outcomes around the time of SUD treatment with a difference-in-differences design. I find that treated students experience declines in chronic absenteeism, disciplinary action, and course failure in the first two years following SUD treatment relative to a matched comparison group. I also find positive long-term impacts on college enrollment and employment at ages 17–20. My findings suggest that SUD treatment among adolescents may have lasting consequences and is a promising tool to promote human capital development among at-risk youth.

Chapter 2, previously published in the Journal of Health Economics, investigates the consequences of "mandatory access" prescription drug monitoring programs (MA PDMPs). MA PDMPs legally require providers to access a state-level database with a patient's prescription history before prescribing controlled substances under certain circumstances. Using a difference-in-differences specification, I find strong evidence that MA PDMPs have increased heroin death rates. My results suggest that even if MA PDMPs reduce prescription opioid deaths, the decrease is offset by a large increase in illegal opioid deaths.

Chapter 3, coauthored with David Beheshti, examines the effect of MA PDMPs on non-opioid-related outcomes. While many policies exclusively target prescription opioid

misuse, PDMPs are designed to monitor the use of a wider range of prescription drugs. Using a difference-in-differences design, we show that MA PDMPs led to decreases in stimulant prescribing. In contrast, we find suggestive evidence that these policies resulted in increases in benzodiazepine prescriptions. Our findings highlight that MA PDMPs do have effects on non-opioid drug prescribing, but these effects differ substantially across drug types.

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Chapter 1

Substance Use Disorder Treatment and Human Capital: Evidence from At-Risk Youth*

1.1 Introduction

Substance use disorders (SUDs) are a major and growing public health concern in the United States, and the rate of severe events associated with substance use has been dramatically rising. From 1999 through 2020, drug overdose death rates more than quadrupled; in 2020, about 91,800 people died from drug overdose deaths, translating to an average of more than 250 deaths each day (CDC, 2021). Beyond contributing to overdose deaths, SUDs may have profound effects on all aspects of individuals' lives and may have particularly far-reaching effects on adolescents suffering from these disorders. Adolescence is a critical period for brain development and for investments in health and human capital. It is also a time when many individuals initiate and increase alcohol and other substance use,

*I am deeply grateful to Marika Cabral, Mike Geruso, and Cody Tuttle for their invaluable guidance and support. For helpful comments, I thank Daniel Ackerberg, Jori Barash, David Beheshti, Lindsey Bullinger, Youjin Hahn, Arun Kolar, Ajin Lee, Andrew Lee, Catherine Maclean, Rich Murphy, Seth Neller, Todd Olmstead, Maya Rossin-Slater, Mu Yang Shin, Jinyeong Son, Nicole Stedman, Victoria Udalova, and participants at the University of Texas at San Antonio, the APPAM 2022 Fall Research Conference, and the Southern Economic Association 2022 Conference. The research presented here utilizes confidential data from the State of Texas supplied by the Education Research Center (ERC) at The University of Texas at Austin. The views expressed are those of the authors and should not be attributed to the ERC or any of the funders or supporting organizations mentioned herein, including The University of Texas at Austin or the State of Texas. The conclusions of this research do not reflect the opinion or official position of the Texas Education Agency, the Texas Higher Education Coordinating Board, the Texas Workforce Commission, or the State of Texas. Any errors are my own.

and untreated SUDs in adolescence often persist into adulthood and may last decades (Kessler et al., 2005).

However, despite the numerous potential benefits from SUD treatment receipt during adolescence, it is estimated that less than 1 in 10 adolescents with a SUD have access to treatment (SAMHSA, 2019). As policymakers weigh expanding access to SUD treatments, it is critical to understand the effectiveness of these treatments among adolescents. However, little is known about how SUD treatment affects adolescents, in part due to data limitations and empirical challenges. This is a particularly important gap in knowledge given the large potential for SUDs in adolescence to derail an individual's life and the large prevalence of SUDs among adolescents.[1]

This paper aims to fill this gap by estimating the short- and long-run impacts of SUD treatment on adolescents' educational and labor market outcomes, focusing on a common type of SUD treatment program for adolescents—treatment center schools. These schools are residential centers that provide clinical treatment for SUDs and have a school on site. Using administrative data from Texas, I estimate the impacts of SUD treatment center schools among at-risk adolescents—specifically, youths aged 12–16 years who have previously been detained in a juvenile detention center. This population is of particular interest for two key reasons. First, SUDs are highly prevalent among juvenile detainees.[2] Second, juvenile detainees represent a large share of the adolescents served by SUD treatment center schools.[3] More generally, the juvenile justice system is a leading source of referrals to both residential and non-residential SUD treatment services for adolescents. Between 2000 and 2018, about half of all admissions to SUD treatment for adolescents nationwide were referred by the justice system.[4] Therefore, a first-order question for

[1] In 2020, 6.3% of youth aged 12 to 17 met the criteria for a SUD (SAMHSA, 2021). The rate of substance abuse is even higher among at-risk youth. For example, a third of youth aged 10 to 17 in the juvenile justice system meet the criteria for a SUD (Aarons et al., 2001; Wasserman et al., 2010).

[2] About half of juvenile detainees meet the criteria for a SUD (Teplin et al., 2002; McClelland et al., 2004; Islam et al., 2020).

[3] Roughly a third of all adolescents who attended a SUD treatment center school in Texas between 2000 and 2018 were previously detained in a juvenile detention center.

[4] The Treatment Episode Data Set - Admissions, 2000–2018.

understanding the impacts of SUD treatment programs for adolescents is to study their effects on the adolescent population involved in the juvenile justice system.

This paper uses longitudinal administrative data that link individual-level records across three state government agencies in Texas. These data cover the universe of all individuals ever enrolled in K–12 public schools in Texas, and the data include comprehensive information from individual K–12 educational records as well as linked information on juvenile detention records, SUD treatment center school attendance, college enrollment, and labor market outcomes in young adulthood. One of the key challenges in identifying the long-run impact of interventions in adolescence is the lack of data that follow individuals from adolescence to young adulthood. I overcome this challenge by using the linked data that allow me to provide a comprehensive analysis of the impacts of SUD treatment center schools. In my analysis, I include the universe of SUD treatment center schools in Texas between 1999 and 2018 and estimate the impact of these schools on short-run outcomes (e.g., attendance, course failure) and longer-run outcomes (e.g., completed secondary school education, college enrollment, and employment measured by age 20).

To estimate causal effects of attending a SUD treatment center school on short-run educational outcomes, I examine changes in outcomes around the timing of SUD treatment initiation by taking advantage of the high-frequency nature of the data. Specifically, I use a difference-in-differences approach in which I compare within-individual changes in outcomes for adolescents who entered a SUD treatment center school within six months following detention to those experienced by a matched comparison group. To form a matched comparison group for each treated individual, I focus on individuals who were detained at the same time as the treated individual and who also suffered from a SUD, but were not enrolled in a treatment center school after detention. Among these matched controls, I use "exact" and "fuzzy" matching techniques to restrict attention to matches with the same basic demographic characteristics as the treated individual (e.g., age, gender, race/ethnicity, and socioeconomic status) and to exclude those with very different values of key measures at baseline (e.g., absence rate and juvenile detention history). The assumption underlying this

research design is that, in the absence of SUD treatment center school attendance, outcomes among treated individuals would have trended similarly to those among the matched comparison group. I use the high-frequency data on educational outcomes to provide support for this assumption. Specifically, I illustrate that outcomes across the two groups evolved similarly in the months prior to detention and continued to evolve similarly after detention but before placement in the SUD treatment center school. The outcomes for the treatment group only diverge after enrollment in the treatment center school.

I find that attending a SUD treatment center school has a positive impact in the short run. Attending a SUD treatment center school reduces the share of school days that an individual is absent by 5.1 percentage points (or 28% relative to the control group mean); reduces chronic absenteeism by 12 percentage points (23%); and reduces the likelihood of not being observed within Texas public school system by 4.9 percentage points (11%) in the first two years following SUD treatment initiation. In addition, attending a SUD treatment center school leads to a 7.5 percentage point decrease in the likelihood of being disciplined in school (28%) and a 5.5 percentage point reduction in the course fail rate (16%) in the first two years following SUD treatment initiation.

I also analyze the impact of attending a SUD treatment center school on long-run outcomes—such as completed secondary school education, college enrollment, and labor market outcomes—through age 20. Since long-term outcomes are only observed once for each individual, it is not possible to analyze within-individual changes in these outcomes. Instead, I analyze long-run impacts by including match group fixed effects to compare outcomes between treated individuals and matched control individuals. In this specification, I also include additional controls—such as county fixed effects and other pre-detention characteristics—to account for any further potential differences between the treatment and control individuals.

The findings indicate that attending a SUD treatment center school has lasting positive impacts on educational and labor market outcomes through age 20. Attending a SUD treatment center school leads to a 4.4 percentage point increase in the likelihood of completing grade 10 (15.4% relative to the control group mean) and a 1.7 percentage point

increase in the likelihood of grade 11 completion (10.2%). I find no statistically significant effect on high school graduation. To summarize the effects of treatment center schools on completed secondary education, I analyze the effect on the maximum grade level completed in secondary school. These results indicate that attending a SUD treatment center school leads to 0.11 additional years of schooling in secondary school.

I also find that treatment center school attendance substantially increases college attendance and employment through age 20. Attending a SUD center school leads to a 1.3 percentage point (12%) increase in the likelihood of enrolling in college by age 20. This increase is almost entirely explained by an increase in two-year college attendance among youth who would not have attended college. SUD treatment center attendance also leads to a 2 percentage point (2.7%) increase in the likelihood of being employed at ages 17–20.[5] This estimated increase in employment is large. It is roughly a third the size of the estimated effect of moving children who live in distressed public housing to lower-poverty neighborhoods (Chyn, 2018) and twice the size of the estimated effect of a paid summer employment program (Gelber et al., 2016).

My study contributes to a growing literature quantifying the returns to SUD treatment services. Recent studies document that access to SUD treatment facilities leads to reductions in local crime, emergency visits, and drug overdose deaths (e.g., Bondurant et al., 2018; Corredor-Waldron and Currie, 2022; Swensen, 2015; Wen et al., 2017).[6][7] Prior work has focused on examining the short-run impacts of SUD treatment using aggregate data and has focused on SUD treatment available in the community at large—rather than SUD

[5] I find no evidence that SUD treatment center school attendance increases earnings at ages 17–20, though an important limitation is that earnings are only measured through late adolescence; any increases in lifetime earnings because of the estimated increases in educational attainment may not appear until later ages.

[6] Horn et al. (2021) investigate the impacts of SUD treatment centers on local property values and find no evidence that SUD treatment centers negatively affect local property values. Arora and Bencsik (2021) show that connecting eligible individuals arrested for drug possession to SUD treatment services reduces re-arrest rates.

[7] Other work has documented the link between mental health and human capital (e.g., Busch et al., 2014; Cuellar and Dave, 2016; Currie and Stabile, 2006; Heller et al., 2017), labor market outcomes (e.g., Biasi et al., 2021), and criminal behavior (e.g., Deza et al., 2022; Heller et al., 2017; Jácome, 2020).

treatments aimed specifically at the adolescent population suffering from SUDs. This paper advances this literature in two key ways. First, this study provides an in-depth analysis of the impact of SUD treatment programs on adolescents, focusing on human capital accumulation and later employment outcomes. It is particularly important to understand the impacts of interventions targeted toward adolescents, given that interventions earlier in life tend to have larger impacts and are often more cost-effective than interventions targeting adults (Hendren and Sprung-Keyser, 2020).

Second, this paper provides the causal estimates of the long-run effects of SUD treatments more generally. My findings illustrate that SUD treatment among at-risk adolescents increases educational attainment in the short- and long-run, and back-of-the-envelope calculations suggest that projected increases in lifetime earnings based on these increases in educational attainment alone may be large enough to offset a substantial share of the costs of this treatment.

More broadly, my work contributes to a wider literature investigating the effect of interventions for at-risk children or children from disadvantaged backgrounds. Prior studies have investigated the impacts of interventions such as summer youth employment programs (e.g., Gelber et al., 2016), placement in disciplinary schools (Meiselman and Verma, 2021), moving children to lower-poverty neighborhoods (e.g., Chyn, 2018), and placing children who are abused or neglected into foster care (e.g., Bald et al., 2022; Doyle, 2007). Because SUDs are prevalent among disadvantaged youth, understanding returns to SUD treatment among this population is of particular interest to policymakers. The findings of this paper demonstrate that increasing access to SUD treatment center schools could be a promising tool to promote human capital development among at-risk youth.

Finally, my paper directly addresses issues in an active policy landscape. In response to the worsening substance use epidemic, President Biden's budget for fiscal year 2023 proposes historic investments to address the growth in SUDs, including large increases in funding for treatment services for adults and adolescents (White House, 2022). While it is increasingly important to understand the returns to SUD treatment programs, little is known about the impacts of SUD treatments—especially the impacts of these programs on

adolescents. By providing causal estimates of the short- and long-run impact of SUD treatment center schools—a resource that is critical to at-risk adolescents with severe SUDs—this paper illustrates that SUD treatment services for adolescents may not only positively impact human capital development but also provide far-reaching benefits to the affected individuals and society more broadly.

1.2 Background

1.2.1 Substance Use Disorder Treatment Center Schools

SUD occurs when "the recurrent use of alcohol and/or drugs causes clinically significant impairment, including health problems, disability, and failure to meet major responsibilities at work, school, or home" (SAMHSA, 2022). SUD treatment for adolescents is delivered in many different settings, which fall within two categories: non-residential (e.g., early intervention services, outpatient treatment) and residential (residential/inpatient treatment, medically managed intensive inpatient treatment). Roughly a third of youth admissions to SUD treatment are for residential services,[8] which are aimed at individuals with severe SUDs.

One of the most common types of residential SUD treatment for adolescents is a SUD treatment center school. As of 2020, SUD treatment center schools represent 83% of all residential beds for SUD treatment among adolescents in Texas. SUD treatment center schools are nonhospital, licensed residential treatment centers that have a school on site. While there is some variation in the specific services provided by residential SUD treatment centers, all centers provide safe housing and medical care in a 24-hour supervised setting. These centers offer intensive care and support, including comprehensive evaluations, therapy, and an individualized treatment plan to meet individuals' specific behavioral and mental health needs (Somers et al., 2021). Beyond standard residential SUD residential treatment services, SUD treatment center schools also provide educational services based on

[8]Treatment Episode Data Set, 2000–2018.

standard, age-appropriate curriculum.[9][10] Compared with regular public schools, classrooms in these treatment center schools have low student-to-teacher ratios, allowing teachers to provide as much one-to-one assistance as possible (Letourneau, 2014).

1.2.2 Juvenile Detention and Assignment to a Treatment Center School

In this paper, I estimate the impacts of SUD treatment center schools among at-risk adolescents who have previously been detained in a juvenile detention center. Juvenile detention centers are primarily used to temporarily hold juveniles while they await a court hearing, disposition, or placement in a different facility.[11] In 2019, about 1 in 4 (26%) delinquency cases were referred to juvenile court involved detention, with the average length of stay of 27 days (Puzzanchera et al., 2017). Youth in juvenile detention have the right to education, and juvenile detention centers provide youth with access to educational programs (Umpierre, 2014).

Assignment to a SUD Treatment Center School In all states, juvenile detention facilities use mental health screening tools to identify youths with mental health and/or substance use disorders and youths who need further assessment.[12] Among juvenile detainees with an identified need for SUD treatment, some individuals are assigned to SUD treatment programs, including SUD treatment center schools. Juvenile detainees can be

[9]Coursework completed within a treatment center school leads to credits awarded by the associated school or school district, and some treatment center schools have authority to grant diplomas as well. For more details, see: `https://www.transformingyouthrecovery.org/wp-content/uploads/2017/09/ARS_The_State_of_Recovery_High_Schools_2016_Biennial_Report..pdf` (accessed October 2022).

[10]Youth in residential facilities have the right to education, regardless of the length of stay, and most residential treatment facilities have an on-site school with a standard age-appropriate education curriculum (Umpierre, 2014).

[11]For some cases (roughly 5-10%), juvenile detention centers are also used for longer-term, court-mandated treatment programs following post-trial sentencing (Jason Baron et al., 2023). Although my analysis includes all detention cases, the results are qualitatively similar if I exclude the long-term detention episodes that are in the top 10% of the distribution of the detention length.

[12]One of the most commonly implemented screening measures in juvenile justice settings is Massachusetts Youth Screening Instrument-Second Version (MAYSI-2), which is a standardized screening tool for mental health needs of detained youths. The MAYSI-2 is a 52 yes/no item screening tool and only takes 15 minutes to administer and thus can be easily used in detention facilities.

assigned a SUD treatment center school either by court order or by referral.[13] First, judges can order placement into a SUD treatment center school at the time of disposition. Second, juvenile detainees can be referred to a SUD treatment center school (either during detention or after release from detention) by other referral sources, including probation officers, healthcare providers, and schools. Among these sources, the major source of referral to youth residential SUD treatment programs is probation officers.[14][15] About 60–65 percent of justice-involved youths are mandated to probation on release, providing the juvenile probation officer a unique opportunity to connect youths to SUD treatment (Holloway et al., 2013; Hockenberry and Puzzanchera, 2020).

Access to SUD Treatment Services Judges (and probation officers) take into account the severity of an individual's SUD when deciding whether to require (or refer) the individual to attend a SUD treatment center school. However, beyond the severity of a SUD, several factors may influence judges' decisions to order (and probation officers' decisions to refer) juvenile detainees to SUD treatment. A key factor that may influence these decisions is the availability of services. While about half of juvenile detainees meet the criteria for a SUD, which is more than eight times higher than among the general adolescent population, only ten percent of youth in the juvenile justice system who need SUD treatment services are connected to appropriate care (McClelland et al., 2004; Kelly et al., 2005; Teplin et al., 2002; McClelland et al., 2004; Islam et al., 2020). Aside from the current availability in local treatment center schools, other factors cause variation in the judges' (and probation

[13]Among adolescents who entered a SUD treatment center school following detention in Texas between 1999 and 2018, about 40 percent entered the school by court order.

[14]Between 2000 and 2018, referrals from the court/justice represent the largest share (47.5%) of all admissions to youth residential treatment programs, followed by an individual (17.7%), alcohol/drug use care provider (15.4%), other community referrals (11.3%), other healthcare providers (6.4%), school (1.5%), and employer (0.14%); among referrals from the court/justice system, the largest share (47.6%) are from probation/parole, implying that probation officers is the major source of referral to residential SUD treatment among youths in the juvenile justice system (Treatment Episode Data Set-Admissions, 2000–2018).

[15]It is important to note that SUD treatment services for adolescents are very costly and funding from the juvenile justice system is crucial for accessing these services (Ebener and Kilmer, 2003). It is much harder for adolescents outside of the juvenile justice system to receive SUD treatment services, partially explaining that the juvenile justice system is the major source of referral to youth SUD treatment.

officers') decisions, such as (i) their attitudes towards youth substance use and SUD treatment services, (ii) their perceptions about the availability and quality of services, (iii) established networks between service providers and the court, and (iv) clinical backgrounds of the decision-maker in SUD treatment services (Breda, 2001; Yurasek et al., 2021).[16]

As a consequence, there may be a large variation in the rates of referral to a SUD treatment center school across judges/probation officers and across time, even conditional on youths' severity of SUDs. This institutional feature is helpful for identification because there may be substantial overlap in the support of individuals who do and do not access SUD treatment center schools. My matched difference-in-differences approach builds on this institutional feature by identifying matched control individuals who did not attend a treatment center school but who appear otherwise comparable to individuals who did attend a treatment center school.

Timing of Treatment After Detention As noted above, treatment center schools are capacity constrained. These capacity constraints may impact both whether an individual is referred to a treatment center school and the timing of enrollment in the treatment center school after being released from detention. For example, some individuals may enter the SUD treatment center school immediately after release, but it can take several weeks to several months for other individuals to enter.[17] Figure 1.1 provides a graphical illustration of the timeline from the pre-detention period to the post-SUD treatment period among adolescents who attend a SUD treatment center school after detention. I define the intermediate pre-period as the period between the timing of placement into a detention center and the timing of enrollment in a SUD treatment center school. As discussed above, the length of the intermediate pre-period varies across

[16]For instance, a probation officer may be "unsure of where to refer youth for further evaluation and ultimately just refer for mental health services or do not refer at all" (Yurasek et al., 2021).

[17]Not only capacity constraints but also other systemic barriers can affect the timing of enrollment in the treatment center school. For example, probation officers may "shop" for programs, connecting the youth to multiple interviews by multiple programs (Ebener and Kilmer, 2003). This may delay the actual SUD treatment among adolescents after the release from detention.

individuals and can be as long as several months.[18]

1.3 Data

To estimate the impact of attending residential SUD treatment centers on educational and labor market outcomes among juvenile detainees, I use individual-level administrative data from several sources. The data cover the universe of public school records in Texas. These data are then linked to information on education (K–12 education, college education), juvenile detention, residential SUD treatment center school attendance, and employment/earnings. This section describes each of the administrative data sources and outcome variables I construct for the analysis.

1.3.1 Individual Educational and Labor Market Outcomes

Educational Outcomes I use administrative microdata on educational outcomes that are obtained from two sources. First, I use data from the Texas Education Agency (TEA) that cover all students in all public K–12 schools in Texas over the academic years 1996–1997 through 2019–2020.[19] The TEA data contain information on students' attendance, graduation, type and reason for disciplinary actions, course completion and pass/fail results, standardized test scores, and a reason for leaving Texas public school system. The data further contain information on student characteristics, including age, gender, race/ethnicity, disability, and eligibility for free or reduced-price lunch.

TEA data on enrollment and disciplinary actions are available for each student and for 6 six-week grading periods in a given academic year.[20] Using these records, I construct the

[18]The length of the intermediate pre-period reflects both the length of detention and the time lag between release from detention and enrollment in a SUD treatment center. As described in Section 1.3.3, individuals in my analysis sample spend 17.3 days in a detention center on average.

[19]Although the TEA data cover the period 1992–2020, I focus on the years 1996–2020 because data on disciplinary actions are only available from 1998 onward. Because these data are used to construct key measures in my analysis, I exclude earlier years.

[20]If a student switches schools within a given six-week grading period and within the Texas public school system, the TEA data contain separate enrollment and disciplinary action records for each student and for each school.

following five outcomes: (1) a continuous absence rate, which I measure as the ratio of the number of days absent to the number of days a student is enrolled in any school in Texas public school system; (2) an indicator for chronic absenteeism, which I define as a continuous absence rate being equal to or larger than ten percent; (3) an indicator denoting being enrolled in any school in Texas public school system; (4) an indicator for whether a student is chronically absent or not in Texas public school system (which is a combined measure of (2) and (3)); and (5) an indicator for whether any disciplinary action is taken against a student in school. Note that the outcomes (1), (2), and (5) are defined by conditioning on being observed in Texas public school system, while the outcomes (3) and (4) are not. TEA data on course completion and course pass/fail results are only available at the academic year level. Using these data, I construct an additional outcome: course pass rate, which I define as the ratio of the number of courses passed relative to the number of courses completed. I also construct an indicator for graduating high school by age 20, using TEA data on whether and at what age a student graduated.

Second, I use the Texas Higher Education Coordinating Board (THECB) data that cover all students in all public and most private institutions of higher education in the state of Texas. The THECB data are linked to the TEA data at the individual level. Using the THECB data, I construct the following outcomes measured through age 20: (1) an indicator for ever having enrolled in any college; (2) an indicator for ever having enrolled in a two-year college but not in a four-year college; and (3) an indicator for ever having enrolled in a four-year college. THECB data do not contain information on out-of-state college enrollment or enrollment at some private institutions in Texas.

Labor Market Outcomes I use administrative, quarterly microdata on employment and wage for all workers covered by Unemployment Insurance (UI) obtained from the Texas Workforce Commission (TWC).[21] The TWC data are linked to the TEA and THECB data at the individual level. Using the TWC data, I construct the following outcomes at ages 17–20: (1) an indicator for being employed, measured as having positive wage in any quarter; (2) average annual earnings (including zeros), measured in 2020 dollars. When an individual is

[21]For more details, see: `https://www.twc.texas.gov/tax-law-manual-chapter-3-employer-0`.

not identified as being employed in a given year, her annual earnings are coded as zero. I do not have information on out-of-state employment.

1.3.2 Residential Substance Use Disorder Treatment Centers

Since SUD treatment center schools have a school on site, all students enrolled in these schools are included in the TEA data. I identify residential SUD treatment centers using data from the Texas Department of State Health Services (DSHS). The Texas DSHS provides a document listing all licensed SUD treatment facilities in Texas. For each facility, the data report license number, county, the name of the provider, address, the number of residential beds, the number of outpatient slots, setting(s) provided (outpatient, detoxification ambulatory/outpatient, residential detoxification, intensive residential, supportive residential), and gender and age group(s) served (adolescent, adult) for each setting. In addition, I use the Health Treatment Services Locator database provided by the Substance Abuse and Mental Health Services Administration (SAMHSA) to further obtain information on facility operation (e.g., private, public), payment/insurance/funding accepted, treatment approaches (e.g., anger management), and other service details.

Between 1999 and 2018, there were 14 residential SUD treatment center schools in Texas with a total of 428 residential beds.[22][23] As described above, the TEA K–12 education data contain detailed information on enrollment (e.g., days enrolled, days absent) for each student and for 6 six-week grading periods in a given academic year. This allows me to identify whether and for how long a student is enrolled in a school at a SUD treatment center. Appendix Figure A.2 presents the distribution of length of stay within a SUD treatment center school.[24] On average, individuals stay 49 days in a SUD treatment center

[22] Appendix Figure A.1 presents the location of 14 treatment center schools.

[23] In fact, there were 20 residential SUD treatment centers between 1999 and 2018. Six out of 20 residential SUD centers for adolescents do not have an on-site school accredited by the TEA and thus are not included in the TEA data. Most of these six facilities are either small in size or specifically designed for adolescents under exceptional circumstances. One facility is specifically designed for pregnant or newly parenting adolescents. Four facilities are small in size—the number of residential beds ranges from 12 to 16. Small facilities may choose to provide formal education through partnerships with local schools in the community rather than to provide education by an on-site school.

[24] The length of stay is winsorized at 180 days (i.e., about one academic year).

school.

1.3.3 Juvenile Detention Centers

In this paper, I estimate the impact of SUD treatment schools among at-risk adolescents who were previously detained in a juvenile detention center by comparing changes in outcomes between adolescents who did and did not attend a SUD treatment center school after detention. As mentioned above, juvenile detention centers are required to provide youth with access to education. Most juvenile detention centers have a school within their facilities, and thus are included in the TEA data. I identify 37 juvenile detention facilities in the TEA data using data from the Texas Juvenile Justice Department (TJJD) that list all registered pre-adjudication juvenile detention facilities in Texas.[25] Since the TJJD data only list currently active facilities, I use data from several additional sources, including county websites, and identify 11 additional facilities that were ever active between 1999 and 2018. The final analysis sample includes 48 juvenile detention facilities in Texas. The TEA data on enrollment allows me to identify whether and for how long a student is enrolled in a school within a juvenile detention facility in a given period. Appendix Figure A.3 shows the distribution of length of detention for 48 juvenile detention centers in my sample. The average length of detention is 17.3 days and half of the sample are detained for less than 13 days. In Appendix Figure A.4, I present trends in the number juvenile detention cases and the percentage of cases in which a juvenile enters a SUD treatment center school within a year following detention.[26]

1.4 Empirical Design

To estimate the causal effects of attending a SUD treatment center school on educational and labor market outcomes, I employ a difference-in-differences research design in which I compare within-individual changes in outcomes following SUD treatment to those experienced

[25] For more details, see: `https://www2.tjjd.texas.gov/publications/other/searchfacilityregistry.aspx` (accessed July 2022).

[26] Appendix Figure A.1 shows the location of juvenile detention centers that are included in my analysis. Note that the map only presents juvenile detention centers that are active between 2019 and 2020.

by adolescents who have the same basic demographic characteristics and suffer from a SUD, but were not enrolled in a SUD treatment center school after detention. In this section, I begin by describing my sample and the treatment group. I then discuss how I form a matched comparison group for each treatment individual. Finally, I explain my empirical strategies for the short- and long-run analyses.

Sample and the treatment group In my analyses, I focus on adolescents who are detained in a juvenile detention center at any point between ages 12 and 16 over the academic years 1999–2000 to 2017–2018.[27] I make two more sample restrictions. I exclude adolescents who are detained for more than 95 days (i.e., greater than value at 99th percentile in terms of length of detention). And, I restrict attention to individuals those who are observed in the TEA data for at least 3 six-week grading periods—about half of an academic year—during the last year prior to detention.[28] Within this sample, I define the treatment group as individuals who enter a SUD treatment center school within three grading periods (about six months) after being placed into a juvenile detention center.[29]

1.4.1 Control Group Construction

Individuals who attended a SUD treatment center school are likely to differ from those who did not attend in many aspects. For example, some of the individuals who did not attend a SUD treatment center school may not suffer from a SUD and thus not need SUD treatment. In Appendix Table A.1, I investigate how these individuals differ in observable characteristics. The table presents average individual characteristics and academic performance measured in the pre-detention period for individuals who were enrolled in a SUD treatment center school following detention (column (1)) and all individuals who were

[27] Although my sample covers the years 1996–2020, I restrict attention to detention episodes between 1999 and 2018 to follow individuals from three years before to two years after SUD treatment.

[28] About 8% of the sample are observed for two or fewer grading periods and thus are dropped by this restriction.

[29] Among adolescents who attended a SUD treatment center school within six grading periods (i.e., a year) following juvenile detention between 2000–2018, 71.2% entered the center within three grading periods. Since I do not have individual-level data on referral sources or the timing of referrals, I focus on adolescents who enter a SUD treatment center school within three grading periods after being placed into a detention center to mitigate concerns about unobserved shocks other than detention around the timing of SUD treatment.

detained during my sample period, regardless of their SUD treatment receipt (column (3)). The sixth column presents the differences between mean characteristics between these two groups, and the seventh column presents $p-$values from tests of these differences.

A comparison of columns (1) and (3) indicates that individuals who enter a SUD treatment center school differ from the average juvenile detainee in a number of observable characteristics, including demographic characteristics (e.g., gender and race/ethnicity) and academic performance (course pass rate and standardized test scores in reading and math) measured in the pre-detention period. Compared to the average juvenile detainee in Texas, individuals who enter a SUD treatment center school after detention are less likely to be female, Black, eligible for free/reduced-price lunch, and in a special education program; more likely to be White, Hispanic, and in a large central metro; and slightly older. In addition, Panels B and C show that treatment individuals have higher absenteeism, longer detention history, and lower academic performance at baseline, which may reflect that treatment individuals have a severe SUD, while some of the others may not suffer from a SUD prior to detention.

The key assumption underlying my difference-in-differences design is the parallel trends assumption. However, the substantial differences between detainees who did and did not attend a SUD treatment center school in a number of observable characteristics and academic performance at baseline raise concerns about differential trends between these individuals. To reduce these concerns, I match each treatment individual with a set of control individuals who suffered from a SUD and were similar in the basic demographic characteristics did not attend a SUD treatment center school during my sample period (1996–2020). For this match, I first restrict attention to control individuals with a SUD. I then use both exact and fuzzy matching methods together to identify individuals who have the same basic demographic characteristics as the treatment individual and exclude those who have very different values of key measures in the pre-detention period. In the baseline analysis, I focus on the control sample that I identify using both exact and fuzzy matching methods together, but in Section 1.5.3, I discuss the robustness of my results to omitting the fuzzy matching procedure or to using an alternative, non-matching based approach. Below,

I explain each step I conduct to form a matched comparison group.

Step 1. Identifying control individuals with a SUD Not all detainees need SUD treatment services. To identify control individuals who are likely to meet the criteria for a SUD, I use an indicator for being *disciplined for substance-related problems* in school during the two years prior to detention as a proxy for having a SUD.[30] As a result, my control group consists of individual who did not attend a SUD treatment center school but who were disciplined for substance-related problems during the two years prior to detention. Note that I do not make this restriction for the treatment group because I consider all treatment individuals as having a SUD regardless of their substance-related disciplinary action history.[31] However, I show that the results are very similar if I make this restriction for the treatment group as well, though the confidence intervals are slightly wider (see Section 1.5.3).

Appendix Table A.1 shows that how treatment individuals (column (1)) differ from control individuals with substance-related discipline history (column (2)) in a wide range of observable characteristics. The fourth column presents the differences between mean characteristics between these two groups, and the fifth column presents $p-$values from tests of these differences. A comparison of columns (1) and (2) for characteristics in Panel C suggests that my proxy for a SUD is successful at identifying individuals who have similar academic achievement in the pre-detention period. However, even after focusing on control individuals who are likely to be eligible for SUD treatment, I still see large differences in

[30]The TEA data on disciplinary actions contain information on the type of and the reason for each individual and for each disciplinary action. Disciplinary actions for substance-related problems are defined by combining the following discipline action reason codes: (1) marijuana or controlled substance or dangerous drug, (2) alcohol, and (iii) abuse of a volatile chemical. For more details, see: `http://ritter.tea.state.tx.us/peims/standards/1314/app_additional_information_related_to_discipline_actio.html` (accessed October 2022).

[31]Another reason is that if I make the same restriction for the treatment group, it substantially cuts the sample size and reduces statistical power. About 39% of treatment individuals are disciplined for substance-related problems in the pre-detention period, implying that 61% of them will be dropped if I make the same sample restriction for the treatment group. Given that the results are robust to making the same restriction for the treatment group (see Section 1.5.3), my baseline analysis focuses on including the full treatment sample to increase the sample size.

demographic characteristics (Panel A). Compared with individuals who have substance-related discipline history but are not enrolled in a SUD treatment center school, individuals who attend a SUD treatment center school are more likely to be female and White; less likely to be Hispanic, Black, eligible for free/reduced-price lunch, and in a special education program. These large differences in demographic characteristics motivates me to use the matching methods to further restrict the control sample to individuals who are similar in these characteristics. As noted above, I discuss the robustness of my results to omitting the fuzzy matching or to using an alternative, non-matching based approach in Section 1.5.3.

Step 2. Exact matching on basic demographic characteristics I use the exact matching on the following matching variables: (1) the timing of detention (e.g., a treatment and the matched control individuals are detained in the same grading period in the same academic year), (2) gender, (3) race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic, other), (4) age (no more than one-year difference), (5) eligibility for free/reduced price lunch (measured in the two years prior to detention), (6) indicator for being enrolled in a special education program (measured in the last two years prior to detention) and (7) urbanicity category based on county of detention center.[32][33]

Step 3. Refinement using a fuzzy matching approach Finally, I do the fuzzy matching to exclude exact matches with very different values in terms of key measures at baseline. Specifically, I use the following three variables: (1) average absence rate in the year prior to detention,[34] (2) the number of grading periods in which an individual is ever detained, measured two years prior to detention, and (3) the number of grading periods in which an individual is ever detained that is measured one year prior to detention. There are

[32]Counties are categorized using 2013 National Center for Health Statistics (NCHS) Urban-Rural Classification Scheme for Counties. This county-level scheme consists of four metropolitan (large central metro, large fringe metro, medium metro, and small metro) and two nonmetropolitan (micropolitan and noncore). For more details, see: `https://www.cdc.gov/nchs/data_access/urban_rural.htm` (accessed August 2022).

[33]The TEA data do not contain information on home address. I use county of a detention center as proxy for home address.

[34]More exactly, this is measured in the 6 six-week grading periods prior to detention.

several different ways to calculate distance between each treatment individual and a control individual. For example, a single matching variable can be used to measure the level of similarity in terms of that variable, while all fuzzy matching variables can be used together to measure overall similarity. I use the former approach to construct a comparable control group for each treatment individual, but my results are qualitatively similar if I use the latter approach instead. My fuzzy matching takes the following steps. First, I calculate the distance (i.e., the absolute value of difference) between a treatment individual and a control individual, separately for each of the three fuzzy matching variables. Then, for each matching variable, I drop control individuals with outlier distance values, defined as values greater than 90 percentile.[35]

I define a "qualified" match as an exact match with non-outlier distance values in terms of the three fuzzy matching variables. Out of 5,182 treatment individuals, 863 individuals do not have any exact matches; and 285 individuals have at least one exact match but do not have any qualified matches; and 4,034 individuals have at least one qualified match. The final analysis sample consists of the 4,034 treatment individuals and 35,714 matched control individuals.[36]

Table 1.1 reports average individual characteristics and academic performance measured in the pre-detention period for the 4,034 treatment individuals included in the final analysis sample (column (1)) and the matched control individuals (column (2)). The third column presents the differences between mean characteristics between these two groups, and the fourth column presents $p-$values from tests of these differences. The two groups are identical in characteristics used in the exact matching (except for age, for which I allow for a one-year difference), and similar in characteristics used in the fuzzy matching as well as academic performance measured in the pre-detention period, which I do not use for the matching. It is worth highlighting that my difference-in-differences research design is not based on the assumption that the two groups are identical in all dimensions. The key

[35] About 19% of the exact matches are dropped.

[36] Appendix Table A.2 reports average individual characteristics across (i) the baseline treatment sample (those with at least one qualified match), (ii) treatment individuals with at least one exact match but no qualified matches and (iii) treatment individuals with no exact matches.

identification assumption of my research design is the parallel trends assumption, and I will discuss the validity of this assumption in Section 1.5. In all regressions throughout my analyses, each control individual within a given match group is given equal weight, and these weights are summed up to one. treatment individuals are assigned a weight of one.

One might have concerns about using absence rate, which is itself used as an outcome variable, as a matching variable. There are several things to note that assuage these concerns. First, in Section 1.5.3, I show that the results are very similar if absence rate is excluded from the fuzzy matching procedure. Second, I only match on the level of average absence rate measured in the year prior to detention, not the trend in absence rate during the entire pre-detention period. This allows me to assess whether absence rate among the treatment and matched control group evolves similarly prior to juvenile detention. Third, in Section 1.5.3, I show that outcomes that are not used for the matching—including course fail rate and disciplinary action history—are very similar across the treatment and control groups both in level and trend before SUD treatment.

1.4.2 Short-Run Analysis

In the short-run analysis, I examine how residential SUD treatment impacts academic outcomes in the short run, focusing on outcomes that can be measured both before and after SUD treatment for each individual (e.g., attendance and course pass rate). For the short-run analysis, I restrict attention to individuals who were in or below grade 10 at the time of detention in order to follow adolescents two years before and after SUD treatment. As described above, I do not require that students stay in the Texas public school system before or after detention, as long as they are observed for at least 3 six-week grading periods in the last pre-detention year. I use this sample to estimate difference-in-difference models in which I measure within-individual changes in outcomes following SUD treatment, relative to the matched control individuals. My difference-in-differences specification is:

$$Y_{igt} = \rho Treatment_i \times Post_{gt} + \alpha_{gt} + \delta_i + \varepsilon_{igt}, \tag{1.1}$$

where Y_{igt} is an outcome in period t for adolescent i who is in match group g.

$treatment_i$ is an indicator for individuals who attended a SUD treatment center. For each match group, $Post_{gt}$ is defined as an indicator for periods during or after the period of the treatment individual's SUD treatment initiation. Note that this indicator turns on not only for a treatment individual but also for control individuals within the same match group in the post-SUD treatment period. I include match group–by–time fixed effects, α_{gt}, which flexibly account for time trends in the outcomes within each match group. I also include individual fixed effects, δ_i, which account for time-invariant differences between treatment and control individuals. Standard errors are clustered at the individual level. The key coefficient of interest is ρ, which summarizes the difference in the change in outcomes following SUD treatment between treatment and control individuals within each match group.

The key identifying assumption of the difference-in-differences approach is that in the absence of residential SUD treatment, outcomes would have evolved similarly for treatment and control individuals in each match group. To assess the validity of this assumption, I plot raw trends in outcomes between treatment and control individuals and conduct an event study analysis. My event study regressions take the following form:

$$Y_{igt} = \sum_{k=-12, k\neq -6}^{12} \gamma_k Treatment_i \times \mathbf{1}\{t - E_i = k\} + \sigma_{gk} + \mu_i + \nu_{igt}, \tag{1.2}$$

where E_i is the period when individual i initially received SUD treatment. $k = t - E_i$ are periods relative to the time of a treatment individual's SUD treatment initiation. A negative k denotes $|k|$ periods prior to SUD treatment initiation. Again, note that all control individuals have non-missing values for E_i and $k = t - E_i$, which are defined based on the period in which the treatment individual in their match group initiates SUD treatment. I also include a full set of match group–by–relative time fixed effects, σ_{gk}, to flexibly account for match group–specific trends in outcomes, as well as individual fixed effects, μ_i.[37] The six grading periods before SUD treatment initiation is used as the

[37] Time fixed effects will be absorbed since I include a full set of match group–by–relative time fixed effects.

reference period.[38] Any observations outside the ±12 event time window are dropped. The key coefficients of interest are γ_k, which summarize the within-individual changes in outcomes relative to the reference period among adolescents who attend a SUD treatment center school, compared to the matched control individuals who do not. Standard errors are clustered at the individual level as in equation (1.1).

1.4.3 Long-Run Analysis

In my long-run analysis, I estimate the impact of residential SUD treatment on educational and labor market outcomes that are measured in early adulthood. For the long run analysis, I focus on adolescents in my sample who are aged 20 or older as of 2020, the last year of my sample period.[39][40] The latter requirement on age leads me to focus on the treatment individuals who attended a SUD treatment center school during the academic years 1999–2000 and 2016–2017. My final long-run analysis sample consists of 3,252 treatment individuals and 28,723 matched control individuals (10,841 unique individuals). Again, each control individual in the same match group is given equal weight and weights are summed up to one. Treatment individuals are assigned a weight of one.

Since an individual's long-run outcomes can only be observed after the SUD treatment, I cannot include individual fixed effects. Instead, my econometric model includes fixed effects for match group so that I can measure the difference in the outcomes between a treatment individual and the matched controls. My long-run analysis specification takes the following

[38] As described above, the treatment individuals in my sample received SUD treatment 0 to 3 grading periods after detention. In other words, they were placed in a detention center between event times -3 and 0. Event time -6, the reference period in my specification, is a pre-detention period for all individuals in my sample.

[39] To be specific, I include adolescents who are detained at some point between ages 12–16 during the academic years 1999–2000 and 2017–2018 (i.e., the same restriction used in the short-run analysis) and aged 20 or older as of 2020. The TEA provides annual data on an individual's age as of September 1st. Since I do not have information on the date of birth, I assume that each individual was born on September 1st.

[40] I only look at outcomes through age 20 because the sample size significantly decreases as I extend to later ages. For example, some treatment center schools opened around 2015–2016, and I am only able to follow adolescents until ages 16 to 20 for individuals who attended those centers. To include all treatment center schools in my analysis as well as increase the sample size, I focus on outcomes measured through age 20.

form:

$$Y_{igc} = \lambda Treatment_i + \pi_g + \omega_c + \kappa' X_i + \xi_{igc}, \tag{1.3}$$

where Y_{igc} is an outcome for individual i in match group g who was detained in a juvenile detention center in county c. $Treatment_i$ is an indicator for individuals who attended a SUD treatment school. I include match-group fixed effects, π_g, to account for differences between match groups. I also include a vector of individual-level controls, X_i, including the length of detention and an indicator for ever being employed in the pre-detention period. X_i also includes three pre-detention measures (average absence rate, juvenile detention history one year prior to detention, juvenile dentention history two years prior to detention) that were used for the fuzzy matching procedure. These measures account for potential differences between the treatment and control individuals in the pre-detention period. To understand the difference between my short- and long-run analysis models, I estimate the impact of SUD treatment center schools on my short-run educational outcomes using both models and compare the estimates in Section 1.5.

1.5 Results

1.5.1 Short-Run Effects on Educational Outcomes

Raw Trends in Outcomes Figures 1.2 and 1.4 present raw data trends in the fraction of adolescents enrolled in a SUD treatment center school and the mean absence rate, respectively, from 12 six-week grading periods before (i.e., about two academic years) to 13 six-week grading periods after *placement into detention*. Each figure plots trends separately for four sub-groups that are defined based on the length of the intermediate pre-period. The top left panel includes match groups in which the treatment adolescents enter the SUD treatment center school immediately after detention (i.e., within the same grading period when they are placed into detention); the top right panel includes match groups in which the treatment adolescents enter the SUD treatment center school one grading period after placement into detention; and the bottom left (right) panel includes match groups in which the treatment adolescents enter the school two (three) periods after placement into detention. In each panel, trends are presented separately for treatment and

matched control individuals.

The four sub-groups included in Figures 1.2 and 1.4 are pooled together in Panels (a) in Figure 1.3 and Figures 1.5–1.8. In these figures, I present raw trends in my short-run outcomes from 12 six-week grading periods before (i.e., about two academic years) to 13 six-week grading periods after *the time of SUD treatment initiation*, separately for treatment and matched control individuals. All individuals in the sample are placed into a juvenile detention center at some point between event time -3 and 0, the shaded area, and the average length of detention is 17.3 days. Therefore, I define event time periods between -12 and -4 as the pre-detention period. Event time periods 0 to $+12$ are defined as the post-treatment period. Raw data trends indicate that for all the short-run analysis outcomes, the treatment individuals are trending similarly to the matched control individuals during the last 12 grading periods prior to SUD treatment initiation, providing evidence in support of the parallel trends assumption. It is also important to note that not only the trends but also the levels are also very similar across the two groups in the entire pre-detention period.

Panel (a) in Figure 1.3 shows raw data trends in the fraction of adolescents enrolled in a SUD treatment center school around the time of SUD treatment initiation, combining the four sub-groups included in Figure 1.2. In event time zero, all treatment individuals are enrolled in a SUD treatment center school, and then the fraction of treatment individuals staying in the center goes down over time as they leave the center. The average length of stay is about 49 school days (or about one and a half six-week grading periods). During the periods in or after event time $+4$, the share of treatment individuals enrolled in a SUD treatment center school is 10.8%, which means that less than 10.8% spend more than four grading periods (about two-thirds of an academic year) in a center.

Raw trends in the average absence rate and the fraction of adolescents who are chronically absent from school (i.e., an absence rate being equal to or greater than ten percent) are presented in Panels (a) and (c) in Figure 1.5, respectively. Absences are increasing in event time prior to detention for both groups. This may reflect the fact that absenteeism increases with age as well as the fact that an adolescent's risk for delinquency is

likely to increase over time prior to detention. However, starting with the period of SUD treatment initiation, I see a divergence in these trends, with individuals who enter a SUD treatment center having a large drop in absences and chronic absenteeism. I see the largest drop in absenteeism in the first two grading periods following event time zero.

While absence rate and chronic absenteeism are measured by conditioning on being observed within the Texas public schools system, outcome variables used in Figure 1.6 are measured for all individuals and for all event time periods. Panel (a) shows raw trends in the fraction of adolescents who are not observed in the public school system, and Panel (c) presents raw trends in the fraction of adolescents who are either chronically absent from school or not observed in the public school system. In Panel (a) in Figure 1.6, I see that the share of adolescents who are not observed in the public school system begins to increase around the time of detention among both the treatment and control groups.[41] Then, in event time zero, this outcome becomes zero among the treatment group as they enter a SUD center school. Similarly, Panel (c) shows that there is a sudden, large drop in the likelihood of chronic absenteeism or not being in the public school system in event time zero, and the magnitude of the effect becomes smaller over time.

For the analyses using disciplinary action outcomes throughout the paper, I only include match groups where the treatment individuals were ever disciplined for a substance-related problem in the pre-detention period—the same restriction made for the control individuals—to make both groups more comparable, though I also present the results using the full sample in Appendix Figure A.5. Panel (a) in Figure 1.7 indicates that the treatment individuals are less likely to be disciplined in school following SUD treatment. In fact, raw trends in the fraction of adolescents disciplined in school for any reasons are very similar when I use the full sample—individuals with and without substance-related disciplinary action history (see Panel (a) in Appendix Figure A.5). This implies that treatment individuals who were never disciplined specifically for substance-related problems were often disciplined for other reasons, making the overall likelihood of any disciplinary

[41] As noted above, I restrict my sample to individuals who are observed for at least three grading periods during the six grading periods prior to detention. I do not make any further restrictions on enrollment in the public school system.

action very similar across these individuals and the matched control individuals.

Next, in Panel (c) in Figure 1.7, I show that the fraction of adolescents disciplined for substance-related reasons is also trending similarly across the treatment and control groups in the pre-detention treatment period. One concern with using substance-related disciplinary action as a proxy for SUD among control individuals is that the severity of SUD may trend differentially over time across the treatment and control groups. Panel (c) in Figure 1.7 indicates that the likelihood of being disciplined for substance-related problems was trending very similarly in the entire pre-treatment period, providing supportive evidence that the risk of substance use and delinquency evolves similarly over time across the two groups prior to the time of SUD treatment initiation.

Finally, Figure 1.8 plots the trends in the average course fail rate from three years before to two years after the SUD treatment initiation. Note that I use yearly-level data for the course fail rate outcome because the data on course completion is only available at the academic year level. Panel (a) in I see that the average course fail rate for the treatment group decreases beginning in the year of treatment relative to the matched control group, and this effect persists over the first two academic years.

Event Study Results Panels (b) of Figures 1.3–1.8 plot the regression analogue to raw trends presented in Panels (a). I plot the coefficients and 95% confidence intervals on the interactions between the indicator for a treatment individual and the indicators for the periods around the time of SUD treatment initiation from equation (1.2). Event study estimates indicate that the interpretations from raw data trends are robust to the regression adjustment (i.e., inclusion of individual and match group–by–time fixed effects). For all the outcomes examined in the short-run analysis, there are no statistically significant differences between the treatment and matched control individuals in the pre-treatment period, providing evidence in support of the parallel trends assumption.

Panels (b) and (d) in Figure 1.5 indicate that treatment individuals experience

declines in absenteeism following SUD treatment.[42] Although a drop in absence rate would be partially mechanical, given that individuals in a treatment center school attend a school within a residential facility, understanding the impact of SUD treatment attendance on absenteeism is particularly important for several reasons. First, treatment individuals experience a large drop in absenteeism in the first two years after SUD treatment. This could be a key channel through which SUD treatment center schools can have a longer-term impact on the treatment individuals. For instance, decreased absence rate can affect the likelihood of graduating high school or the maximum grade level completed in secondary school, which I will investigate in Section 1.5.2. Second, it is important to understand whether SUD treatment center school attendance has a persistent impact on absenteeism in the post-discharge period. Although it is difficult to isolate the impact of treatment center school attendance in the post-discharge period because how long an individual stay in a treatment center school is endogenously determined (i.e., it reflects treatment individuals' behaviors and choices), a simple analysis suggests that treatment individuals experience a persistent drop in absenteeism even after they leave SUD treatment and attend other public schools (see Appendix Figure A.7).[43]

Panel (b) in Figure 1.8 shows that the effect of SUD treatment center school attendance on the continuous course fail rate is visually more pronounced with the event study regression. Following SUD treatment, treatment individuals are less likely to fail a

[42]To understand how the numerator and denominator of the absence rate change, I present in raw trends in the number of days absent and the number of days enrolled in Panels (a) and (b) in Appendix Figure A.6, respectively. The number of days absent (enrolled) are measured while an individual is enrolled in either the public school system or a school within a juvenile detention center. Changes in absence rate for the treatment and control groups mostly reflect the changes in the number of days absent. Note that the total number of days enrolled in a given six-week period decreases over time for both groups, partially driving an increasing trend in absence rate in the post-treatment period.

[43]Note that some individuals are absent from school while they are enrolled in a SUD treatment center school, though the absent rate within SUD treatment center schools is relatively low on average. Appendix Figure A.8 shows the distribution of absence rate within a treatment center school measured as the total days absent from a SUD treatment center school relative to the days enrolled in the same center school. For almost 70 percent of the treatment individuals in my sample, the absence rate within a SUD treatment center school is zero, but the other 30 percent are absent from a SUD center school for at least one school day. Therefore, the drop in absenteeism while individuals are in a SUD treatment center school is not purely mechanical.

course relative to the matched control individuals, and this effect is persistent in the first two post-treatment years.[44] As mentioned above, a large fraction of individuals leave the public school system in the first two years. As a result, only 69.5% of the treatment individuals have non-missing course completion records in the following academic year of SUD treatment initiation (i.e., event time +1). To address concerns about compositional changes, I show that my results for the course fail rate are robust if I use a balanced sample instead, confirming that the course fail rate estimates are not driven by student compositional changes (see Section 1.5.3).

Figure 1.9 presents the coefficients and 95% confidence intervals from equation (1.1), in which I pool the post-treatment periods to capture the average effects of attending a SUD treatment center on each of my short-run outcomes. Table 1.2 reports the corresponding regression estimates. As shown in columns (1) and (2), attending a SUD treatment center school leads to an average decrease in the absence rate of 5.1 percentage points (or 27.5% relative to the control group post-treatment period mean, $p-$value<0.001) and a 11.9 percentage point decrease in chronic absenteeism (23.5%, $p-$value<0.001) in the first 13 grading periods following SUD treatment. Moreover, as reported in column (3), the likelihood of not being observed within the Texas public school system decreases by 4.9 percentage points (10.9%, $p-$value<0.001). In column (4), I combine the measure of chronic absenteeism and the measure of not being observed in the public school system and find that SUD treatment center school attendance reduces the likelihood of chronic absenteeism or not being observed in the public school system by 10.4 percentage points in the first 13 periods (13.7%, $p-$value<0.001). The estimates in column (5) indicate that SUD treatment school attendance reduces the likelihood of being discipline in school by 7.5 percentage points in the first 13 periods following SUD treatment (28.1%, $p-$value<0.001). Finally, as reported in the last column, the course fail rate decreases by 5.5 percentage points in the first two academic years following SUD treatment (16.1 percent, $p-$value< 0.001).

Heterogeneity Analyses I investigate heterogeneity in the estimated effects of

[44] Appendix Figure A.9 shows event study results using the number of courses taken (Panel (a)) and the number of courses failed (Panel (b)) as the outcomes. The results pattern for the number of courses failed is similar to that for the course fail rate presented in Figure 1.8.

SUD treatment center schools on the short-run outcomes across a number of individual and treatment characteristics. Specifically, I define the following sub-groups based on individual characteristics: (1) Non-Hispanic White, (2) Non-White (Hispanic or Non-Hispanic Black), (3) female, (4) male, (5) economically disadvantaged (measured using free/reduced-price lunch receipt in the two years prior to detention), (6) not economically disadvantaged, (7) in a special education program (measured in the two years prior to detention), and (8) not in a special education program. In addition, I investigate heterogeneity by treatment characteristics (including age at the time of SUD treatment) using the following sub-groups: (1) treatment at ages 13–14, (2) treatment at age 15, (3) treatment at age 16, (4) by court order, and (5) not by court order (i.e., by referral).[45]

To fully understand the heterogeneity in the treatment effect, it is important to explore heterogeneity in the length of stay—a measure of treatment intensity—across the sub-groups. In the box plots in Figures 1.10–1.11, the whiskers show the lower and upper extreme values (excluding outliers); and vertical lines show the 25th percentile, median, 75th percentile of the length of stay, expressed as a percentage of one academic year.[46] The box plots in Figure 1.10 indicate that females have the longest length of stay among the sub-groups, while adolescents who are in a special education program prior to detention spend the shortest period of time in a SUD treatment center school on average. The distribution of the length of stay is similar across the other sub-groups. Figure 1.11 shows

[45]For each enrollment record, the TEA data provide data on "attribution code", which indicate several circumstances including whether the student attends an open enrollment charter school; the student is in a residential treatment facility and was court-ordered into the facility; and the student is in a residential treatment facility and the student was not court-ordered into the facility. I identify students who are court-ordered into a SUD treatment center school using the codes indicating that a student is court ordered into a residential treatment facility. I consider a student is not court-ordered into the facility if the student has any other attribution codes. Since data on court order status are only available from 2009–2010 onward, my heterogeneity analyses by court order status only include adolescents who enter a SUD treatment center in or after the academic year 2009–2010 and have a non-missing attribution code. Roughly 40 percent of the final sample in the heterogeneity analysis by court order status are court-ordered into a SUD center school.

[46]Specifically, I calculate the length of stay by the following steps. First, I assume that every academic year has 180 days. Second, I winsorized the length of stay at 180 days and divided it by 180 days to express the length of stay as a percentage of one academic year.

that the length of stay is similar across different ages at the time of SUD treatment initiation. Adolescents who are court-ordered into the programs are enrolled in the program for a substantially longer period of time than those who are not.

In Figures 1.12–1.17, I show the results for my heterogeneity analysis for each sub-group and for each of my short-run outcomes. Panel (a) shows the heterogeneity by demographic characteristics, and Panel (b) presents the heterogeneity by treatment characteristics. I present the coefficients and associated 95% confidence intervals from equation (1.1). In these analyses, I interact the indicators for sub-groups with the $Treatment_i \times Post_{gt}$ term, and report the estimates on these interaction terms.[47] My heterogeneity analysis for the short-term outcomes suggests that the impacts of SUD treatment center schools are nearly universal but with the following differences. First, for all the outcomes, I find the largest effects on females among my demographic sub-groups. One potential explanation is that females spend the longest time in a SUD treatment center school among the demographic sub-groups, as shown in Figure 1.10. Second, although SUD treatment center school attendance reduces the likelihood of not being observed in the public school in the post-treatment period among almost all sub-groups, I do not see any effect among those who were in a special education program in the pre-detention period.

1.5.2 Long-Run Effects on Educational and Labor Market Outcomes

Figure 1.18 shows the estimates of the effects of attending a SUD treatment center school on adolescents' educational and labor market outcomes by age 20. The figure presents the coefficients and 95% confidence intervals from equation (1.3) for each of my long-run outcomes. Tables 1.3 and 1.4 present the corresponding regression results, where I report coefficients, standard errors that are clustered at the individual level in parentheses, and $p-$values in brackets.

The estimates in Tables 1.3 and 1.4 indicate that attending a SUD treatment center school leads to a 4.4 percentage point increase in the likelihood of completing grade 10

[47] For example, when investigating the heterogeneity by gender, I use indicators for females and males and interact them with the $Treatment_i \times Post_{gt}$ term.

(15.4% relative to the control group outcome mean, $p-$value<0.001) and a 1.7 percentage point increase in the likelihood of grade 11 completion by age 20 (10.2%, $p-$value=0.006).[48][49] I find no statistically significant effect of attending a SUD treatment center school on high school graduation ($p-$value=0.561).[50] To summarize the effects of treatment center schools on completed secondary education, I investigate the effect on the maximum grade level completed. As reported in column 4, the estimates indicate that attending a SUD treatment center school leads to 0.11 additional years of schooling.

I find that attending a SUD center school leads adolescents to be 1.3 percentage point more likely to enroll in any college by age 20 (11.5%, $p-$value=0.018). This increase is almost entirely driven by an increase in two-year college attendance (see columns (2) and (3)). Only a tiny number of individuals in my sample attended a four-year college by age 20 (control group mean=0.6%), implying that SUD treatment center school attendance leads to an increased two-year college enrollment among youth who would not have attended college. I also find that treatment individuals experience an increased likelihood of being employed at ages 17–20 by 2 percentage points (2.7%, $p-$value=0.007). I combine the measures of college enrollment and employment and find that SUD treatment center school attendance leads to a 2.1 percentage point increase in the likelihood of being enrolled in any college by age 20 or employed between 17–20 or both (2.7%, $p-$value=0.005).[51]

Heterogeneity analyses by demographic characteristics I examine heterogeneity in the impacts of SUD treatment center school attendance on the long-run outcome by student demographic and treatment characteristics. In Figures 1.19–1.21, I present the coefficients and 95% confidence intervals from equation (1.3) for each

[48] I define grade 10 completion as ever being enrolled in Texas public school system in grade 11. I define grade 11 completion similarly.

[49] Although not reported, I do not find evidence that attending a SUD treatment center is systematically associated with completing grade 10 (or 11) prior to detention.

[50] 13.2% of my control group graduate high school by age 20.

[51] As presented in Appendix Table A.3, I find no evidence that SUD treatment center school attendance increases earnings at ages 17–20. The estimated increases in secondary school schooling and college enrollment between 17–20 following SUD treatment could be one possible explanation. As stated above, an important limitation is that earnings are only measured through age 20. Any increases in earnings because of the estimated increases in educational attainment may not appear until later adulthood.

sub-group.[52] Heterogeneity in long-run impacts by gender, race/ethnicity, receipt of free or reduced-price lunch, and participation in a special education program are presented in Panels (a), (b), (c), and (d) in Figure 1.19, respectively. Heterogeneity in long-run impacts by age at the time of SUD treatment initiation is presented in Figure 1.20. Panels (a) and (b) in Figure 1.21 show heterogeneity by court order status.

The estimates reported in Figure 1.19 indicate that the impact of SUD treatment center schools on college enrollment and employment is much larger among females (14.5% of the long-run analysis sample) and adolescents who were not eligible for free/reduced price lunch in the pre-detention period (24% of the long-run analysis sample). I also find that the size of the effect on college enrollment is larger for Whites, and the effect on employment is mostly driven by non-Whites (22.8% of the long-run analysis sample). Among adolescents who were in a special education program prior to detention, the effect of SUD treatment schools is indistinguishable from zero for almost all outcomes and is significant and negative for high school graduation. The estimates presented in Figure 1.20 indicate that the effect of SUD treatment schools on grade 10 and grade 11 completion is largest among those who were aged 16 (oldest among my sample) at the time of SUD treatment initiation, reflecting the fact that their grades were closer to grade 10 or 11. For the college and employment outcomes, I see a larger effect of SUD treatment center schools on college enrollment among adolescents who were 13–15 years old at the time of SUD treatment, while I see a larger effect on employment among those who were 16 years old at the time of SUD treatment.

Finally, Panel (b) in Figure 1.21 reveals important heterogeneity in the impact of SUD treatment center school attendance on my long-run outcomes by court order status. I find positive impacts of SUD treatment center schools on both treatment individuals who are court-ordered into the program and those who are not (i.e., who enter the program by referral). In particular, I see a larger impact on grade 10 completion, grade 11 completion, and high school graduation among those who enter a center by court order, while I observe a larger effect on college enrollment and employment among those who enter the program by

[52] In these analyses, I interact indicators for sub-groups with an indicator for the treatment individuals and report the estimates for the interaction terms.

referral. Although this analysis is conducted using 37.7% of my long-run analysis sample,[53] the results provide important evidence that SUD treatment center schools improve student outcomes regardless of whether they are court-ordered into the program or not.

1.5.3 Robustness Analysis

As described in Section 1.4, I use different empirical models in the short- and long-run analyses. To investigate the difference between the two models, I estimate the long-run analysis model (equation (1.3)) using my short-run analysis outcomes and compare those estimates with the baseline estimates. For this exercise, I first construct a version of the short-run analysis outcomes by taking the average of the outcome values between relative periods 0 and +12 (i.e., the first two years after residential SUD treatment). Using this sample, I then run both short- and long-run analysis models. Figure 1.22 presents coefficients and 95% confidence intervals from these estimations separately for each econometric model. Importantly, the results are robust across the two models, suggesting that the difference between short- and long-run analysis models does not drive my results.

Alternative explanation: difference in underlying ability In Section 1.5.2, I show that SUD treatment center school attendance leads to an increase in college enrollment and employment by age 20. However, there could be a concern that my results are driven by the difference in underlying ability or academic performance in the baseline period. To address this concern, I examine the impact of SUD treatment center school on academic performance measured in the pre-treatment period, which is similar to a falsification test. In Figure 1.23, I report the coefficients and 95% confidence interval from equation (1.3) with the following outcomes as the dependent variable: (1) the average past course pass rate, (2) the average past Z-score for standardized reading tests, and (3) the average past Z-score for standardized math tests, all measured in the two academic years prior to SUD treatment initiation. The coefficients are close to zero and statistically insignificant, providing evidence that my results are not driven by the difference in academic performance in the pre-treatment period.

[53] As noted before, the data on whether an individual is court-ordered into a residential facility is only available from the academic year 2009–2010 onward.

Alternative explanation: the effect of detention or mean reversion In Figure 1.5, I show that the treatment group experienced a sudden and large drop in absenteeism, disciplinary action, and course pass rate beginning in the period of SUD treatment initiation. However, there could be a concern that reduction in absenteeism or improvements in academic performance following SUD treatment initiation may be driven by the differential impacts of detention across the treatment and control groups and/or differential mean reversion effects—a return to the individual's typical performance—across the two groups rather than positive causal effects of treatment center school attendance. To address this concern, I investigate whether SUD treatment center attendance leads to a reduction in absenteeism among those who enter a SUD treatment center school not within three grading periods but after five or six grading periods (about a year).[54] If a reduction in absence rate is solely driven by the differential detention effects or differential mean reversion effects across the two groups, I would not expect to see any substantial changes in absenteeism at the time of SUD treatment initiation for those who enter a treatment center school after five or six grading periods. To perform this test, I assign individuals into seven groups based on the length of the intermediate pre-period (i.e., the period between detention and SUD treatment initiation), ranging from zero to six. In Appendix Figure A.10, I plot raw trends in chronic absenteeism separately for these groups (for brevity, I omit the group who enter a SUD treatment center school four periods after detention). In the top left panel, I show the trends in chronic absenteeism for those who enter the treatment school in the same period of detention. Then in the remaining panels, I show the trends for sub-groups with 1, 2, 3, 5, and 6 period-long intermediate pre-period, respectively. The solid gray vertical line denotes the time of detention, and the red dashed vertical line denotes the time of SUD treatment initiation.

In all panels, I observe a certain level of drop in absence during the intermediate pre-period, which may reflect factors such as the deterrence effect during detention and the mean reversion pattern. However, in all panels, the largest drop in absenteeism coincides with the exact time of SUD treatment initiation, suggesting that the reduction in absenteeism measured in event time zero is not driven by the alternative explanations mentioned above.

[54]Note that in my baseline analysis, I restrict attention to adolescents who enter a SUD treatment center school within three grading periods.

Robustness analysis: unbalanced vs. balanced panel My short-run analysis uses an unbalanced panel of adolescents who are observed in the TEA data in each of the 25 grading periods surrounding SUD treatment initiation (12 periods before to 13 periods after). In Appendix Figure A.11, I explore the sensitivity of my estimates using a balanced panel instead. However, as shown in Figure 1.6, a large fraction of adolescents leave the Texas public school system in the first two years following SUD treatment initiation, implying that only a small fraction of the sample will be consistently observed for all 25 periods. To relax this balanced sample restriction, I use semester-year-level data for absenteeism and disciplinary action outcomes instead of six-week grading period-level data for this analysis.[55] In particular, I overlay my event study estimates that are obtained using an unbalanced sample (i.e., baseline sample) with results obtained from a sample in which I only include individuals that are consistently observed from four semesters before to three semesters after SUD treatment initiation. The results across the two samples are very similar, indicating that my baseline estimates are not driven by compositional changes in the sample.[56]

Robustness analysis: limiting the sample to youth with prior substance-related discipline history As discussed in Section 1.4.2, to be eligible for the control group, individuals should have been disciplined for substance-related problems in the 12 grading periods prior to detention, but I do not make the restriction for the treatment group. Appendix Figures A.12–A.14 shows that both short- and long-run analyses results are similar (but the confidence intervals are slightly wider) if I only include match groups where both the treatment and matched control individuals were ever disciplined for substance-related reasons prior to detention. The results suggest that this sample restriction does not drive my findings.

Alternative matching: exact matching only I also test the robustness of my estimates using two alternative ways of matching. First, I exclude the absence rate from the set of matching variables. Second, I only do the exact matching omitting the fuzzy matching.

[55] Empirical specification is the same as equation (1.2). One semester prior to SUD treatment initiation is the reference period.

[56] Although not reported, the event study results for course fail rate are also robust to only including individuals who have non-missing course fail rate record from two years before to one year after SUD treatment initiation.

Using these two alternative ways of matching, I show the following sets of results: (a) raw data plots and event study results from equation (1.2) for each of my short-run analysis outcomes, (b) the difference-in-difference estimates for my short-run analysis outcomes from equation (1.1), and (c) the estimates for my long-run analysis outcomes from equation (1.3). In Appendix Figures A.15–A.20, I show the sets of results derived from the alternative matching approach in which I drop the absence rate from the fuzzy matching. In Appendix Figures A.21–A.26, I present the sets of results I obtain using the exact matching only and omitting the fuzzy matching. For all the regression results, I overlay the baseline estimates and the estimates derived from an alternative matching approach. The estimates indicate that my results are qualitatively similar when I exclude absence from the set of matching variables or when I do the exact match only.

1.6 Discussion and Comparison with Previous Studies

Comparison to Other Interventions in Disadvantaged Populations To contextualize my estimates of the effects of attending a SUD treatment center school on college enrollment and employment, I discuss how my findings compare to results from papers that examine the impacts of interventions for at-risk youth or youth from disadvantaged backgrounds. Although I focus on justice-involved youth and other interventions target youth from disadvantaged backgrounds more generally, these comparisons could be helpful given substantial overlap between these two populations. Using administrative data from Illinois, Chyn (2018) finds that moving children who lived in severely distressed public housing to lower-poverty neighborhoods between ages 7 and 18 leads to a 9 percent (or 4 percentage point) increase in employment at ages 19–26. I find a 2.7 percent (or 2 percentage point) increase in employment at ages 17–20, which is nearly a third the size of the estimated impact of moving to less-disadvantaged neighborhoods. My estimate of the impact of SUD treatment schools on employment is relatively large given that youths spend on average 49 days in a SUD treatment center school and thus the duration of the treatment is much shorter than that of moving to lower-poverty neighborhoods. This relatively large magnitude could reflect the fact that untreated SUDs can have adverse impacts on all aspects of a young person's life, and thus even access to

SUD treatment for a relatively short-term period can have large positive impacts that can persist into adulthood.

My estimates can also be compared to Gelber et al. (2016)'s study on the impact of the New York City (NYC) Summer Youth Employment Program (SYEP) on college enrollment and employment.[57] They find that providing youths aged 14–21 with paid summer employment for up to seven weeks increases employment by 1 percentage point in 1–4 years after the program but has no impact on college enrollment. My estimates of a 2 percentage point increase in employment at ages 17–20 and 1.3 percentage point increase in college enrollment are larger in magnitude. While the intervention in their setting is of a similar duration as the average treatment duration in my setting, the intervention I analyze is more targeted both in the treatments provided (health and education services) and the population served (those who are suffering from severe SUDs). These differences in targeting may contribute to the difference in estimated effects.

Benchmarking Benefits Against Costs My estimates suggest that attending a SUD treatment center school has positive impacts on the maximum grade completed in secondary school, college enrollment, and employment at ages 17–20. To understand the cost effectiveness of providing access to residential SUD treatment, I compare the benefits of attending a SUD treatment center school with the associated costs. First, I take the estimated cost per adolescent residential treatment episode of $13,643.1 (in 2020 dollars) from French et al. (2008). Second, I conduct a back-of-the-envelope calculation based on my estimates of the impact of attending a treatment center school on years of schooling in secondary school. Assuming that an additional year of schooling leads to a ten percent increase in earnings (Card, 1999) and that this effect on earnings is constant through age 64, I find that my estimated 0.11 increase in years of education in secondary school leads to a $5,008.12 (in 2020 dollars) increase in the present discounted value of lifetime earnings for each individual, indicating that the benefits from increased schooling in secondary school alone can cover 36.7% of the total costs of providing access to residential SUD treatment.

[57] Youths who participated in this program on average came from disadvantaged family backgrounds and were disproportionately minorities.

1.7 Conclusion

Substance use and SUDs are significant public health challenges in the United States. SUDs can have particularly profound effects on adolescents, given that adolescence is a critical period for developing healthy behaviors and accumulating human capital. Despite the urgent need for greater implementation of effective SUD treatments, there is a lack of causal evidence of the effect of SUD treatment programs on individuals, especially on adolescents. Quantifying the causal effects of SUD treatment programs on affected individuals and understanding the heterogeneity in the effect of such programs across individuals are critical for policy design, as the policymakers must decide how to allocate limited resources across different types of programs and across individuals.

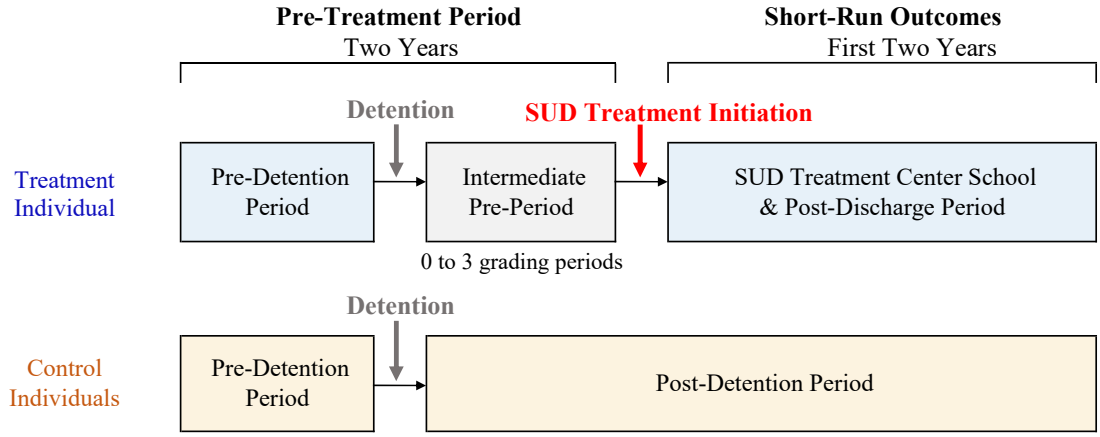
Using individual-level administrative panel data from Texas, this paper provides evidence on the causal impact of attending a SUD treatment center school on later educational and employment outcomes through age 20 among at-risk youth—youths who were previously detained in a juvenile detention center at some point between ages 12 and 16. I show that attending a SUD treatment center school reduces absenteeism, disciplinary action, and course failure relative to matched control individuals. This paper also establishes for the first time that these schools have long-lasting positive impacts on completed secondary education, college enrollment, and employment at ages 17–20.

This paper demonstrates that providing access to a SUD treatment center school—an increasingly popular type of SUD treatment programs for adolescents—has substantial benefits for justice-involved youth, who represent about half of all SUD treatment admissions among youth aged 12–17 years. My back-of-the-envelope calculations suggest that projected increases in lifetime earnings based on the increases in educational attainment in secondary school alone can offset roughly a third of the costs of this treatment. These estimated benefits may understate the total benefits, if attending a treatment center school also leads to unmeasured improvements in health (e.g., reductions in mortality or health care spending) and reductions in crime (e.g, reductions in costs incurred by the justice system or victims). Interventions during adolescence have particularly

important implications for substance use policies because many individuals begin their use of addictive substances during this period. Policymakers might consider improving access to SUD treatment among adolescents as one important way to address SUD problems as well as increase human capital accumulation among at-risk youth.

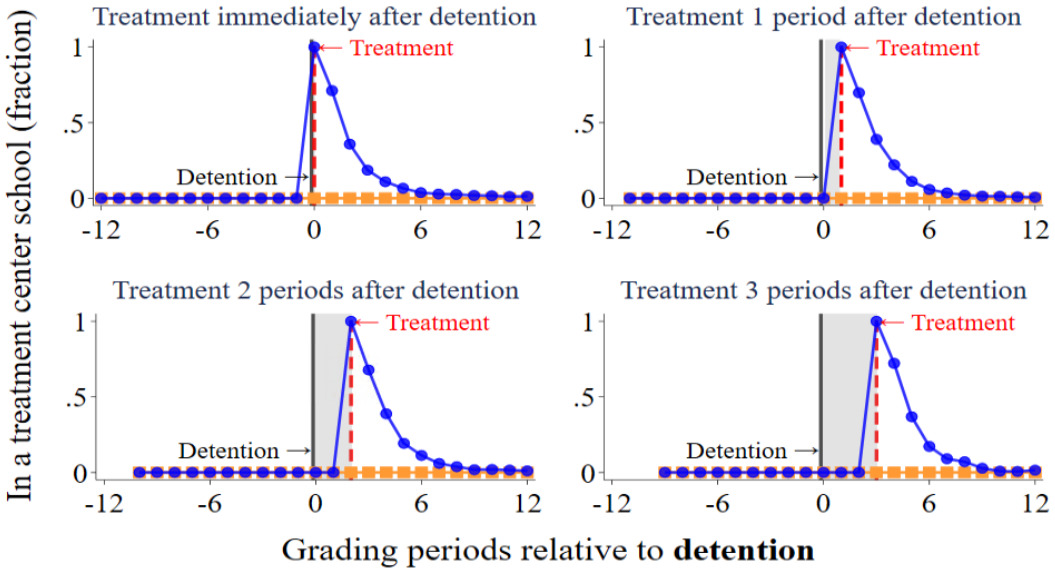
1.8 Figures and Tables

Figure 1.1: Description of the Short-Run Analysis Design



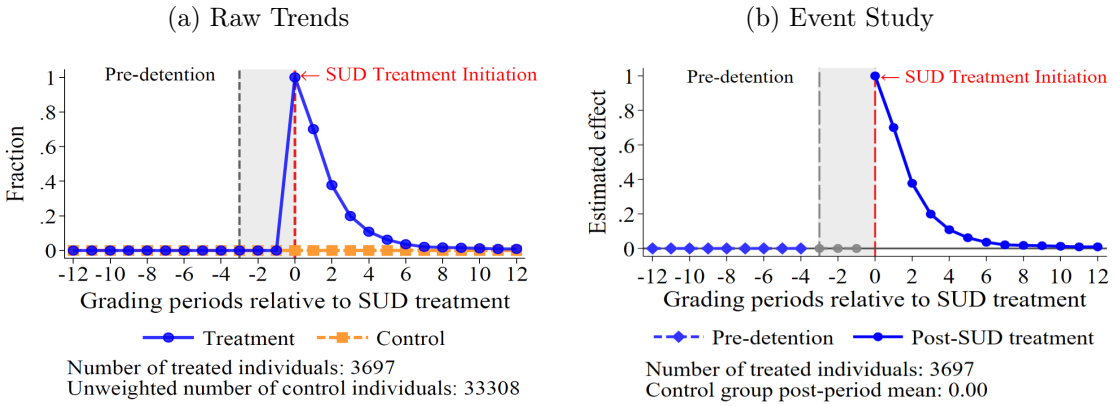
Notes: For each treatment individual, I identify control individuals who have the same basic demographic characteristics and suffer from a substance use disorder (SUD), but were not enrolled in a SUD treatment center school after detention. For each match group, the study post-period is define as the periods during or after which the treatment individual enters a SUD treatment center school. The study pre-period is defined as the periods prior to the time of the treatment individual's SUD treatment initiation. The study pre-period can be divided into the pre-detention period and the intermediate period. The intermediate pre-period is defined as the period between the timing of placement into a detention center and the timing of enrollment in a SUD treatment center school.

Figure 1.2: Raw Trends in Treatment Center School Enrollment Across Treatment and Control Individuals



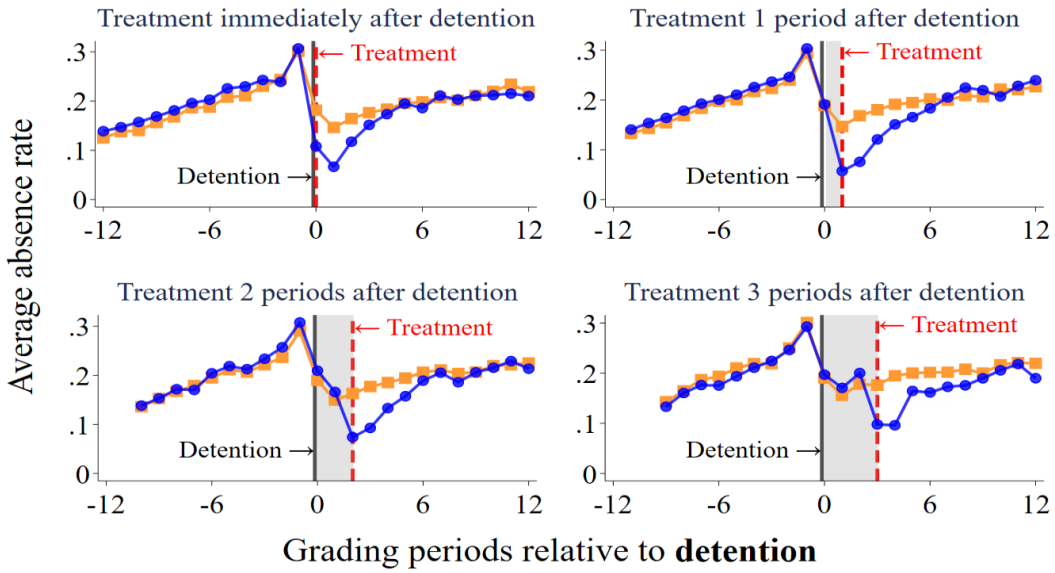
Notes: The figure presents raw data trends in the fraction of adolescents who are enrolled in a SUD treatment center school from 12 six-week grading periods before (i.e., about two academic years) to 13 six-week grading periods after *placement into detention*. The figure plots raw trends separately for four sub-groups that are defined based on the length of the intermediate pre-period. The top left panel includes match groups in which the treatment adolescents enter the SUD treatment center school immediately after detention (i.e., within the same grading period when they are placed into detention); the top right panel includes match groups in which the treatment adolescents enter the SUD treatment center school one grading period after placement into detention; and the bottom left (right) panel includes match groups in which the treatment adolescents enter the school two (three) periods after placement into detention. In each panel, I plot raw trends in the outcome over time separately for treatment and matched control individuals.

Figure 1.3: Treatment Center School Enrollment: Raw Trends and Event Studies



Notes: The figure plots raw data trends and event study results. In Panel (a), I present raw trends in the fraction of adolescents enrolled in a substance use disorder (SUD) treatment center school from 12 six-week grading periods before (i.e., about two academic years) to 13 grading periods after the time of SUD treatment initiation, separately for treatment and matched control individuals. Panel (b) plots the regression analogue to raw trends presented in Panel (a). I plot the coefficients and 95% confidence intervals on the interactions between the indicator for a treatment individual and the indicators for the periods around the time of SUD treatment initiation from equation (1.2).

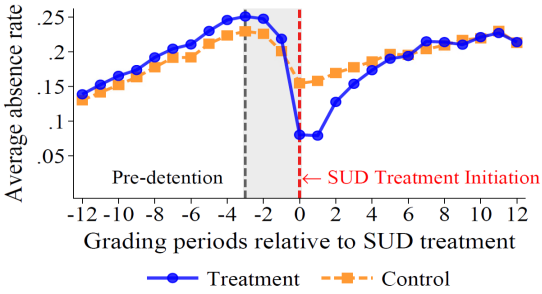
Figure 1.4: Raw Trends in Absence Rate Across Treatment and Control Individuals



Notes: The figure presents raw data trends in the mean absence rate from 12 six-week grading periods before (i.e., about two academic years) to 13 six-week grading periods after *placement into detention*. The figure plots raw trends separately for four sub-groups that are defined based on the length of the intermediate pre-period. The top left panel includes match groups in which the treatment adolescents enter the SUD treatment center school immediately after detention (i.e., within the same grading period when they are placed into detention); the top right panel includes match groups in which the treatment adolescents enter the SUD treatment center school one six-week grading period after placement into detention; and the bottom left (right) panel includes match groups in which the treatment adolescents enter the school two (three) grading periods after placement into detention. In each panel, I plot raw trends in the outcome over time separately for treatment and matched control individuals.

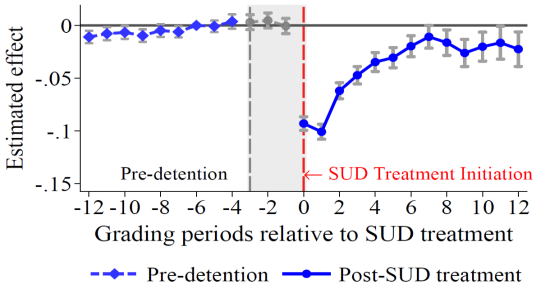
Figure 1.5: Absenteeism: Raw Trends and Event Studies

(a) Absence Rate: Raw Trends



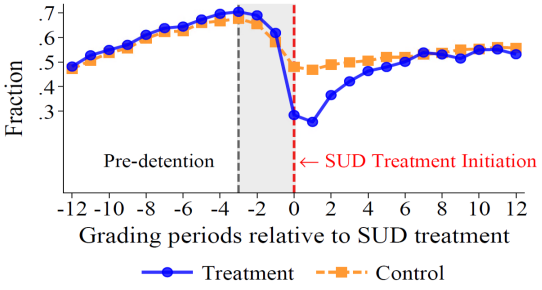
Number of treated individuals: 3697
Unweighted number of control individuals: 33308

(b) Absence Rate: Event Study



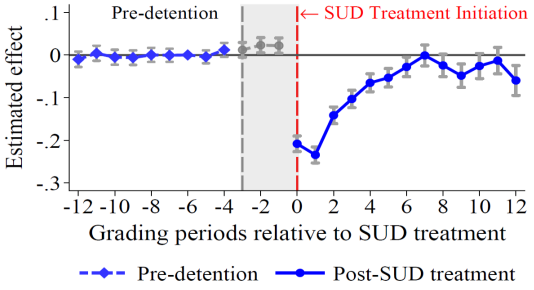
Number of treated individuals: 3697
Control group post-period mean: 0.19

(c) Chronic Absenteeism: Raw Trends



Number of treated individuals: 3697
Unweighted number of control individuals: 33308

(d) Chronic Absenteeism: Event Study

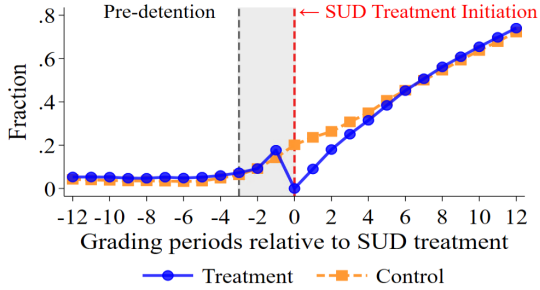


Number of treated individuals: 3697
Control group post-period mean: 0.51

Notes: The figure plots raw data trends and event study results. In Panels (a) and (c), I present raw trends in my short-run outcomes from 12 six-week grading periods before (i.e., about two academic years) to 13 grading periods after the time of SUD treatment initiation, separately for treatment and matched control individuals. Panels (b) and (d) plot the regression analogue to raw trends presented in Panels (a) and (c), respectively. I plot the coefficients and 95% confidence intervals on the interactions between the indicator for a treatment individual and the indicators for the periods around the time of SUD treatment initiation from equation (1.2). Standard errors are clustered at the individual level.

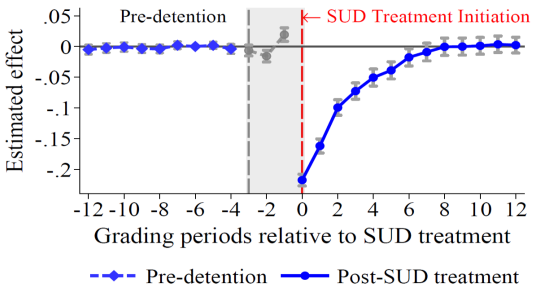
Figure 1.6: Not Being in Public School: Raw Trends and Event Studies

(a) Not in Public School System: Raw Trends



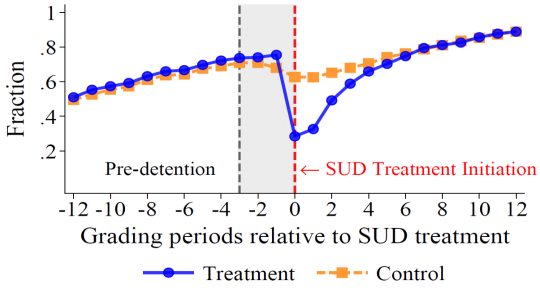
Number of treated individuals: 3697
Unweighted number of control individuals: 33308

(b) Not in Public School System: Event Study



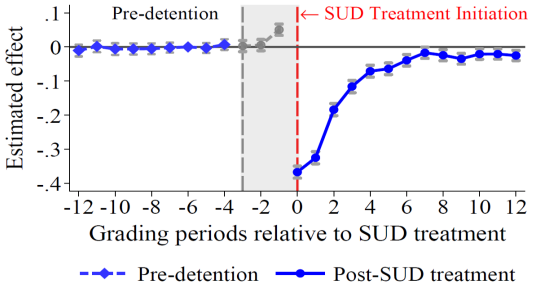
Number of treated individuals: 3697
Control group post-period mean: 0.45

(c) Not in Public School Or Chronic Absenteeism: Raw Trends



Number of treated individuals: 3697
Unweighted number of control individuals: 33308

(d) Not in Public School Or Chronic Absenteeism: Event Study

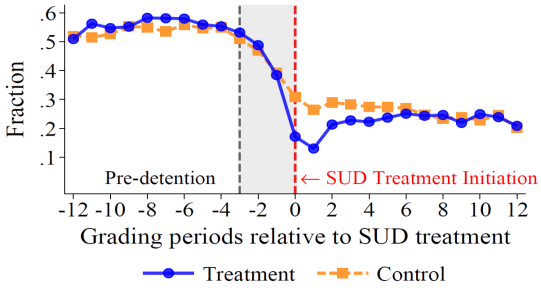


Number of treated individuals: 3697
Control group post-period mean: 0.76

Notes: The figure plots raw data trends and event study results. In Panels (a) and (c), I present raw trends in my short-run outcomes from 12 six-week grading periods before (i.e., about two academic years) to 13 grading periods after the time of SUD treatment initiation, separately for treatment and matched control individuals. Panels (b) and (d) plot the regression analogue to raw trends presented in Panels (a) and (c), respectively. I plot the coefficients and 95% confidence intervals on the interactions between the indicator for a treatment individual and the indicators for the periods around the time of SUD treatment initiation from equation (1.2). Standard errors are clustered at the individual level.

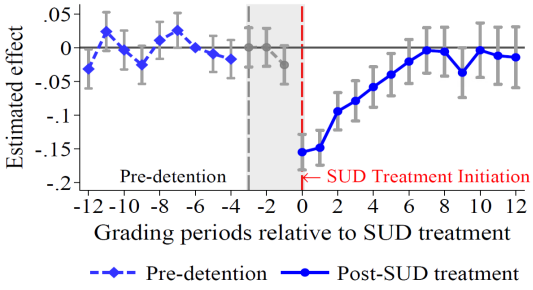
Figure 1.7: Disciplinary Action in School: Raw Trends and Event Studies

(a) Any Disciplinary Action: Raw Trends



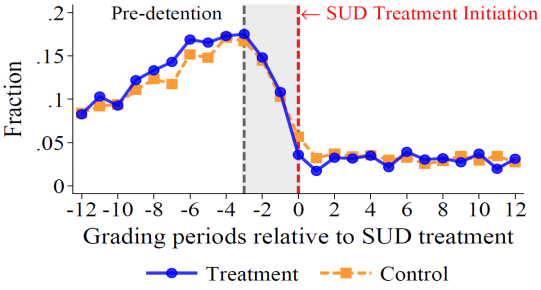
Number of treated individuals: 1391
Unweighted number of control individuals: 14361

(b) Any Disciplinary Action: Event Study



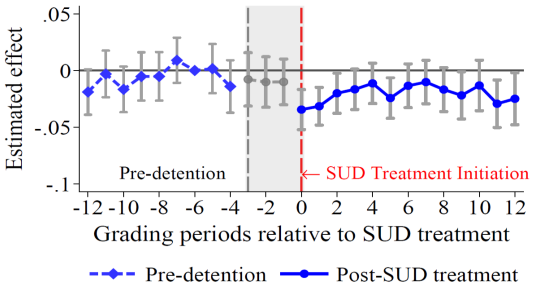
Number of treated individuals: 1391
Control group post-period mean: 0.27

(c) Disciplinary Action for Substance-related Problems: Raw Trends



Number of treated individuals: 1391
Unweighted number of control individuals: 14361

(d) Disciplinary Action for Substance-related Problems: Event Study

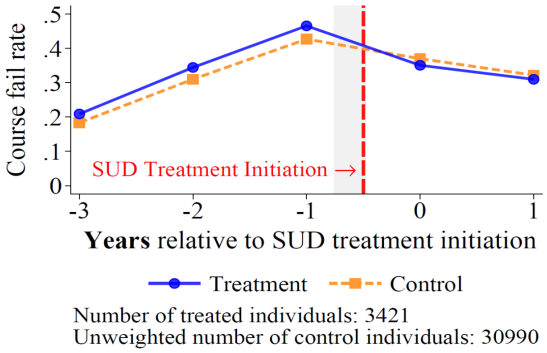


Number of treated individuals: 1391
Control group post-period mean: 0.04

Notes: The figure plots raw data trends and event study results. In Panels (a) and (c), I present raw trends in my short-run outcomes from 12 six-week grading periods before (i.e., about two academic years) to 13 grading periods after the time of SUD treatment initiation, separately for treatment and matched control individuals. Panels (b) and (d) plot the regression analogue to raw trends presented in Panels (a) and (c), respectively. I plot the coefficients and 95% confidence intervals on the interactions between the indicator for a treatment individual and the indicators for the periods around the time of SUD treatment initiation from equation (1.2). Standard errors are clustered at the individual level.

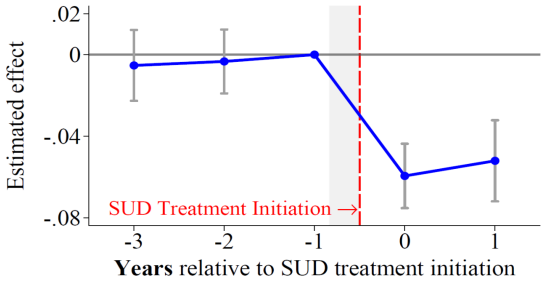
Figure 1.8: Course Fail Rate: Raw Trends and Event Studies

(a) Course Fail Rate: Raw Trends



Number of treated individuals: 3421
Unweighted number of control individuals: 30990

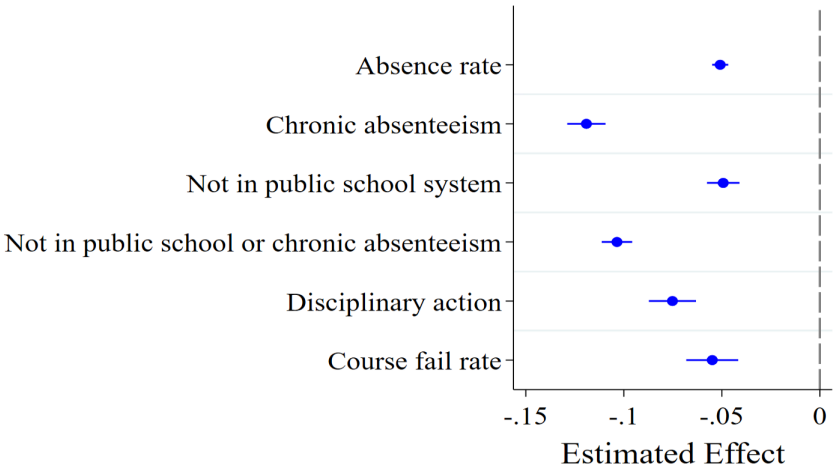
(b) Course Fail Rate: Event Study



Number of treated individuals: 2568
Control group post-period mean: 0.34

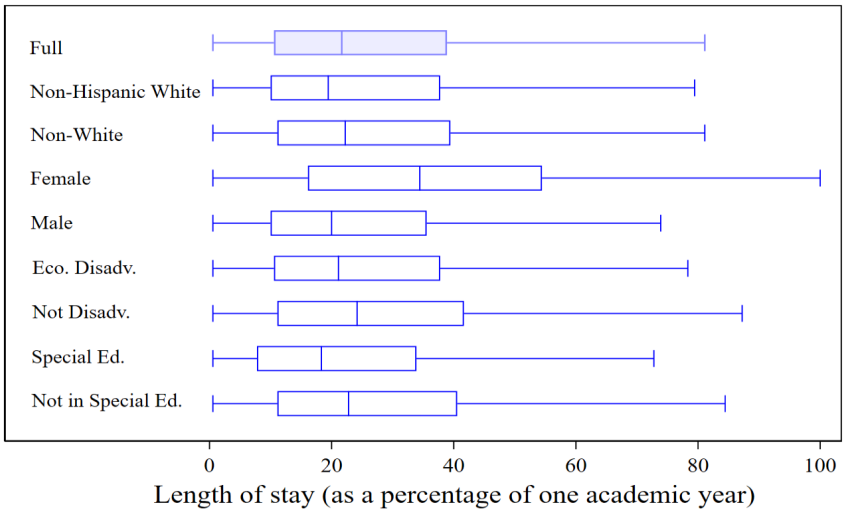
Notes: The figure plots raw data trends and event study results. In Panel (a), I present raw trend in the average course fail rate from three academic years before and two academic years after SUD treatment, separately for treatment and matched control individuals. Panel (b) plots the regression analogue to raw trends presented in Panel (a). I plot the coefficients and 95% confidence intervals on the interactions between the indicator for a treatment individual and the indicators for the years around the time of SUD treatment initiation from equation (1.2). Standard errors are clustered at the individual level.

Figure 1.9: Impacts of SUD Treatment Center School Attendance on Short-Run Educational Outcomes



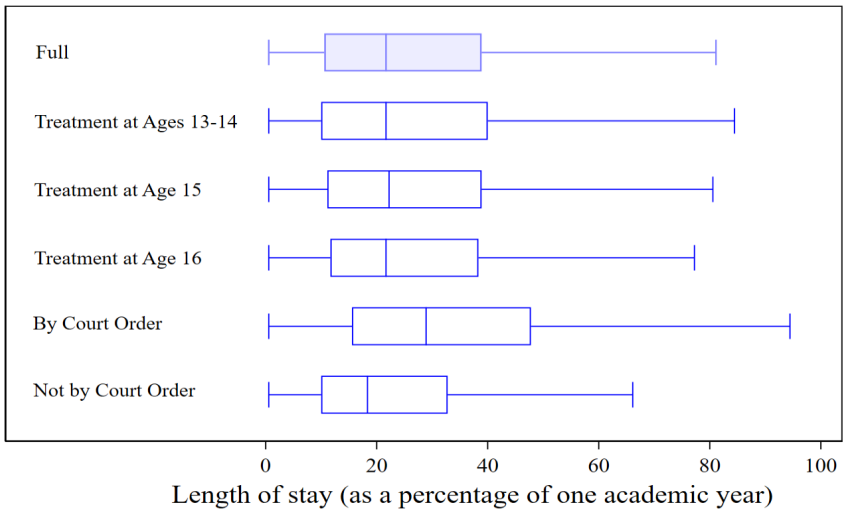
Notes: The figure plots the coefficients and 95% confidence intervals from equation (1.1), in which I pool the post-treatment periods to capture the average effects of attending a SUD treatment center on each of the following short-run outcomes: (i) the continuous absence rate, (ii) an indicator for chronic absenteeism, (iii) an indicator for not being in the public school system, (iv) an indicator for being disciplined in school, and (v) the continuous course fail rate. Standard errors are clustered at the individual level.

Figure 1.10: Distribution of the Length of Stay by Individual Characteristics



Notes: These box plots show the distribution of the length of stay in a treatment center school within the first year of SUD treatment initiation separately for each sub-group. The whiskers show the lower and upper extreme values (excluding outliers); and vertical lines show the 25th percentile, median, 75th percentile of the length of stay. The length of stay is winsorized at 180 school days (i.e., about an academic year) and then expressed as a percentage of one academic year.

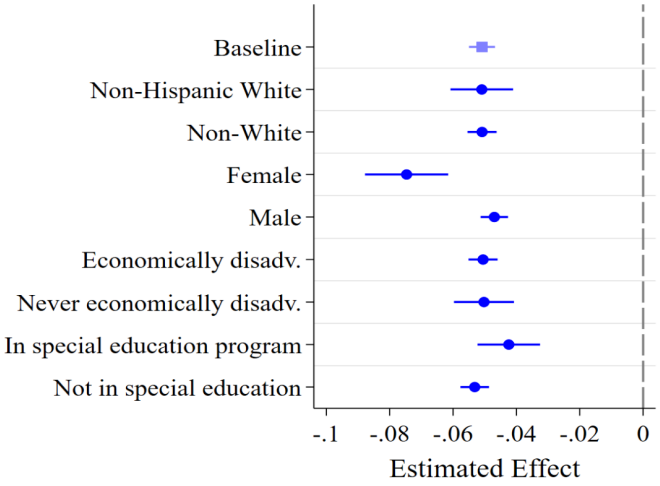
Figure 1.11: Distribution of the Length of Stay by Treatment Characteristics



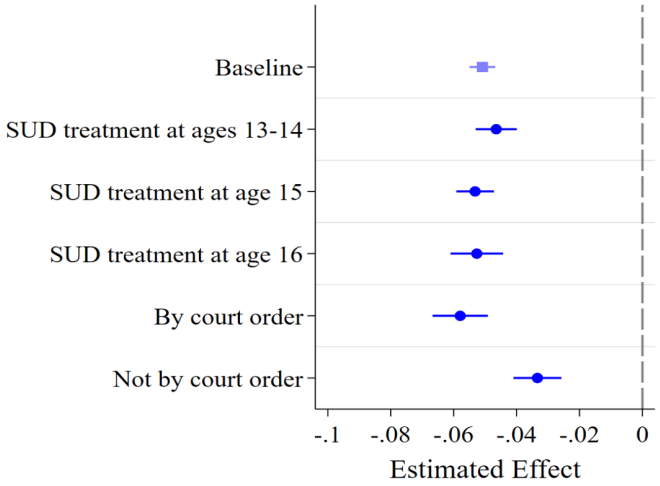
Notes: These box plots show the distribution of the length of stay in a treatment center school within the first year of SUD treatment initiation separately for each sub-group. The whiskers show the lower and upper extreme values (excluding outliers); and vertical lines show the 25th percentile, median, 75th percentile of the length of stay. The length of stay is winsorized at 180 school days (i.e., about an academic year) and then expressed as a percentage of one academic year.

Figure 1.12: Heterogeneity in the Short-Run Effects: Absence Rate

(a) By Demographic Characteristics



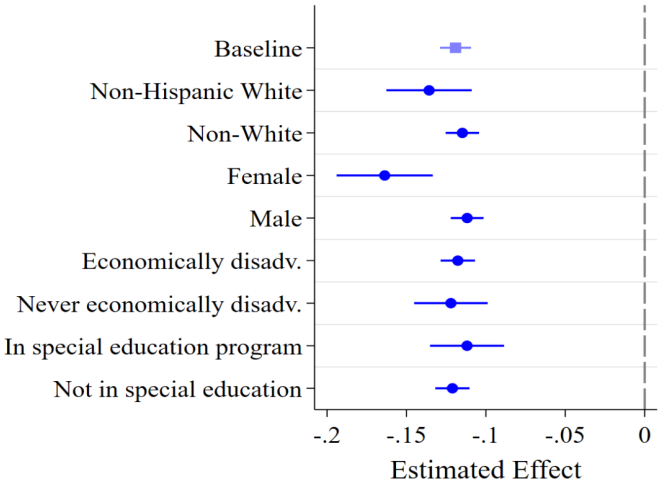
(b) By Treatment Characteristics



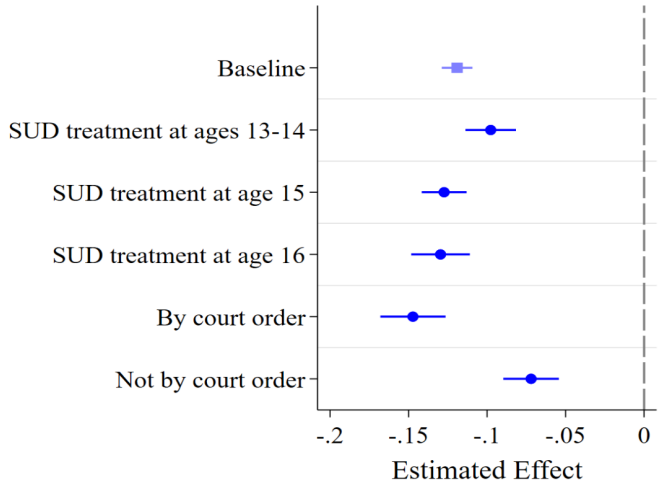
Notes: The figure presents the effect of treatment school attendance on the outcome for individuals belonging to the sub-group presented on the y-axis. Panel (a) includes the following sub-groups based on individual characteristics: (1) Non-Hispanic White, (2) Non-White (Hispanic or Non-Hispanic Black), (3) female, (4) male, (5) economically disadvantaged (measured using free/reduced-price lunch receipt in the two years prior to detention), (6) not economically disadvantaged, (7) in a special education program (measured in the two years prior to detention), and (8) not in a special education program. In Panel (b), I investigate heterogeneity by treatment characteristics using the following sub-groups: (1) treatment at ages 13–14, (2) treatment at age 15, (3) treatment at age 16, (4) by court order, and (5) not by court order (i.e., by referral).

Figure 1.13: Heterogeneity in the Short-Run Effects: Chronic Absenteeism

(a) By Demographic Characteristics



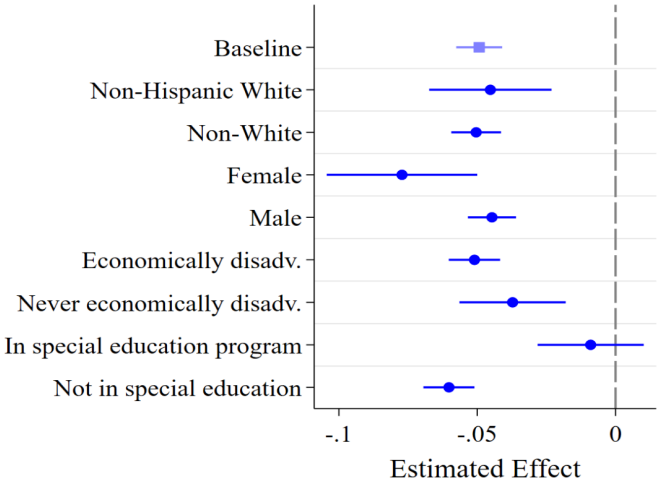
(b) By Treatment Characteristics



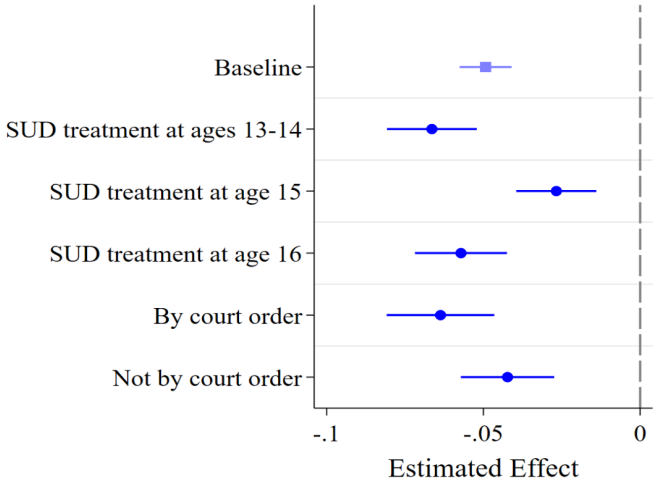
Notes: The figure presents the effect of treatment school attendance on the outcome for individuals belonging to the sub-group presented on the y-axis. Panel (a) includes the following sub-groups based on individual characteristics: (1) Non-Hispanic White, (2) Non-White (Hispanic or Non-Hispanic Black), (3) female, (4) male, (5) economically disadvantaged (measured using free/reduced-price lunch receipt in the two years prior to detention), (6) not economically disadvantaged, (7) in a special education program (measured in the two years prior to detention), and (8) not in a special education program. In Panel (b), I investigate heterogeneity by treatment characteristics using the following sub-groups: (1) treatment at ages 13–14, (2) treatment at age 15, (3) treatment at age 16, (4) by court order, and (5) not by court order (i.e., by referral).

Figure 1.14: Heterogeneity in the Short-Run Effects: Not in Public School

(a) By Demographic Characteristics



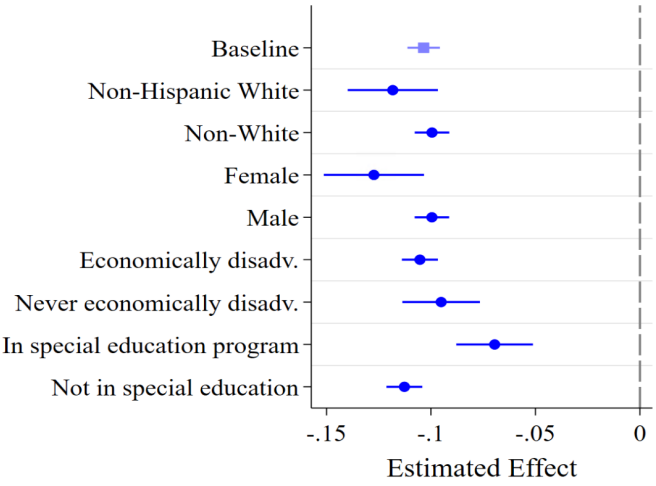
(b) By Treatment Characteristics



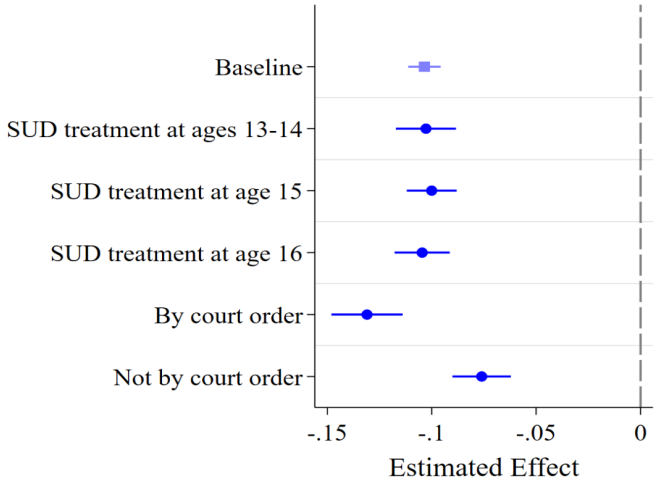
Notes: The figure presents the effect of treatment school attendance on the outcome for individuals belonging to the sub-group presented on the y-axis. Panel (a) includes the following sub-groups based on individual characteristics: (1) Non-Hispanic White, (2) Non-White (Hispanic or Non-Hispanic Black), (3) female, (4) male, (5) economically disadvantaged (measured using free/reduced-price lunch receipt in the two years prior to detention), (6) not economically disadvantaged, (7) in a special education program (measured in the two years prior to detention), and (8) not in a special education program. In Panel (b), I investigate heterogeneity by treatment characteristics using the following sub-groups: (1) treatment at ages 13–14, (2) treatment at age 15, (3) treatment at age 16, (4) by court order, and (5) not by court order (i.e., by referral).

Figure 1.15: Heterogeneity in the Short-Run Effects: Not in Public School or Chronic Absenteeism

(a) By Demographic Characteristics



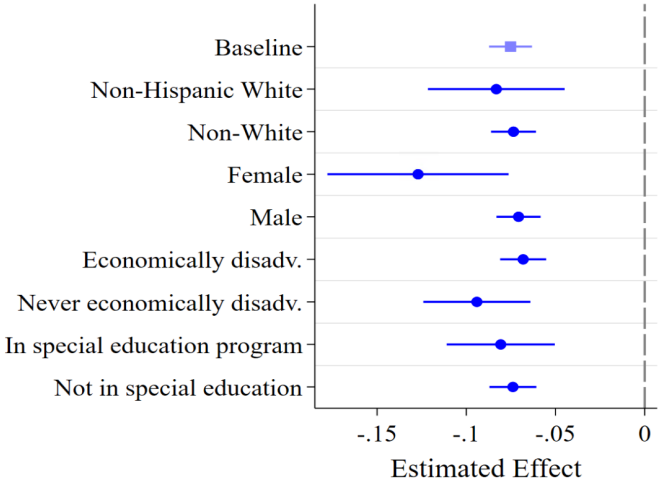
(b) By Treatment Characteristics



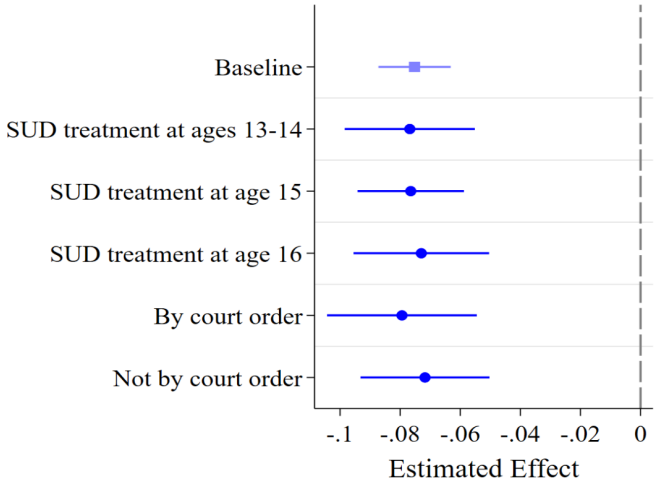
Notes: The figure presents the effect of treatment school attendance on the outcome for individuals belonging to the sub-group presented on the y-axis. Panel (a) includes the following sub-groups based on individual characteristics: (1) Non-Hispanic White, (2) Non-White (Hispanic or Non-Hispanic Black), (3) female, (4) male, (5) economically disadvantaged (measured using free/reduced-price lunch receipt in the two years prior to detention), (6) not economically disadvantaged, (7) in a special education program (measured in the two years prior to detention), and (8) not in a special education program. In Panel (b), I investigate heterogeneity by treatment characteristics using the following sub-groups: (1) treatment at ages 13–14, (2) treatment at age 15, (3) treatment at age 16, (4) by court order, and (5) not by court order (i.e., by referral).

Figure 1.16: Heterogeneity in the Short-Run Effects: Disciplinary Action

(a) By Demographic Characteristics



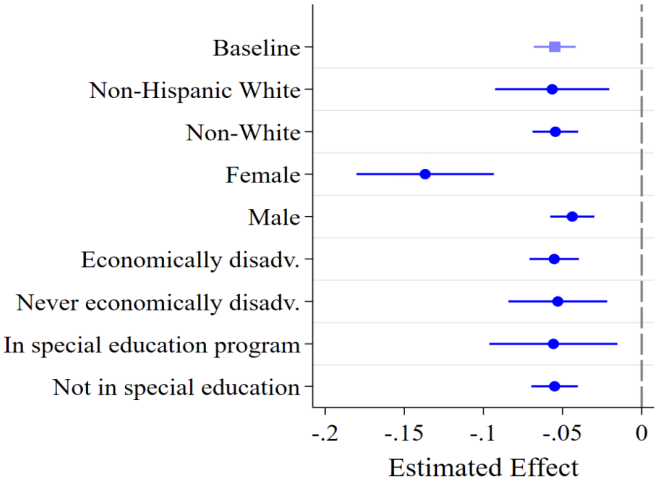
(b) By Treatment Characteristics



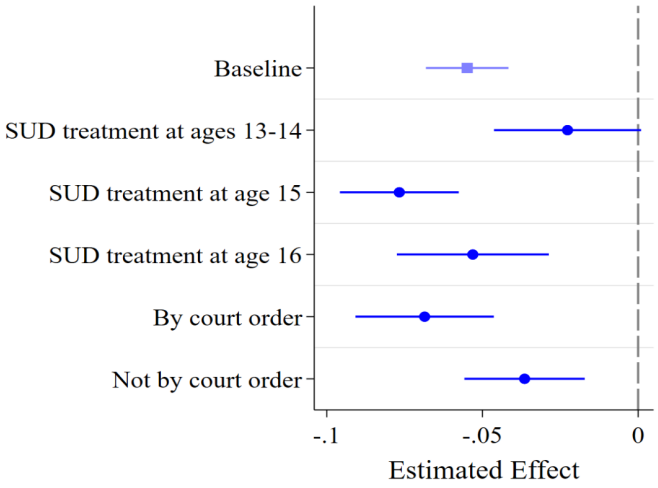
Notes: The figure presents the effect of treatment school attendance on the outcome for individuals belonging to the sub-group presented on the y-axis. Panel (a) includes the following sub-groups based on individual characteristics: (1) Non-Hispanic White, (2) Non-White (Hispanic or Non-Hispanic Black), (3) female, (4) male, (5) economically disadvantaged (measured using free/reduced-price lunch receipt in the two years prior to detention), (6) not economically disadvantaged, (7) in a special education program (measured in the two years prior to detention), and (8) not in a special education program. In Panel (b), I investigate heterogeneity by treatment characteristics using the following sub-groups: (1) treatment at ages 13–14, (2) treatment at age 15, (3) treatment at age 16, (4) by court order, and (5) not by court order (i.e., by referral).

Figure 1.17: Heterogeneity in the Short-Run Effects: Course Fail Rate

(a) By Demographic Characteristics

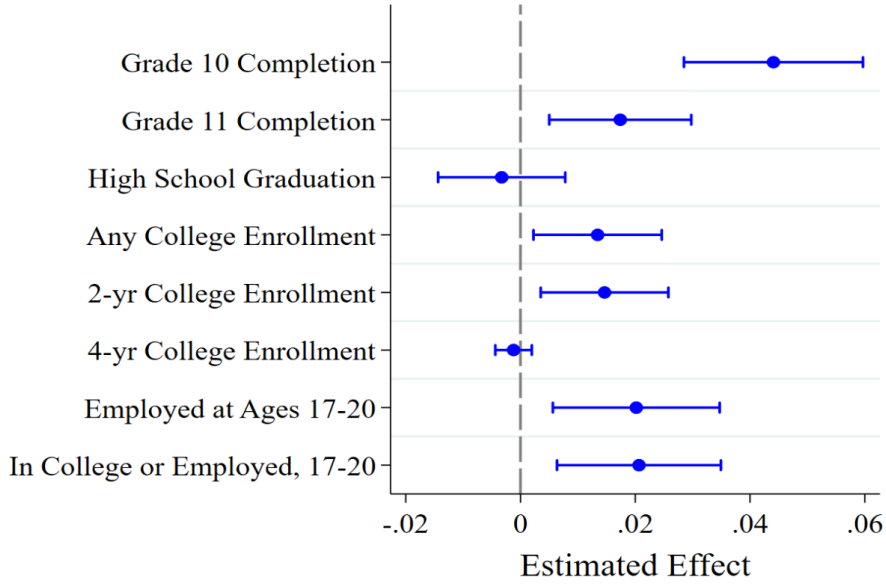


(b) By Treatment Characteristics



Notes: The figure presents the effect of treatment school attendance on the outcome for individuals belonging to the sub-group presented on the y-axis. Panel (a) includes the following sub-groups based on individual characteristics: (1) Non-Hispanic White, (2) Non-White (Hispanic or Non-Hispanic Black), (3) female, (4) male, (5) economically disadvantaged (measured using free/reduced-price lunch receipt in the two years prior to detention), (6) not economically disadvantaged, (7) in a special education program (measured in the two years prior to detention), and (8) not in a special education program. In Panel (b), I investigate heterogeneity by treatment characteristics using the following sub-groups: (1) treatment at ages 13–14, (2) treatment at age 15, (3) treatment at age 16, (4) by court order, and (5) not by court order (i.e., by referral).

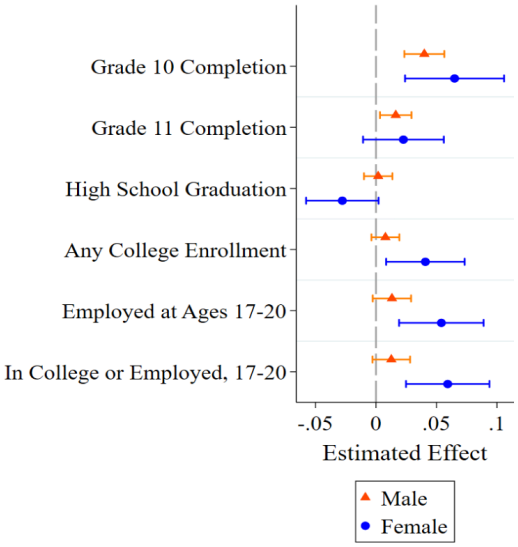
Figure 1.18: Long-Run Impacts of SUD Treatment Center School Attendance on Educational Outcomes and Employment



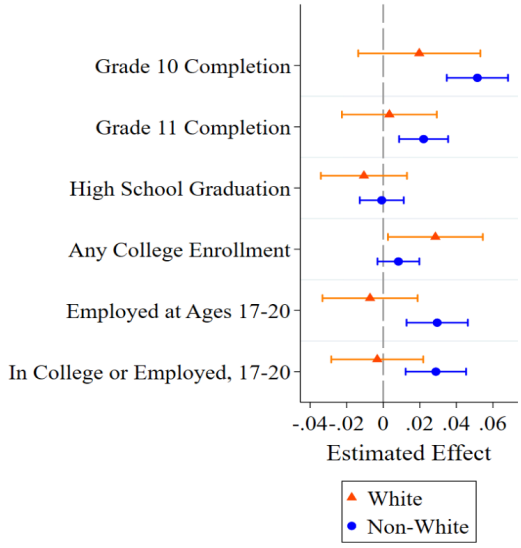
Notes: The figure shows the long-run impacts of SUD treatment center school attendance on educational outcomes through age 20 and employment at age 17–20. Specifically, the figure plots the coefficients and 95% confidence intervals on the indicator for treatment individuals from estimation of equation (1.3).

Figure 1.19: Heterogeneity in the Long-Run Effects by Individual Characteristics

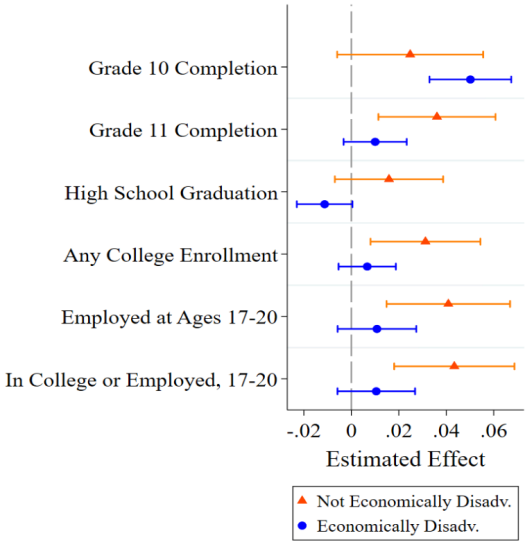
(a) Heterogeneity by Gender



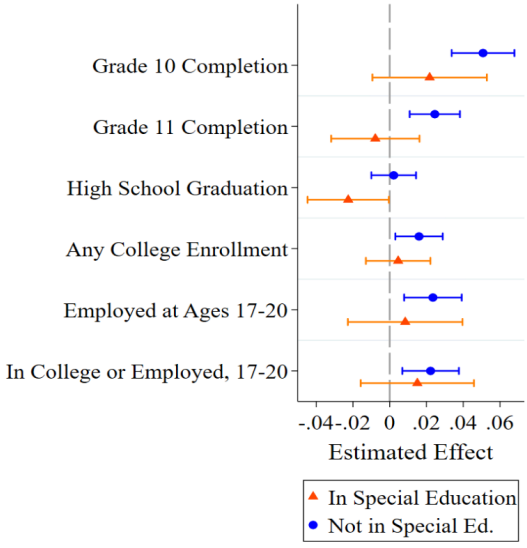
(b) Heterogeneity by Race/Ethnicity



(c) Heterogeneity by Socioeconomic Status



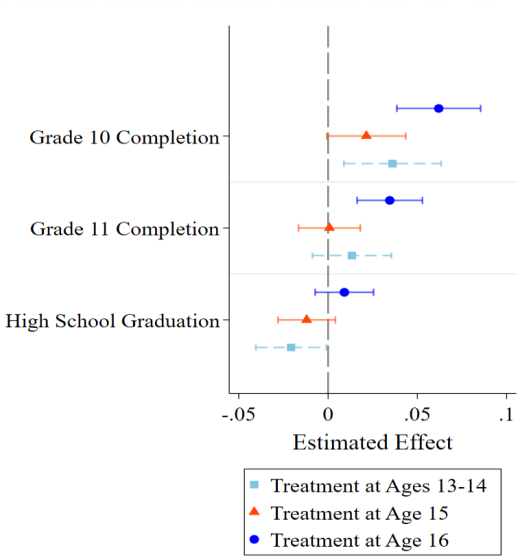
(d) Heterogeneity by Special Education



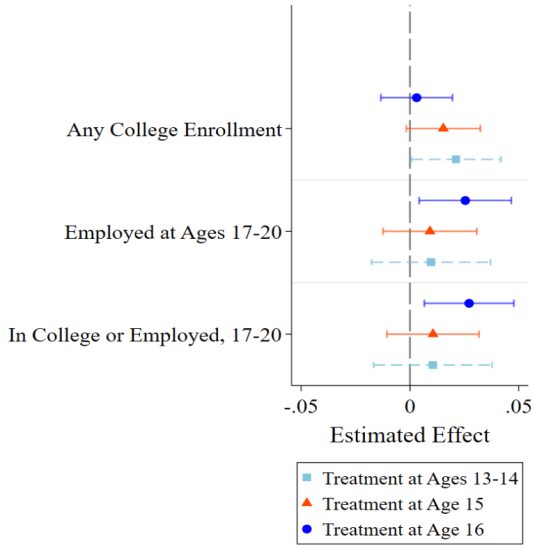
Notes: The panels in this figure present the long-run impacts of SUD treatment center school attendance on educational and employment outcomes for individuals belonging to each sub-group.

Figure 1.20: Heterogeneity in the Long-Run Effects by Age at the Time of SUD Treatment

(a) Grade Completion and Graduation

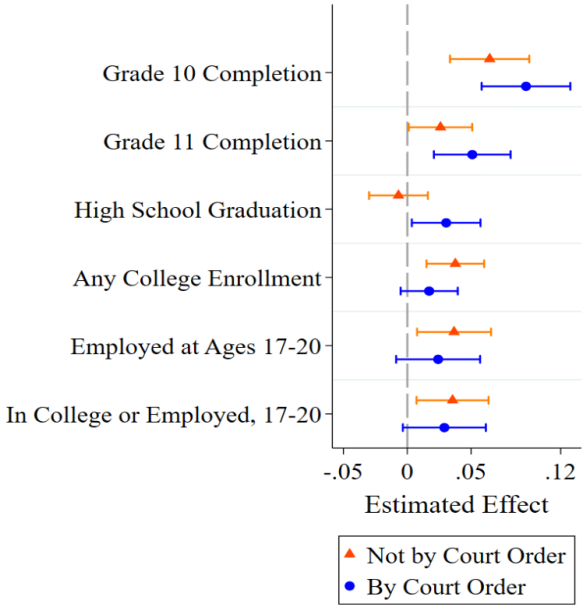


(b) College Enrollment and Employment



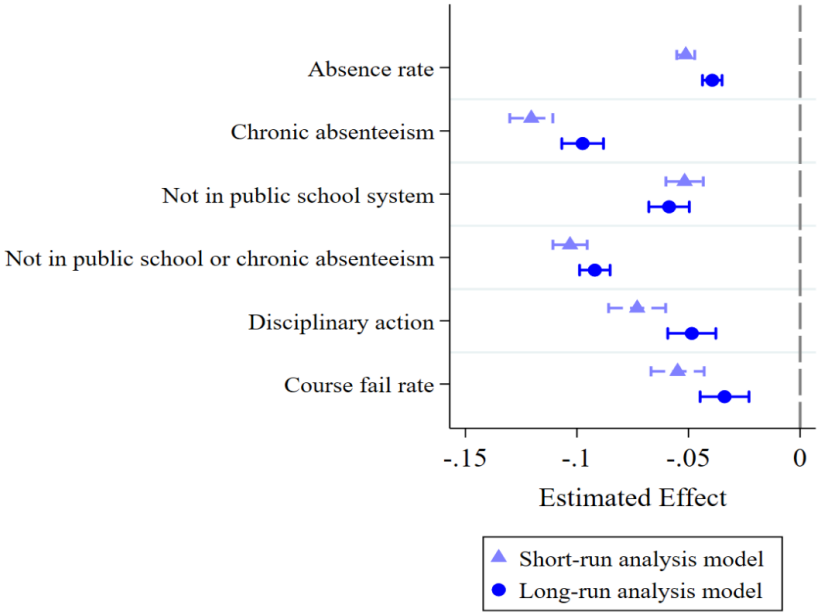
Notes: The panels in this figure present the long-run impacts of SUD treatment center school attendance on educational and employment outcomes for individuals belonging to each sub-group.

Figure 1.21: Heterogeneity in the Long-Run Effects by Court Order Status



Notes: The panels in this figure present the long-run impacts of SUD treatment center school attendance on educational and employment outcomes for individuals belonging to each sub-group.

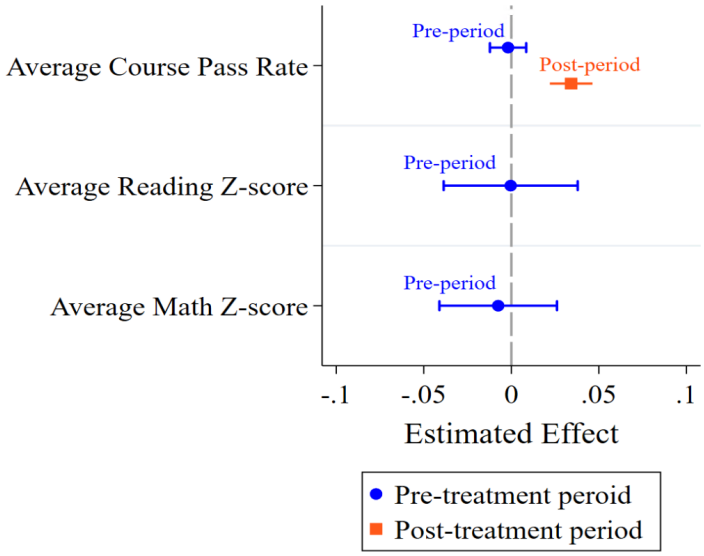
Figure 1.22: Long-Run Analysis Model with Short-Run Analysis Outcomes



Notes: The figure plots the coefficients and 95% confidence intervals from estimation of the long-run analysis model (equation (1.3)) with my short-run analysis outcomes as the dependent variables. My baseline estimates are presented in light blue.

Figure 1.23: Alternative Explanation: Difference in Underlying Ability

(a) Academic Performance in the Pre-Treatment Period



Notes: The figure plots the coefficients and 95% confidence intervals from estimation of equation (1.3) with the following outcomes as the dependent variables: (1) the average past course pass rate, (2) the average past Z-score for standardized reading tests, and (3) average past Z-score for standardized math tests, all measured in the two academic years prior to SUD treatment initiation. The estimates in red indicate output from equation (1.3) with the average course pass rate measured in the first two academic years following SUD treatment as the dependent variable.

Table 1.1: Average Individual Characteristics Across Treatment and Matched Control Individuals

	Treatment	Matched Controls	Diff	*p-val*
	(1)	(2)	(1) - (2)	
A. Individual Characteristics (Exact Matching Variables)				
Female	0.146	0.146	0.000	[1.000]
Non-Hispanic White	0.230	0.230	0.000	[1.000]
Hispanic	0.610	0.610	0.000	[1.000]
Non-Hispanic Black	0.157	0.157	0.000	[1.000]
Age at detention	14.841	14.859	-0.018	[<0.001]
Free/reduced-price lunch	0.752	0.752	0.000	[1.000]
Special education	0.207	0.207	0.000	[1.000]
Urbanicity of county				
Large central metro	0.619	0.619	0.000	[1.000]
Large fringe metro	0.163	0.163	0.000	[1.000]
Medium metro	0.183	0.183	0.000	[1.000]
Small metro	0.029	0.029	0.000	[1.000]
Micropolitan	0.004	0.004	0.000	[1.000]
Noncore	0.003	0.003	0.000	[1.000]
B. Average Absence Rate and Detention History at Baseline (Fuzzy Matching Variables)				
Average absence rate, 1 yr before	0.235	0.217	0.018	[<0.001]
Share of periods detained, 1 yr before	0.098	0.083	0.015	[<0.001]
Share of periods detained, 2 yr before	0.033	0.031	0.002	[0.001]
C. Academic Performance at Baseline (Non-Matching Variables)				
Grade at detention	9.023	9.055	-0.033	[<0.001]
Average past course pass rate, above median	0.609	0.615	-0.006	[0.103]
Average past reading z-score, above median	0.553	0.556	-0.003	[0.399]
Average past math z-score, above median	0.597	0.597	0.000	[0.972]
Number of individuals (weighted)	4,034	4,034		
Number of total individuals (unweighted)	4,034	35,714		
Number of unique individuals	4,034	13,212		

Notes: This table reports average individual characteristics and academic performance measured in the pre-detention period for the 4,034 treatment individuals included in the final analysis sample (column (1)) and the matched control individuals (column (2)). The third column presents the differences between mean characteristics between these two groups, and the fourth column presents $p-$values from tests of these differences.

Table 1.2: Short-Run Effects of SUD Treatment Center School Attendance on Educational Outcomes

	Absence Rate	Chronic Absence	Not in Public School	(2) or (3)	Disc. Action	Course Fail Rate
	(1)	(2)	(3)	(4)	(5)	(6)
Treated Individual x Post	-0.0509	-0.1191	-0.0493	-0.1035	-0.0752	-0.0549
	(0.0021)	(0.0050)	(0.0042)	(0.0040)	(0.0061)	(0.0068)
	[<0.001]	[<0.001]	[<0.001]	[<0.001]	[<0.001]	[<0.001]
Control group (post-treatment period) mean	0.1852	0.5076	0.4535	0.7566	0.2681	0.3420
Effect size relative to the control group mean	-27.48%	-23.46%	-10.87%	-13.68%	-28.05%	-16.05%
Treated individuals	3,697	3,697	3,697	3,697	1,391	2,568
Control individuals (weighted)	3,697.0	3,697.0	3,697.0	3,697.0	1,391.0	2,499.3
Control individuals (total)	33,308	33,308	33,308	33,308	14,361	23,987
Control individuals (unique)	12,145	12,145	12,145	12,145	7,441	9,070
Individual-grading period observations	717,734	717,734	925,125	925,125	306,433	-
Individual-year observations	-	-	-	-	-	88,529
R-squared	0.549	0.504	0.663	0.498	0.496	0.700

Notes: This table presents coefficients, standard errors (in parentheses), and $p-$values [in brackets] from estimation of equation (1.1). Standard errors are clustered at the individual level.

Table 1.3: Long-Run Effects of SUD Treatment Center School Attendance on Educational Outcomes by Age 20

	Grade 10 Completion	Grade 11 Completion	High School Graduation	Maximum Grade Level Completed
	(1)	(2)	(3)	(4)
Treated Individual	0.0441	0.0174	-0.0033	0.1141
	(0.008)	(0.0063)	(0.0057)	(0.0163)
	[<0.001]	[0.006]	[0.561]	[<0.001]
Control group outcome mean	0.2857	0.1707	0.1320	9.1309
Effect size relative to the control group mean	15.44%	10.19%	-2.50%	1.25%
Treated individuals	2,967	3,240	3,252	3,252
Control individuals (weighted)	2,963.2	3,239.6	3,252.0	3,252.0
Control individuals (total)	27,461	28,682	28,723	28,723
Control individuals (unique)	10,152	10,818	10,841	10,841
Observations	30,428	31,922	31,975	31,975
R-squared	0.469	0.464	0.433	0.576

Notes: This table presents coefficients, standard errors (in parentheses), and $p-$values [in brackets] from estimation of equation (1.3). Standard errors are clustered at the individual level.

Table 1.4: Long-Run Effects of SUD Treatment Center School Attendance on College Enrollment and Employment by Age 20

	Enroll Any College	Enroll 2-yr Col.	Enroll 4-yr Col.	Employed, Ages 17–20	In College or Employed
	(1)	(2)	(3)	(4)	(5)
Treatment Individual	0.0134	0.0146	-0.0012	0.0202	0.0206
	(0.0057)	(0.0057)	(0.0016)	(0.0074)	(0.0073)
	[0.018]	[0.010]	[0.456]	[0.007]	[0.005]
Control group outcome mean	0.1150	0.1089	0.0061	0.7571	0.7662
Effect size relative to the control group mean	11.65%	13.41%	-19.67%	2.67%	2.69%
Treatment individuals	3,160	3,160	3,160	3,160	3,160
Control individuals (weighted)	3,186.4	3,186.4	3,186.4	3,186.4	3,186.4
Control individuals (total)	28,161	28,161	28,161	28,161	28,161
Control individuals (unique)	10,610	10,610	10,610	10,610	10,610
Observations	31,321	31,321	31,321	31,321	31,321
R-squared	0.387	0.377	0.357	0.382	0.383

Notes: This table presents coefficients, standard errors (in parentheses), and $p-$values [in brackets] from estimation of equation (1.3). Standard errors are clustered at the individual level.

Chapter 2

Must-Access Prescription Drug Monitoring Programs and the Opioid Overdose Epidemic: The Unintended Consequences*

2.1 Introduction

Opioid overdoses have reached epidemic levels in the United States. The Centers for Disease Control and Prevention (CDC) calls the opioid crisis "the worst drug epidemic in US history" (Kolodny et al. 2015). Between 1999 and 2017, overdose deaths from opioids, including prescription opioids and illegal opioids, increased six-fold. To contain the epidemic, federal and state governments implemented various policies that limit access to prescription opioids to reduce addiction, and several studies show that these drug control policies did reduce prescription opioid abuse (e.g., Buchmueller and Carey (2018); Cicero and Ellis (2015); Grecu et al. (2019)). Additionally, deaths from prescription opioids decreased by more than 6% between 2011 and 2013 and have remained relatively steady since then. These decreases, however, were limited to prescription opioids. During the same period, deaths from illegal opioids, such as heroin and illicitly made fentanyl, began to increase sharply, and between 2011 and 2016 they more than tripled.[1] As a result, overdose

*This manuscript is the peer-reviewed version of the following article: "Must-Access Prescription Drug Monitoring Programs and the Opioid Overdose Epidemic: The Unintended Consequences," which has been published in its final form in the *Journal of Health Economics*. The final published version is available at https://doi.org/10.1016/j.jhealeco.2020.102408 (Kim, 2021a). I thank the editor, two anonymous referees, David Beheshti, Marika Cabral, Michael Geruso, Sungwon Lee, Seth Neller, Kosali Simon, Tom Vogl, and seminar participants at the University of Texas at Austin and the Texas Health Economics Conference 2019 for helpful comments.

[1]Throughout this paper, when I use the term *opioid* to refer to all opioids, heroin is included.

deaths from opioids, including both legal and illegal opioids, steadily increased between 1999 and 2017 and grew faster in the last few years, as shown in Figure 2.1. The worsening epidemic sparked a debate about the effectiveness of supply-side interventions: do policies that limit access to legal opioids cause users to transition from prescription opioids to illegal opioids?

I study whether and to what extent a supply-side intervention can have a spillover effect on illegal opioid use, focusing on prescription drug monitoring programs (PDMPs), one of the most widely adopted statewide drug policies. A PDMP is a state-operated database of patient prescriptions for controlled substances. Authorized providers can access the PDMP database to identify the inappropriate use of pain medications. From the pre-1990s to 2016, all but one state implemented PDMPs (which I refer to as voluntary-access PDMPs). However, because provider access was voluntary, only a small percentage of providers actually enrolled in the program or requested patient histories (PDMP Center of Excellence 2014). From 2010 to 2012, the median PDMP registration rate of licensed prescribers who prescribed at least one controlled substance prescription was only 35%. (Kreiner et al. 2014).

In response to the low participation rates, 16 states implemented a must-access provision between 2007 and 2016, in addition to the existing voluntary-access PDMP. Must-access PDMPs legally require providers to use the PDMP before prescribing or dispensing under certain conditions. Kentucky's mandate on enrollment and PDMP use was associated with about an 8.5% lower overall dispensing of controlled substances in the first year following implementation, showing that mandates can effectively improve PDMP use (Substance Abuse 2017).

However, by making prescription opioids less accessible, must-access PDMPs may lead individuals to switch from prescription opioids to illegal opioids such as heroin or illegal fentanyl. For example, 94% of opioid-addicted individuals who switched from prescription opioids to heroin reported doing so because prescription opioids "were far more expensive and harder to obtain" (Cicero et al. 2014). Given the increased accessibility and reduced prices of heroin, the policy may have caused a significant transition from nonmedical use of prescription opioids to heroin use.

Using the National Vital Statistics System (NVSS) Multiple Cause of Death mortality files from 2003 to 2016, I exploit the variation in overdose deaths involving heroin or other types of opioids resulting from PDMP laws. The heroin mortality rate covers the entire population and has been widely used by researchers studying heroin use (e.g., Alpert et al. (2018); Evans et al. (2019); Kilby (2015); Meinhofer (2018a)). I also use administrative data from the Drug Enforcement Administration (DEA) to quantify how must-access PDMPs affected the legal supply of opioids and to account for the confounding effect of the OxyContin reformulation on heroin death rates. The OxyContin reformulation of 2010 was a nationwide drug policy aimed at reducing opioid abuse and is potentially a strong confounder.[2] Following the approach suggested by Alpert et al. (2018), I control for the differential effects of the reformulation across states by adding to my econometric model a measure of pre-reformulation OxyContin use interacted with the half-year fixed effects. I include this interaction in my preferred specification to address potential omitted variable bias that can arise from the pre-existing correlation between implementation of must-access PDMPs and exposure to the OxyContin reformulation.

I find strong evidence that must-access PDMPs increased heroin death rates and that voluntary-access PDMPs had no substantial effect. Using a difference-in-differences specification that allows the treatment effect to vary over time, I show that the heroin death rate began to increase in the year of policy implementation, and the size of effects steadily grew over time.[3] My estimates indicate that two years after implementation, must-access PDMPs were associated with 0.9 more heroin deaths per 100,000 in a half-year period, relative to control states. The largest detrimental effect of the policy occurred three years after implementation. I graphically present my difference-in-differences estimates and show that the trends in heroin mortality were not different across treatment and control states prior to implementation, providing evidence in support of the parallel trends identifying

[2]Several studies have shown that the OxyContin reformulation caused a transition from nonmedical use of prescription opioids to heroin use, and the heroin death rate began to increase sharply following the reformulation (e.g., Alpert et al. (2018); Evans et al. (2019)).

[3]It may take time for both opioid abusers and physicians to adjust their behavior. Consumers may gradually switch to illegal drugs and providers may also take time to become familiar with the PDMP system and to adjust their prescribing behavior. Some studies emphasize that it is costly for providers to adjust their practice style (e.g., Clemens and Gottlieb (2014); Frank and Zeckhauser (2007)).

assumption.

Moreover, I find that the increase in heroin mortality coincided with a sudden decrease in prescription opioid mortality and a decrease in the legal supply of opioids following policy implementation, suggesting that the policies caused users to transition from prescription opioids to heroin. My estimates suggest that even if must-access PDMPs reduced prescription opioid deaths, the decrease was offset by an increase in deaths from illegal opioids, including heroin. Overall, I show that must-access PDMPs had no substantial effect on total opioid-related deaths in the short term because of these offsetting effects.[4] In the longer term, however, the policies were associated with increased total opioid deaths because the large increase in illegal opioid deaths surpassed the decrease in prescription opioid deaths.

The findings of this paper add to the literature on the spillover effect of PDMPs on heroin use. Most of the previous papers in this literature have focused on the period before 2014 and have found weak or no effects of PDMPs on heroin-related outcomes. Earlier studies could not distinguish between voluntary- and must-access PDMPs (e.g., Radakrishnan (2015); Nam et al. (2017)), and some prior work has used survey or treatment admissions data, which are likely to underreport heroin use, and find no effects (e.g., Radakrishnan (2015); Ali et al. (2017); Grecu et al. (2019)).[5] Kilby (2015) finds only a temporary effect of voluntary-access PDMPs on heroin mortality. Using data through 2013, Meinhofer (2018a) finds suggestive evidence that must-access PDMPs increased heroin-related overdose deaths, although these findings are sensitive to the model specification.[6] My paper contributes to this literature by providing robust, causal estimates

[4]Total opioid-related deaths indicate the deaths that involved any opioid, including both prescription opioids and illegal opioids, at the time of death.

[5]Ali et al. (2017) show that heroin use, dependence, and initiation have no statistically significant association with either voluntary-access or must-access PDMPs but find a statistically significant association between voluntary-access PDMPs and the increased number of days of heroin use.

[6]My paper is different from Meinhofer (2018a) on two dimensions—data period and research design. Both studies use the same mortality data, but I use data through 2016 and Meinhofer (2018a) uses data through 2013. Also, the outcome variables and model specifications are different in the two papers: I use death rates as the outcome and control for other co-occurring opioid-related policies, while Meinhofer (2018a) uses the log of deaths as the outcome and includes the log of population and state-specific time trends in her econometric model. In Appendix Section C, I discuss in detail the differences between the two studies.

of the medium-term effect of must-access PDMPs on heroin-related mortality. Two key innovations allow for this contribution. First, I utilize data through 2016, allowing for the inclusion of several additional must-access PDMP implementations and a longer post-treatment period. My findings suggest that estimating the longer-run impact is crucial to identifying the effect on heroin-related deaths.[7] Second, I demonstrate that the results are robust to controlling for other co-occurring state and national opioid-related policies. By employing more recent data than prior work, I am able to include several more reforms in the analysis that allow me to flexibly account for potential confounding effects of state and national opioid-related policies.

This paper also contributes to the literature on the unintended consequences of supply-side drug policies. Evans et al. (2019) and Alpert et al. (2018) investigated the consequences of the 2010 OxyContin reformulation and found strong evidence of the movement from legal opioids to heroin. My findings add to this literature and suggest that a supply-side intervention that controls access to legal opioids can have the unintended consequence of increased illegal drug use.

The results of this study have clear policy implications: the existence of accessible and affordable close substitutes may reduce the effectiveness of supply-side drug policies. A supply-side intervention can control only the legal supply of opioids but not the demand for opioids. Demand-side interventions, such as improving access to treatment or prevention may be more effective in preventing and mitigating opioid abuse and should be aligned with the existing supply-side policies.

2.2 Background

2.2.1 Opioid Abuse

Opioids are a class of drugs that relieve severe pain. Opioids include the illegal drug heroin, synthetic opioids such as fentanyl, and prescription medications, such as oxycodone, hydrocodone, and morphine. If used medically, prescription opioids help relieve pain. However, continued use or abuse of opioids can lead to addiction, tolerance, and

[7] In Appendix Section C, I address in detail the consequences of using additional data.

physiological dependence.

2.2.2 Prescription Drug Monitoring Programs

Prescription drug monitoring programs, or PDMPs, are state-level databases that collect information on patients' opioid prescriptions at the point of dispensing (Davis et al. 2014). In most states, authorized providers may access the state's database to identify inappropriate use of pain medications. The earliest programs were based on carbon copies and, thus, did not have the capabilities of the modern electronic system, and only program staff, authorized law enforcement, and regulatory agencies could access the data (OIG 1992). Many early electronic PDMPs required that data be sent only infrequently using methods that are now outdated (Horwitz et al. 2018). PDMPs now involve automated reporting, transitioned to a modern electronic system with increased reporting frequency (Buchmueller and Carey 2018). The types of users permitted to access PDMP data have also been significantly increased: the proportion of state PDMPs that allow physicians to directly access patient-identifiable data increased from 23.1% in 1998 to 93.5% in 2011 (Davis et al. 2014).

However, even if more timely and complete patient prescription history data are available and accessible, provider participation rates are low when PDMP is not mandated (Haffajee et al. 2015).[8] In response to the low participation rates, 16 states implemented a must-access provision on top of the existing voluntary-access PDMPs between 2007 and 2016. Must-access PDMPs legally require providers to use the PDMP before actual prescribing or dispensing under certain conditions. Must-access provisions have successfully increased provider utilization. In Kentucky, Tennessee, New York, and Ohio, must-access provisions increased providers' registration and utilization of PDMPs and decreased the prescription of certain drugs (PDMP Center of Excellence 2016).

Table 2.1 shows the start dates of the laws investigated in this paper. Horwitz et al. (2018) serves as the source of information for the month and year that states first enacted

[8] As noted above, the median PDMP registration rate, defined as the proportion of prescribers registered to use the PDMP among licensed prescribers who issued one or more controlled substance prescription between 2010 and 2012, was 35% (Kreiner et al. 2014).

any type of PDMP.[9] By the end of 2016, all states except Missouri had passed some type of PDMP laws. Effective dates of must-access PDMPs, obtained from Mallatt (2019), are listed in the second column of Table 2.1.[10][11] I use these dates for my main analysis and test the robustness of the results using alternative dates of must-access PDMPs taken from Sacks et al. (2019), which are listed in the third column of Table 2.1. In the last column of Table 2.1, I report dates for pill mill laws suggested by Mallatt (2018).[12]

In my main analysis, I do not take into account differences among must-access laws, although the strength of these laws varies greatly across states. Delaware's PDMP requires provider access only with reasonable suspicion of abuse, as did the initial PDMP of Ohio (until 2015). Prior to 2015, Oklahoma's initial law applied only to methadone, and Vermont's initial PDMP required access only the provider wrote a replacement prescription for one that had been lost or stolen. In contrast, Kentucky, Massachusetts, New Mexico, New York, Tennessee, West Virginia, and the recent laws in Ohio, Oklahoma, and Vermont applied must-access laws to all care settings and ingredients, and required providers to access the PDMP even without suspicion of abuse. In Section 2.5.6, I identify heterogeneity in the effects of a must-access PDMP by the strength of the law.

[9] In Appendix Table B.2, I report heroin mortality results based on alternative enactment dates from the Prescription Drug Abuse Policy System (PDAPS) and the National Alliance for Model State Drug Laws (NAMSDL), which were the most commonly used in previous papers. In the last column of Appendix Table B.2, I also report the results using the dates PDMP data became accessible to any authorized user, suggested by Horwitz et al. (2018), instead of enactment dates.

[10] See Mallatt (2019) and https://sites.google.com/site/justinemallatt/home/pdmp-dates for more detailed information.

[11] South Carolina enacted a must-access law in April 2016, but the law applied only to Medicaid and state health plans. Following Mallatt (2019), I code South Carolina as a voluntary-access PDMP, but I obtain similar results when I code it as having a must-access PDMP in 2016.

[12] Although not reported in the paper, my results are robust to using several alternative start dates of pill mill laws (e.g., PDAPS, Mallatt (2019)). Given the robustness of my results, I follow Mallatt (2018) because it provides dates of pill mill laws that are more comparable to those from Buchmueller and Carey (2018). In this paper, I do not use the policy dates from Buchmueller and Carey (2018) because their sample period is shorter than mine.

2.2.3 Substitution of Heroin

An important feature of the opioid addiction epidemic is the relationship between prescription opioids use and heroin use (Kolodny et al. 2015). Heroin is a highly addictive illegal drug made from morphine and is pharmacologically similar to prescription opioids. Prior nonmedical use of prescription opioids may lead to heroin use (Becker et al. 2008; Muhuri et al. 2013). According to the federal government's National Survey on Drug Use and Health (NSDUH), 79.5% of individuals who use heroin for the first time report previous nonmedical use of prescription opioids (Muhuri et al. 2013).[13] Using national-level data, Becker et al. (2008) showed that heroin users are 3.9 times as likely to report nonmedical use of opioids in the previous year. These studies provide a clear link between prescription opioid abuse and heroin use and suggest that heroin is a close substitute for prescription opioids.

How do must-access laws cause a transition from nonmedical use of prescription opioids to heroin use? Must-access PDMPs directly affect the legal supply of opioids by limiting access to controlled substances. By making prescription opioids less accessible, the policy may also reduce prescription opioid abuse. Several studies have found that must-access PDMPs did reduce prescription opioid abuse (e.g., Birk and Waddell (2017); Buchmueller and Carey (2018); Grecu et al. (2019)). Appendix Figure B.1 suggests that the national trend in the legal supply of opioids is highly correlated with that in prescription opioid deaths.

However, as prescription opioids become less accessible because of the must-access law, individuals may substitute heroin for prescription opioids.[14] The magnitude of the actual substitution is determined by individual characteristics as well as accessibility to substitutes (Alpert et al. 2018). Given the increased accessibility, reduced prices, and the higher purity of heroin, must-access PDMPs may cause a transition from nonmedical use of prescription opioids to heroin use. Moreover, most heroin is now laced with illegal fentanyl, a synthetic

[13]Muhuri et al. (2013) report that the incidence of heroin use among people who reported previous nonmedical use of prescription opioids was 19 times as high as the incidence among individuals who reported no previous abuse.

[14]There is a large black market for opioids, an illegal trading system that avoids government regulation, on which individuals can buy close substitutes. This secondary market provides not only illegal opioids but also legal opioids. Highly regulated opioids, such as oxycodone, have fueled the black market for prescription opioids.

opioid that is 50–100 times stronger than morphine, to improve its potency. Therefore, must-access policies may cause even worse outcomes, by pushing people to more dangerous illegal opioids.

2.3 Data

2.3.1 Mortality

I use the National Vital Statistics System (NVSS) Multiple Cause of Death mortality files to study annual overdose deaths from 2003 to 2016. Following the coding suggested by the CDC, I categorize deaths related to opioids. First, I code drug overdose deaths using the ICD-10 underlying cause-of-death codes of unintentional (X40–X44), suicide (X60–X64), homicide (X85), and undetermined intent (Y10–Y14). Second, I use ICD-10 drug identification codes, which contain information about the drugs found in the body at death. The following four drug identification codes are used: T40.1 for heroin, T40.2 for natural and semisynthetic opioids such as oxycodone and hydrocodone, T40.3 for methadone, and T40.4 for synthetic opioids excluding methadone, such as fentanyl.

In this paper, I investigate four categories of drug overdose deaths: (i) deaths from heroin (T40.1), (ii) deaths from heroin or synthetic opioids other than methadone (T40.1, T40.4), (iii) deaths from natural and semisynthetic opioids (T40.2), and (iv) total deaths from any opioid, including heroin (T40.1–T40.4). Since heroin is an opioid, when I use the term *opioid* to refer to all opioids, heroin is included. A single overdose death often involves the presence of multiple drugs at the time of death.[15] The mortality outcomes I investigate in this paper are total mortality unless otherwise noted; total mortality may involve other drugs present at the time of death.[16] Although this approach does not allow for the death to be attributed to a single drug when multiple drugs are related to the death, increases or decreases in the involvement of a specific drug reflect substitution patterns (Alpert et al. 2018). To identify clear substitution patterns, I also investigate mortality outcomes based

[15]Therefore, a single death might be included in more than one category when calculating the number of overdose deaths involving specific drugs.

[16]For example, total heroin deaths include the deaths that involved not only heroin but also other types of opioids at the time of death.

on the exclusive involvement of a specific opioid. For example, the exclusive measure of heroin mortality, which I refer to as heroin-only mortality, indicates the deaths that involved heroin (T40.1) but not the other types of opioids (T40.2–T40.4) at the time of death.

To measure illegal opioid deaths, I use the second category of overdose death, which combines heroin and synthetic opioids other than methadone (hereinafter referred to as synthetic opioids).[17] While legal uses of fentanyl do exist, increases in synthetic opioid deaths since 2013 have been driven primarily by illicit fentanyl use (Rudd et al. 2016). I combine T40.1–T40.4 to measure total deaths from any opioid, including heroin, which I refer to as total opioid-related deaths. To measure deaths from prescription opioids, I use the third category of overdose deaths, which involves natural and semisynthetic opioids (hereinafter referred to as natural opioids). Earlier studies followed the CDC's traditional method of calculating prescription opioid deaths, which combined T40.2–T40.4. However, due to the recent surge in deaths that may involve illicit fentanyl, the CDC began analyzing synthetic opioids other than methadone (T40.4) separately from T40.2–T40.4.[18] I follow this more conservative method in this paper and also exclude methadone (T40.3), which had abnormal overdose trends, from my measure of prescription opioid deaths.[19] However, including methadone does not change my results pattern.

A major limitation of most prior estimates of opioid mortality rates is that they underreport actual rates because the specific drugs that caused the death are frequently not identified on the death certificates (Ruhm 2018). To obtain more accurate estimates of opioid mortality, I use corrected estimates of mortality rates following the method suggested by Ruhm (2018), which uses information from death certificates that specify at least one drug category to impute drug involvement for cases in which only unspecified drugs were mentioned on the death certificates.[20] Figure 2.1 shows that corrected rates are 20–35%

[17]Throughout this paper, I use the phrase *illegal opioid deaths* synonymously with *heroin and synthetic opioid deaths*.

[18]Data for synthetic opioid deaths involve both legal and illegal synthetic opioids because toxicology testing cannot distinguish between legal and illegal synthetic opioids.

[19]During 2002–2006, the methadone overdose death rate increased, on average, 22.1% per year; however, beginning with 2006 warnings from the Food and Drug Administration (FDA), efforts to reduce the use of methadone for pain have been made; as a result, after 2006, methadone overdose deaths declined 6.5% per year; see Jones et al. (2016) for a detailed description of trends in methadone overdose deaths.

[20]See Ruhm (2018) for more details on the method of computing the corrected mortality rates.

higher every year than reported rates for any type of opioid. Throughout the paper, I use the corrected mortality rates as the outcome variables.

2.3.2 Legal Supply of Opioids

I use administrative data on shipments of prescription opioids from the Drug Enforcement Administration (DEA)'s Automation of Reports and Consolidated Orders System (ARCOS) to examine whether must-access PDMPs reduce the legal supply of opioids. ARCOS is a federal data system initiated in response to the 1970 Controlled Substances Act that tracks the transactions and deliveries of controlled substances from manufacturers to retail distributors at the state level. ARCOS contains data on all Schedule I and II substances, as well as on narcotic substances in Schedule III that are sold or distributed. I use ARCOS data to examine whether must-access PDMPs reduce the total distribution of opioids by state, focusing on oxycodone and hydrocodone.[21] Oxycodone and hydrocodone, which are both semisynthetic opioids widely prescribed to treat pain, are some of the most commonly abused prescription opioids. The national trends in the legal supply of oxycodone and hydrocodone are presented in Panel A of Appendix Figure B.1.

2.3.3 Exposure to the OxyContin reformulation

To address potential omitted variable bias that can arise from the pre-existing correlation between implementation of must-access PDMPs and exposure to the 2010 OxyContin reformulation, I account for the reformulation in my econometric model. I proxy differential exposure to the reformulation across states with a measure of pre-reformulation OxyContin use. Following Alpert et al. (2018), I consider two alternative measures of OxyContin use. First, using ARCOS 2004–2009 data on the legal supply of opioids, I define OxyContin use as the relative importance of oxycodone compared to that of hydrocodone (oxycodone /

[21] I convert oxycodone and hydrocodone in grams, reported in the ARCOS data, into those in morphine milligram equivalents (MMEs) using the standard MME conversion factors (https://www.cms.gov/Medicare/Prescription-Drug-Coverage/PrescriptionDrugCovContra/Downloads/Oral-MME-CFs-vFeb-2018.pdf (last accessed May 2020)) and then dividing by 60 (to convert MMEs to doses).

[oxycodone + hydrocodone]) in per capita (morphine equivalent) doses.[22] Hydrocodone is considered a substitute for oxycodone, and states that disproportionately consume oxycodone relative to hydrocodone are expected to be more affected by the reformulation (Alpert et al. 2018). For each state, I calculate the population-weighted average of the relative importance of oxycodone compared to that of hydrocodone, combining the 2004h1 through 2009h2 periods.

Second, I use data from the National Survey on Drug Use and Health (NSDUH), which is a nationally representative household survey. The advantage of this survey is that it includes information on nonmedical use of OxyContin, although this data was self-reported. In the NSDUH data, OxyContin misuse rate is defined as the percentage of the population over the age of 12 indicating nonmedical use of OxyContin. Using the NSDUH data, Alpert et al. (2018) define each state's pre-reformulation OxyContin misuse as the population-weighted rate of OxyContin misuse that combines the 2004–2005, 2006–2007, and 2008–2009 waves. I use the same measure, which I refer to as the NSDUH measure hereinafter.

Two alternative measures of OxyContin use, the ARCOS measure and the NSDUH measure, are strongly correlated. Alpert et al. (2018) show that states with high shares of oxycodone relative to hydrocodone distribution had higher rates of OxyContin misuse and that their estimation results are similar regardless of whether they use the ARCOS measure or the NSDUH measure. In this study, I control for exposure to the reformulation using the ARCOS measure in my main specifications, although I also present the estimates when I instead use the NSDUH measure to account for exposure to the reformulation.[23]

[22]The ARCOS data provide information on the total supply of oxycodone but not the OxyContin supply specifically. However, OxyContin accounted for a large fraction of oxycodone supply in the pre-reformulation period: for example, in 2002, OxyContin accounted for 68% of oxycodone distribution (Paulozzi 2006).

[23]The major limitation of using ARCOS data to measure the differential effects of the reformulation is that this data source provides information on total oxycodone use through legal channels but does not capture nonmedical use specifically. However, the advantage of using this administrative data is that the ARCOS system is not associated with issues of underreporting, which is one of the main limitations of NSDUH survey data. Given the robustness of my results across the ARCOS and NSDUH measures, I consider the former as my preferred measure.

2.3.4 Descriptive Statistics

Table 2.2 provides basic statistics on outcome variables and the state-half-year level covariates for each group before policy implementation. The analysis of this study focuses on the ten states that passed must-access laws between 2010h1 and 2013h2. I present summary statistics for these ten states and for 34 states that had no must-access PDMPs until 2016h2.[24] I treat states with no PDMP and states with a voluntary-access PDMP, but not those with a must-access PDMP, as a single control group because I find no substantial effects of voluntary-access PDMPs on any of the outcome variables I investigate in this paper. Between 2003h1–2009h2, ten treatment states have more oxycodone doses per capita and a higher rate of prescription opioid death. However, the heroin death rate is lower in the treated states, and there are no substantial differences in other outcomes across the two groups. The age composition and average unemployment rates are similar between the groups. However, the ten treatment states reflect a higher share of whites and a smaller share of Hispanics.

2.4 Empirical Strategy

I examine the causal effects of must-access PDMPs on heroin and opioid-related mortality rates by exploiting variation in the start date of the policy. As my main econometric model, I use a difference-in-difference specification that allows the treatment effect to vary over time, often called the event study specification. This model sets each state's first post-period to period zero and compares the outcomes between the treatment and control states in every pre- and post-period, relative to the last pre-period. The main specification is as follows:

$$Y_{st} = \alpha_s + \alpha_t + \sum_{k \neq -1} \beta_k \mathbf{1}(Policy_{sk}) + X_{st}\delta + oxy_s \cdot \omega_t + \varepsilon_{st} \tag{2.1}$$

where Y_{st} is the number of opioid deaths per 100,000 in a given state s over half-year t. $\mathbf{1}(Policy_{sk})$ is 1 if a given state enacted a must-access PDMP k periods ago, and $k \geq 0$ denotes a post-period. A negative k denotes a pre-period, indicating -k periods prior to

[24] Although 35 states did not implement a must-access PDMP until 2016h2, I exclude Florida from my control group in all analyses. Florida is an outlier that experienced both a sharp increase and a decrease in oxycodone supply within a decade. See Appendix Section B.3 for more details.

implementation. I control for state fixed effects α_s to account for time-invariant state-specific characteristics and time (half-year) fixed effects α_s to account for time-varying national shocks and trends in opioid availability, heroin prices, and other common factors across states. X_{st} is a vector of state- and time-varying control variables, which includes the log unemployment rate, share of the population in six age groups (0–14, 15–24, 25–44, 45–64, 65–84, 85+), share non-Hispanic black, share Hispanic, and share other race.[25] I also include a time-varying indicator for whether the state had a pill mill law.

oxy_s denotes the measure of OxyContin use in state s in the pre-reformulation period (2004h1–2009h2). I use the ARCOS measure to account for pre-reformulation OxyContin use, although I also present the results obtained using the NSDUH measure (see Section 3.4 for a description of these measures). I interact this time-invariant and state-specific OxyContin use variable with the half-year fixed effects, ω_t, to account for the differential effects of the 2010 OxyContin reformulation across states. ϵ_{st} is the error term. Standard errors are clustered at the state level.

β_k are the parameters of interest, which summarize the treatment effect of must-access PDMPs on the outcomes, k periods after implementation. The last pre-period ($k = -1$) is used as the reference period. To estimate the medium-run effects of the policies, I consider an event time window that runs from 15 half-years prior to implementation to six half-years post-implementation. My analyses focus on the ten treated states that were consistently observed during this time window.[26] Because states implemented must-access PDMPs with different timing, some of my treated states are not observed in distant relative periods. To make my treated sample balanced in relative periods, I trim all periods outside the -15/+6 window.[27]

The key identifying assumption is that absent must-access PDMPs opioid death rates

[25] I obtain total population, population by age group, and population by race from the census. I assume that population estimates are recorded in the second half of the year and use linear interpolation to estimate population levels for the first half of the year.

[26] In Appendix Section B.6, I address the consequences of this choice in detail by illustrating that the estimated short-run effect is similar if I consider a shorter time window to add more treated states in the analysis. Given this similarity, I prefer the time window that allows me to look at the longer-term effects.

[27] Sun and Abraham (2021) explain that researchers bin or trim distant relative periods to accommodate unbalanced relative periods, but they provide no theoretical advantage to either approach. If I bin distant periods, I find that the policy has a larger effect on heroin mortality in the last post-period, which is primarily driven by one treated state.

would have continued to trend in parallel across treatment and control states. To visually assess whether there are systematic differences in trends in the outcome between groups prior to policy implementation, I plot the β_k coefficients from the baseline specification (equation 3.1). For interpretation, I report β_2, β_4, and β_6, which indicate the one-year effect, two-year effect, and three-year effect, respectively.

2.5 Results

This section comprises three parts. First, I provide evidence that the per capita legal supply of opioids declined following the states' implementation of must-access PDMPs. Second, I show that must-access PDMPs were associated with increased heroin mortality and that the size of effects grew over time. I also present the negative effects of must-access laws on deaths from prescription opioids and discuss the net effects on total opioid-related mortality. Third, I identify heterogeneity in the effects of must-access PDMPs on my outcomes generated by different strengths of must-access laws.

2.5.1 Effects of Must-Access PDMPs on the Legal Supply of Opioids

First, I investigate the effect of must-access PDMPs on the legal supply of opioids. Figure 2.2 plots the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1). The dependent variable is the per capita legal supply of oxycodone (in morphine equivalent doses) in Panel A and the per capita legal supply of hydrocodone (in morphine equivalent doses) in Panel B.

Figure 2.2 suggests a negative association between must-access PDMPs and the per capita legal supply of opioids. Panel A of Figure 2.2 shows that there was a negative trend break in the per capita legal supply of oxycodone in the first post-period, although there was an upward trend in the entire pre-period. As shown in Panel B, I also find suggestive evidence of a negative association between must-access PDMPs and hydrocodone supply, although the coefficients are statistically insignificant.

2.5.2 Effects of Must-Access PDMPs on Heroin Mortality

Trend Break Estimate In this study, I use a difference-in-difference specification that allows the treatment effect to vary over time (see equation 3.1). However, one concern with this specification is how I test and summarize the estimated effects. Unlike a typical difference-in-differences specification that treats the entire pre-period as the reference period, my baseline specification compares outcomes between the treated and control groups in each post-period, relative to the last pre-period. Therefore, the estimated effects can be sensitive to what happened in the last pre-period. To address this, I report trend break estimates, following Finkelstein (2007), in addition to the original coefficients. My trend break estimates provide the results for testing the n-period change in β_k after the reference period relative to the n-period change in β_k prior to the reference period. For example, the two-year effect is calculated using the following equation:

$$\Delta 5 = (\beta_4 - \beta_{-1}) - (\beta_{-1} - \beta_{-6}) = \beta_4 + \beta_{-6} \tag{2.2}$$

where β_{-1} equals to zero because the last pre-period is used as the reference. $\Delta 5$ summarizes the five-half-year change in the outcome after the reference period relative to the five-half-year change prior to the reference period for the treated states, relative to the control states. In Tables 2.3–2.5, I report the one-year, two-year, and three-year outcome changes, respectively ($\Delta 3, \Delta 5$, and $\Delta 7$). My interpretation of the results relies more heavily on trend break estimates (Δn) than on the original regression coefficients (β_k). However, when two statistics are qualitatively similar, I consider the original regression coefficients as my preferred statistics because they allow for a direct comparison of estimates across figures and tables.

Baseline Heroin Estimates In Figure 2.3, Panel A presents the baseline heroin estimates. Panel A shows the effects of must-access PDMPs on heroin mortality by plotting the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) with total heroin deaths per 100,000 as the dependent variable. The panel shows the point estimates and 95 percent confidence intervals. The corresponding regression coefficients and trend break estimates are presented

in columns 4 and 5 of Table 2.3 Panel A, respectively. In Table 2.3, I report the one-year, two-year, and three-year effects of the policy. Standard errors in all specifications are clustered at the state level.

Figure 2.3 Panel A provides strong evidence that must-access PDMPs were associated with an increased heroin death rate. Panel A shows that the trends in heroin mortality in the pre-period did not differ across the treatment and control states, providing evidence in support of the parallel trends assumption. In the first post-period, however, there was a sudden increase in heroin mortality in states with must-access PDMPs, and the size of effects steadily grew over time. The coefficients are positive and statistically significant in every post-period, although the coefficient for event time +1 is statistically insignificant. Moreover, the sudden increase in heroin mortality coincided with a sudden decline in prescription opioid mortality following implementation (see Figure 2.3 Panel C), providing evidence of the substitution of heroin for prescription opioids.[28]

In Table 2.3 Panel A, the baseline estimates for heroin mortality are reported in column 4, and the corresponding trend break estimates are reported in column 5. Because my baseline estimates reveal no pre-trend in heroin mortality (see Figure 2.3 Panel A), the original regression coefficients (β_k) are qualitatively similar to the corresponding trend break estimates (Δn). Given this similarity, I consider the original baseline estimates as my preferred heroin estimates. Column 4 indicates that a year after implementation, heroin mortality in a half-year period increased by 0.42, and the size of effects grew larger over time. Two years after implementation, having a must-access PDMP was associated with 0.9 more heroin deaths per 100,000 in a half-year period compared with states without the policy. The largest effect occurred in the last post-period, for which I estimate an effect of 1.13.[29]

Consequences of Adding Controls I examine the consequences of controlling for

[28]The results for prescription opioid mortality are provided in section 2.5.4.

[29]Given that the population-weighted mean of the ten treated states' heroin-related death rates (Ruhm-corrected number of deaths per 100,000 in a half-year period) in 2015h2 was 4.1, must-access PDMPs had a substantial impact on heroin deaths in these states. The weighted mean of heroin death rates among the ten treated states increased from 2 in 2012h2 to 4.1 in 2015h2, while that among the 34 control states increased from 1.14 to 2 during the same period.

state- and time-varying covariates and co-occurring opioid-related policies. Panels A–D of Figure 2.4 plot estimates from the baseline difference-in-differences specification (equation 3.1) with different sets of controls and with heroin mortality as the dependent variable. In Figure 2.4 Panel A, I include only the fixed effects for state and time and the indicators for pre- and post-periods (the corresponding regression coefficients are presented in column 1 of Table 2.3 Panel A). I gradually add more controls in subsequent panels to arrive at my baseline estimates presented in Panel D.[30] Using the simple specification in Figure 2.4 Panel A, I find that the coefficients are positive and statistically significant in all the post-periods, though the coefficient for event time +1 is statistically insignificant. However, there is evidence of an upward trend in heroin mortality in the last few pre-periods when both the covariates and the OxyContin reformulation are not controlled for. This pre-trend may reflect the confounding effects of the reformulation, which caused a transition from nonmedical use of prescription opioids to heroin use prior to most of the treatment states' implementation of must-access PDMPs. The shaded area in Panel A indicates the time of the reformulation, which was introduced in 2010h2. The number of treated states at the time of the reformulation in each event time period is presented in the parentheses below that period.

Panel B of Figure 2.4 presents the estimates when the state- and time-varying covariates and an indicator for whether a state had a pill mill law are included in the regression (the corresponding estimates are presented in column 2 of Table 2.3 Panel A).[31] The estimates for the post-periods are largely unaffected by adding these controls, but almost all the coefficients for the pre-periods become statistically insignificant. However, I still observe suggestive evidence of a pre-trend in heroin mortality when the reformulation is not controlled for.

In Panels C and D, I additionally control for the confounding effect of the 2010 reformulation by adding a measure of pre-reformulation OxyContin use interacted with the

[30]The baseline estimates presented in Figure 2.4 Panel D are identical to those in Figure 2.3 Panel A.

[31]Pill mill laws, which impose regulations on pain clinics to prevent them from issuing opioid prescriptions without medical indication, were enacted in three treated states around the time of policy implementation. I view pill mill laws as complements to must-access laws, as Buchmueller and Carey (2018) do. Appendix Figure B.20 suggests that, in the absence of a must-access law, a pill mill law had no independent effect on my outcomes. See Appendix Section B.4 for more details.

half-year fixed effects to the regression. As described in Section 3.4, I use the two alternative measures of OxyContin use: in Panel C, I interact the NSDUH measure with the half-year fixed effects (the corresponding regression coefficients are presented in column 3 of Table 2.3 Panel A), and in Panel D, I interact the ARCOS measure with the half-year fixed effects (the corresponding regression coefficients are presented in column 4 of Table 2.3 Panel A). I obtain similar results across these two measures; compared with the estimates in Panel B, I see in Panels C and D that the size of the estimated effect on heroin mortality slightly decreases in every post-period. Importantly, as noted above, I observe no evidence of a pre-trend when accounting for the reformulation. These findings suggest that controlling for the reformulation allows for a transparent identification of the impact of must-access PDMPs by accounting for the pre-existing correlation between the implementation of must-access PDMPs and exposure to the reformulation.[32]

To summarize, I find a strong and positive association between must-access PDMPs and heroin death rates.[33] Heroin mortality began to increase in the first post-period, and the size of the effects grew over time. The results patterns are similar across different specifications, but I observe an upward pre-trend in heroin mortality when I do not control for the reformulation. I find that two years after implementation, the treated states had 0.9 additional heroin deaths per 100,000 in a half-year period relative to the control states.

Heroin-Only Mortality I examine the more exclusive measure of heroin deaths, which I refer to as heroin-only mortality. The heroin-only mortality indicates the deaths that involved heroin (T40.1) and no other types of opioids (T40.2–4) at the time of death. Column 1 of Table 2.4 reports the trend break estimates (equation 2.2) from the baseline specification (equation 3.1) with heroin-only mortality as the dependent variable. As seen in Column 1, all the trend break estimates are statistically insignificant and much smaller than those obtained using total heroin mortality as the dependent variable (see column 5 of Table 2.3 Panel A). Most heroin is now laced with illicit fentanyl, which may explain why the

[32]For all the outcome variables, I find similar results regardless of whether I use the ARCOS measure or the NSDUH measure. I present the mortality results that I obtain using the NSDUH measure in columns 3 of Tables 2.3 and 2.5 (corresponding regression coefficients are displayed in Appendix Figure B.3).

[33]The findings in this paper complement a contemporaneous working paper by Mallatt (2019), which finds that must-access PDMPs increase heroin crime rates within opioid-dense counties during my sample period.

policy had little effect on heroin-only mortality while having a strong effect on total heroin mortality. This motivates me to investigate the combined deaths from heroin and synthetic opioids, which I use as my measure of illegal opioid mortality.

2.5.3 Effects of Must-Access PDMPs on Illegal Opioid Mortality

I examine how must-access PDMPs affected deaths from illegal opioids, such as heroin and illicit fentanyl. Figure 2.3 Panel B plots the coefficients on the indicators for pre- and post-periods from the main difference-in-differences specification (equation 3.1) with the combined deaths from heroin and synthetic opioids per 100,000 as the dependent variable. The plot shows the point estimates and 95 percent confidence intervals. The corresponding regression coefficients and trend break estimates are presented in columns 4 and 5 of Table 2.3 Panel B, respectively. Columns 1–4 of Table 2.3 Panel B report the original regression coefficients from the baseline specification (equation 3.1) with different sets of controls. In Table 2.3, controls are identical to those in Table 2.3 Panel A. Standard errors in all specifications are clustered at the state level.

As seen in Figure 2.3 Panel B and in columns 4 and 5 of Table 2.3 Panel B, illegal opioid mortality began to increase in the year of implementation, and the size of effects grew over time, although the one-year effect is statistically indistinguishable from zero. Compared to the estimates for heroin mortality (see Figure 2.3 Panel A and columns 4 and 5 of Table 2.3 Panel A), the magnitude of the policy impact on illegal opioid mortality is larger in every post-period, and the effects grew much faster over time.[34]

Illegal-Opioid-Only Mortality Now, I examine the more exclusive measure of illegal opioid mortality. In Figure 2.5, Panel A displays the coefficients from the baseline specification (equation 3.1) with illegal-opioid-only deaths per 100,000 as the dependent variable. The corresponding trend break estimates are reported in column 2 of Table 2.4. The results pattern in Figure 2.5 Panel A is similar to that in Figure 2.3 Panel B, in which total illegal opioid mortality is the outcome variable. These findings suggest that must-access PDMPs

[34]Some caution in the interpretation of my illegal opioid results is needed. There is a limitation in assessing whether there was a pre-trend in illegal opioid mortality because the surge in synthetic opioid deaths began in the states' post-period. Although I find no evidence of a pre-trend in illegal opioid mortality, it may not fully support the parallel trend assumption.

increased illegal opioid mortality not attributable to prescription opioids.

2.5.4 Effects of Must-Access PDMPs on Prescription Opioid Mortality

Figure 2.3 Panel C investigates the impact on prescription opioid mortality by plotting the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) with total prescription opioid deaths per 100,000 as the dependent variable. The plot displays the point estimates and 95 percent confidence intervals. The corresponding regression coefficients and trend break estimates are presented in columns 4 and 5 of Table 2.5 Panel A, respectively. Columns 1–4 of Table 2.5 Panel A report the original regression coefficients from the baseline specification (equation 3.1) with different sets of controls. In Table 2.5, controls are identical to those in Table 2.3. Standard errors in all specifications are clustered at the state level.

In Figure 2.3 Panel C, I see that there was an upward trend in prescription opioid mortality in the last few pre-periods, but following implementation, prescription opioid mortality began to decrease. Although this pre-trend suggests that the trends in prescription opioid mortality were different across the treated and control groups prior to implementation, the negative trend break in the first post-period provides suggestive evidence of a negative association between must-access PDMPs and prescription opioid mortality.

Figure 2.3 Panel C also suggests that the negative effect of the policy on prescription opioid mortality was temporary: the magnitude of effects faded out over time. In Table 5 Panel A, the estimated one-year effects are statistically different from zero in all columns. The trend break estimates in column 5 indicate that a year after policy implementation (or three half-years after the reference period), must-access PDMPs were associated with 1.1 less prescription opioid mortality relative to the three-half-year change prior to the reference period.[35] In contrast, two-year and three-year effects are statistically insignificant in all columns. One possible explanation for the temporary negative effects of must-access PDMPs on total prescription opioid mortality is that people may gradually substitute illegal

[35]The magnitude of the trend break estimates reported in column 5 is larger than that of the original regression coefficients in column 4, because of the upward pre-trend in prescription opioid mortality (see Figure 2.3 Panel C).

opioids for prescription opioids, and deaths involving both prescription and illegal opioids increase over time. I investigate the more exclusive measure of prescription opioid mortality and find evidence supporting this possibility, as presented below.

Prescription-Opioid-Only Mortality In Figure 2.5 Panel B, the dependent variable is deaths caused by prescription opioids but not also heroin, methadone, or synthetic opioids, which I refer to as prescription-opioid-only mortality. The corresponding trend break estimates are reported in column 3 of Table 2.4. Figure 2.5 Panel B shows that the negative effects of must-access PDMPs on prescription-only mortality are more persistent compared with the effects on total prescription opioid mortality (see Figure 2.3 Panel C), although coefficients for the later periods are still statistically insignificant. The more persistent effects on prescription-opioid-only mortality, combined with the temporary effects on total prescription opioid mortality, support the possibility that existing users gradually switched to illegal opioids. In sum, the results for prescription opioid deaths provide another important piece of evidence of a transition from prescription opioids to illegal opioids.

2.5.5 Net Effects of Must-Access PDMPs on Total Opioid-Related Mortality

Finally, I examine the net effects of must-access PDMPs on total deaths from any opioid, including prescription opioids, heroin, and synthetic opioids. In this paper, I provide evidence that must-access PDMPs increased illegal opioid mortality, but I also find the negative effects on prescription opioid mortality. These offsetting effects are more clearly observed in Figure 2.5. Estimating the net effects of must-access PDMPs on the total opioid-related mortality is particularly important because the total opioid death rate is the target at which drug policies are ultimately aimed.

Figure 2.3 Panel D plots the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) with total opioid-related mortality as the dependent variable. The plot displays the point estimates and 95 percent confidence intervals. The corresponding regression coefficients and trend break estimates are presented in columns 4 and 5 of Table 2.5 Panel B, respectively. Columns 1–4 of Table 2.5

Panel B report the original regression coefficients from the baseline specification (equation 3.1) with different sets of controls. Controls are identical to those in Table 2.3. Standard errors in all specifications are clustered at the state level.

Figure 2.3 Panel B shows that the size of the policy impacts on total opioid mortality was close to zero in the first four post-periods and then increased over time. In Table 2.5 Panel B, I observe that the estimated three-year effects are positive and statistically significant in most columns, although the size of the three-year effects are not stable across specifications. In summary, I find that must-access PDMPs had no substantial effect on total opioid-related mortality in the short term because of the offsetting effects; however, in the longer term, the policies were positively associated with total opioid mortality because the increase in illegal opioid mortality outweighed the decrease in prescription opioid mortality.

2.5.6 Heterogeneous Treatment Effects

In my main results discussed above, I do not consider different strengths of must-access laws among the ten treatment states, although they vary greatly among states. To investigate heterogeneity in the effects of must-access laws across different strengths, I divide the ten states' must-access laws into three types: limited laws, discretionary laws, and broad laws.[36] Limited laws are defined as those that apply only to limited ingredients (the initial PDMP in Oklahoma) or laws that require access under limited circumstances (the initial PDMP in Vermont); discretionary laws are those that rely on provider suspicion of abuse (Delaware and the initial PDMP in Ohio); and broad laws are those that apply to all clinic settings and ingredients and require provider access, even without suspicion of abuse.[37] One challenge in my heterogeneity analysis is that the three treatment states strengthened their laws from limited to broad in 2015. Because it is difficult to distinguish between the long-run effect of the initial laws and the immediate effect of the strengthened laws, my heterogeneity analysis focuses on the states' initial must-access laws. The fact that the three treated states strengthened their laws at least two years after the implementation of the initial must-access laws allows me to look at the two-year effect of the initial laws.

[36]This categorization is proposed by Buchmueller and Carey (2018).

[37]Kentucky, Massachusetts, New Mexico, New York, Tennessee, West Virginia, and the recent laws in Ohio, Oklahoma, and Vermont are coded as having a broad law.

Figure 2.6 presents the trend break estimates summarizing the two-year effect that I obtain when I interact the indicators for all the seven post-periods from the baseline specification (equation 3.1) with indicators for three law types in a single regression.[38][39] The red (horizontal) dashed line shows the overall effect among the ten treated states, indicating the two-year trend break estimate ($\Delta 5$) obtained from the baseline specification (see equations 3.1 and 2.2).

Figure 2.6 suggests that broad laws and discretionary laws had stronger effects than limited policies. For most of my outcomes, limited laws had little or no effect, while the other two types of laws were positively associated with heroin mortality (Panel A) and illegal opioid mortality (Panel B), and negatively associated with prescription opioid mortality (Panel C) and oxycodone doses per capita (Panel E). The estimates indicate that the net effect of must-access laws is close to zero, regardless of the strength of the must-access law (Panel D). Interestingly, discretionary laws had even stronger effects than broader laws for some of the outcomes, which is largely driven by Ohio's policy. In Appendix Section B.4, I discuss in detail why Ohio's initial must-access policy, which relied on provider suspicion, had strong impacts.[40] In sum, my heterogeneity analysis suggests that broad and discretionary laws had stronger effects than limited laws, which had little effects on my outcomes, and that discretionary laws had even stronger impacts on some outcomes than broad laws.[41] In addition, in Appendix Figures B.7 and B.8, I present event studies

[38] More specifically, the trend break estimates (summarizing the two-year effects) for each law type presented in Figure 2.6 are calculated as follows: $\Delta 5_j = (\beta_{4j} * \mathbf{1}(\text{Law Type j}) - \beta_{-1}) - (\beta_{-1} - \beta_{-6})$.

[39] I interact law types with the indicators for the post-periods instead of the full set of indicators for pre- and post-period, because each of the limited and discretionary subgroups consists of only two states, and thus the pre-trends in outcomes are noisy for these subgroups (see Panels A.2 and B.2 of Appendix Figure B.7).

[40] I propose three possible explanations for the strong impact of Ohio's initial must-access law—a sharp increase in PDMP utilization, the existence of a complementary law, and high accessibility of heroin. See Appendix Section B.4 for more detail.

[41] My findings are consistent with several of Buchmueller and Carey's (2018) findings, which provide mixed results on heterogeneous effects by subgroups: for their quantity-based outcomes, they find that broad laws had similar but a slightly larger size of effects than discretionary laws and that limited policies had little effect; however, for the other outcomes, their results suggest no clear pattern; for example, they show that discretionary laws had stronger effects than broader policies for some of their shopping outcomes, and in most other cases, the estimate for each law type cannot be distinguished from the overall estimate.

separated by subgroups that I obtain when I limit the treated states to each subgroup; the results shown in these figures are consistent with those presented in Figure 2.6.[42]

2.6 Robustness Tests

In Table 2.6, I test the robustness of heroin results to a number of alternative explanations for the association between must-access PDMPs and increased heroin mortality (the corresponding regression coefficients are displayed in Panel A of Appendix Figure B.10). In column 1 of Table 2.6, I repeat my baseline estimates for heroin mortality (from column 4 of Table 2.3) to test the sensitivity to specification. Standard errors in all columns are clustered at the state level. In column 2, I use the raw reported mortality rate as the dependent variable instead of the Ruhm-corrected mortality rate. I estimate a slightly larger effect when I use raw reported mortality, but the results are qualitatively similar.[43][44] In column 3, I control for whether a state had a voluntary-access PDMP and find similar

[42]Note that in Appendix Figures B.7 and B.8, I use a shorter event time window (-15/+4) than that used in the baseline analysis (-15/+6), to focus on the states' initial must-access laws. The distant periods outside the -15/+4 window are trimmed. Although the mortality results for the discretionary and limited subgroups, each of which includes only two states, are noisy, Appendix Figures B.7 and B.8 provide further evidence of heterogeneity across different types of must-access laws.

[43]Appendix Figure B.9 displays the estimates from the baseline specification (equation 3.1) with the reported mortality rates as the dependent variable.

[44]However, note that the mean values are different across the two measures. The Ruhm (2018) correction suggests how to correct the problem of underreporting of overdose deaths. By construction, raw reported mortality rates have a smaller sample mean and standard deviation than Ruhm-corrected mortality rates. Given the smaller standard deviation of the reported mortality rates, the fact that I find the larger effect on reported heroin mortality than on Ruhm-corrected heroin mortality (see columns 1 and 2 of Table 2.6) supports the possibility that using reported mortality may overstate the policy effect on heroin death rates.

results.[45][46] In column 4, I include Florida in the analysis sample, and the estimates are similar.[47] In columns 5–7, I include several other co-occurring opioid-related policies one by one to test for the sensitivity of my baseline estimates to adding each variable.[48] In column 5, I include a time-varying indicator for whether the state had medical marijuana laws (MMLs) as well as an indicator for whether the state had legal and operational dispensaries and find similar results, although the one-year effect becomes insignificant. In column 6, I add an indicator for whether the state had naloxone access laws (NALs), and in column 7, I add an indicator for whether the state had Good Samaritan overdose prevention laws. Columns 5–7 suggest that my estimates are robust to including several other co-occurring state opioid-related policies. Although not reported, I also obtain similar and statistically significant coefficients when I include these co-occurring policies in a single regression. In column 8, I use alternative start dates of must-access laws taken from Sacks et al. (2019) and find similar effects of must-access PDMPs on heroin mortality, though the one-year effect becomes statistically insignificant with these dates.[49] Appendix Table B.4 conducts the same robustness tests as those in Table 2.6 for the other mortality outcomes, and I find similar results. Finally, Appendix Table B.3 shows the sensitivity of my heroin estimates to dropping one treated state (the corresponding regression coefficients are displayed in Panel

[45]In Appendix Table B.2, I conduct the same test using the following alternative start dates of voluntary-access PDMPs: enactment dates from the PDAPS and NAMSDL, and the modern system user access dates suggested by Horwitz et al. (2018). My estimates are stable across different dates.

[46]I find no substantial effect of voluntary-access PDMPs on the outcome variables investigated in this paper using any source of start dates. First, the size of the coefficients on the indicator for voluntary-access PDMPs is small compared with that for must-access PDMPs. Second, although the coefficients on voluntary-access PDMPs reported in Appendix Table B.2 are statistically significant, they become insignificant when I bin distant relative periods rather than trim them.

[47]As mentioned in Section 2.4, my oxycodone estimates are sensitive to whether I include Florida in the sample. In Appendix Section B.3, I discuss this sensitivity in detail. Although not reported, my oxycodone results are robust to removing one state when Florida is excluded from the control group.

[48]Data on these policies are derived from PDAPS.

[49]In column 8, the treated states are the eight that implemented must-access PDMPs between 2010h2 and 2013h2, which are consistently observed from event time -15 to +6; the control states are the 31 that did not implement must-access policies until 2016h2, excluding Florida (see the third column of Table 2.1 for the start dates). Therefore, the difference between the estimates is driven by not only the inconsistency in start dates but also the differential policy effects across states.

A of Appendix Figure B.11); Panels B–D of Appendix Figure B.11 show the results for the same tests for the other mortality outcomes. I find that my baseline estimates for the mortality outcomes are robust to removing one treated state. My heroin mortality estimates are statistically significant regardless of which treated state is dropped, but when I drop Ohio, the magnitudes of the estimates are slightly attenuated.[50]

2.7 Conclusion

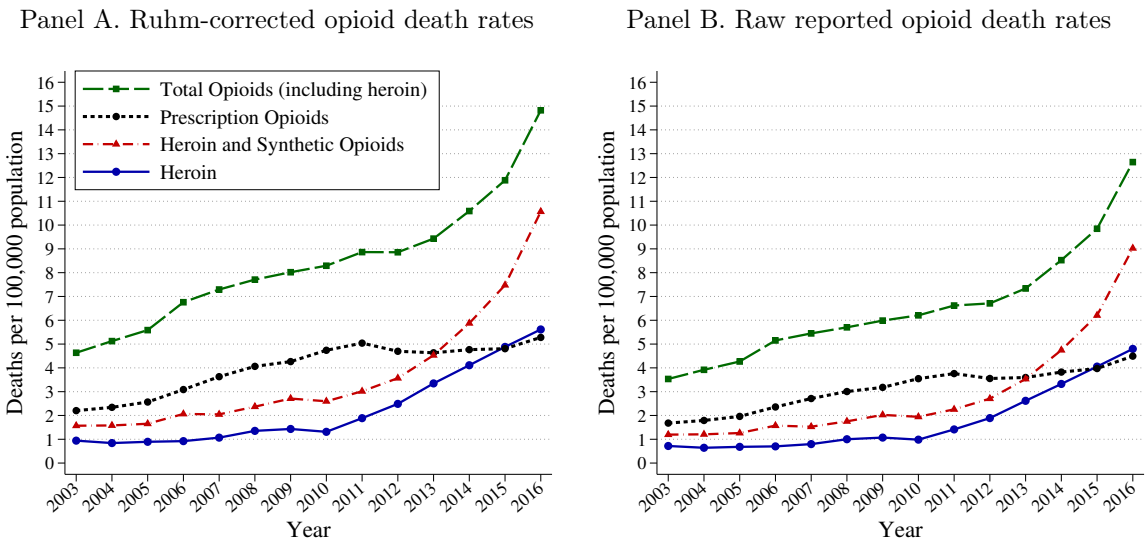
I examine the spillover effects of must-access PDMPs on illegal opioid use. Using a difference-in-differences approach, I find strong evidence that must-access PDMPs have had the unintended consequences of increased heroin use, which has not been reported in most prior studies. This increase began in the year of policy implementation, and the effects grew over time. Two years following implementation, having a must-access PDMP was associated with 0.9 additional heroin deaths per 100,000 in a half-year period compared with control states. I also find that the increase in deaths from heroin coincided with a sudden decrease in prescription opioid mortality following implementation. The estimates from this paper suggest that the negative effects of must-access laws on prescription opioid mortality were offset by the positive effects on illegal opioid mortality. Overall, I show that must-access PDMPs had no effect on total opioid-related mortality in the short term because of the offsetting effects, but in the longer term, the unintended effect on illegal opioid mortality surpassed the intended effect on prescription opioid mortality.

The findings of this study suggest that focusing on supply-side policies that limit access to legal drugs may simply cause users to shift to using close substitutes. Given the existence of accessible and affordable substitutes for prescription opioids, more robust policies are needed to address the opioid overdose epidemic. Demand-side interventions, such as medication treatment of opioid dependence (e.g., opioid substitution therapies), may be more effective at reducing overall opioid abuse. However, in the long run, must-access PDMPs may have different net effects because they can also reduce initiation into

[50] In Appendix Section B.4, I show that Ohio's must-access PDMP had stronger effects on my outcomes than the other states' policies and I propose three possible explanations for the strong effects of Ohio's must-access policy.

prescription opioids. Although must-access PDMPs target existing users, Sacks et al. (2019) suggests that they are in fact effective at reducing opioid initiation. I investigate the medium-run effects of must-access laws in the first three to six years and find that must-access PDMPs have led to worse outcomes. The long-run impacts of these policies will be determined by many factors, such as the composition of new and existing users, the magnitudes of policy effects on those users, accessibility to substitutes, and accessibility to medication treatment and prevention.

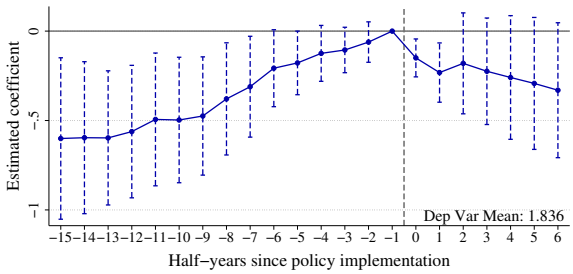
Figure 2.1: National Trends in Ruhm-Corrected and Reported Death Rates



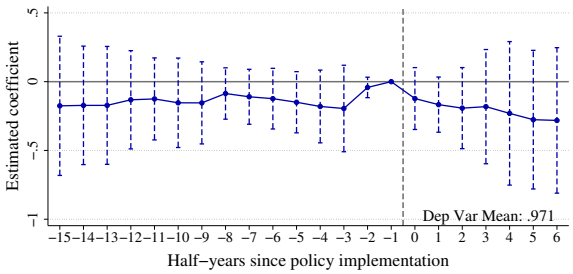
Notes: The figure plots the national trends in Ruhm-corrected and reported numbers of deaths per 100,000 population calculated using mortality data from the National Vital Statistics System (NVSS). Drug overdose deaths are coded using ICD-10 underlying cause of death codes: X40–X44, X60–X64, X85, and Y10–Y14. To identify drug involvement, the following four drug identification codes are used: heroin (T40.1), natural and semisynthetic opioids such as oxycodone and hydrocodone (T40.2), methadone (T40.3), and synthetic opioids excluding methadone, such as fentanyl (T40.4). I calculate total deaths from any opioid, including heroin, by combining T40.1–T40.4. Prescription opioid deaths are identified using T40.2. The deaths from heroin and synthetic opioids combine T40.1 and T40.4. Heroin deaths are identified using T40.1. Reported mortality rates are based on mentions of the specified drugs on the death certificates. Corrected mortality rates are estimated by using the method suggested by Ruhm (2018), which uses information from death certificates that specified at least one drug category to impute drug involvement for cases in which none was specified.

Figure 2.2: Effects of Must-Access PDMPs on the Legal Supply of Opioids

Panel A. Oxycodone doses per capita

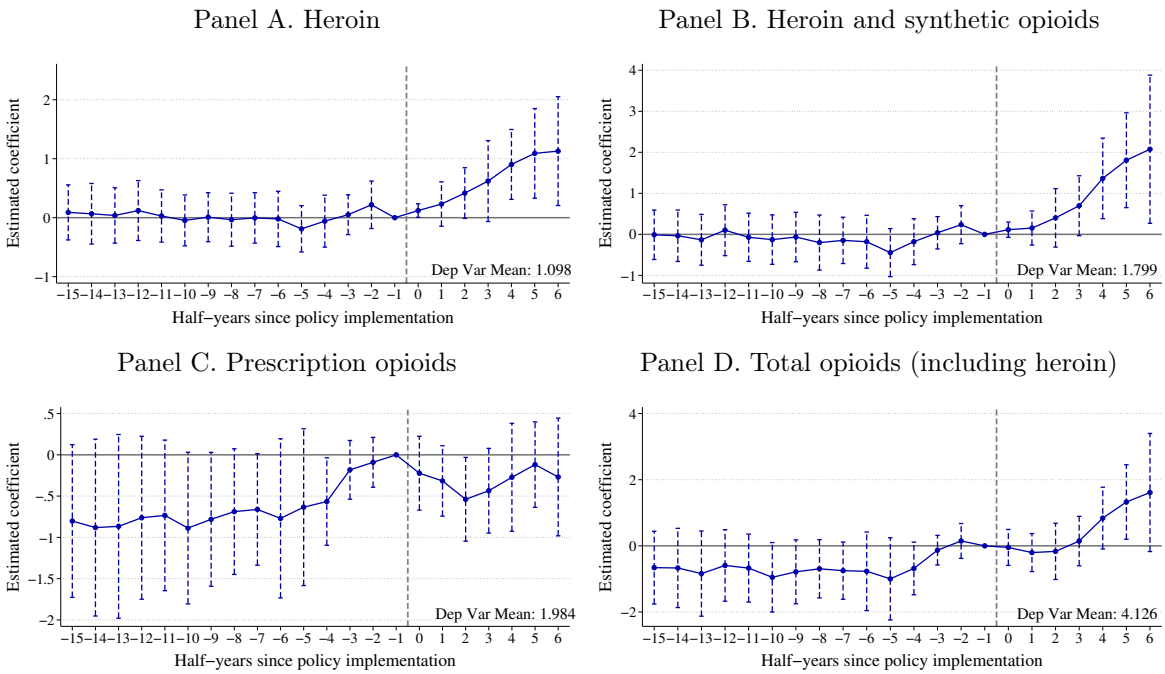


Panel B. Hydrocodone doses per capita



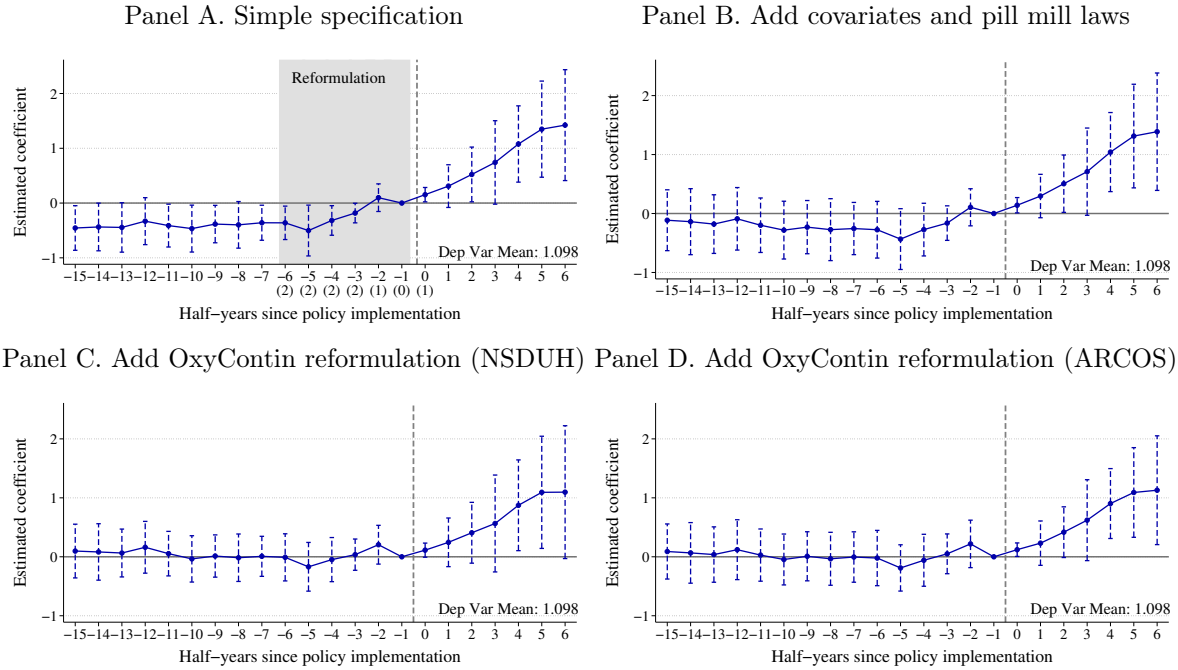
Notes: The figure displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1). The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. Observations are weighted by state population. Outcome variables are the per capita legal supply of oxycodone (Panel A) and hydrocodone (Panel B) in morphine equivalent doses. In all panels, the treated states are the 10 that implemented must-access PDMPs from 2010h2 to 2013h2, and the treated sample is balanced in relative periods from -15 to +6. The distant relative periods outside the -15/+6 event time window are trimmed. The control states are the 34 that did not implement must-access policies until 2016h2, and the control sample is balanced from 2003h1 to 2016h2. Florida is excluded from the control sample in all panels (see Appendix Section B.3). In all panels, I control for fixed effects for state and half-year, an indicator for whether a state had a pill mill law, the ARCOS measure of pre-reformulation OxyContin use interacted with the half-year fixed effects, and the following time-varying covariates: the log unemployment rate, share of the population in six age groups (0–14, 15–24, 25–44, 45–64, 65–84, 85+), share non-Hispanic black, share Hispanic, and share other race.

Figure 2.3: Effects of Must-Access PDMPs on Opioid Mortality



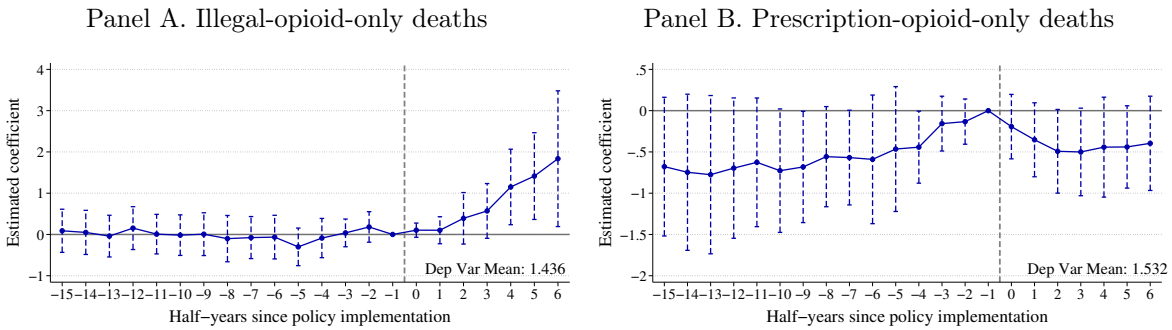
Notes: The figure displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1). The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. Observations are weighted by state population. In Panel A, the dependent variable is heroin deaths per 100,000 (drug code T40.1). In Panel B, the dependent variable is the combined deaths from heroin and synthetic opioids per 100,000 (drug codes T40.1, T40.4). In Panel C, the dependent variable is prescription opioid deaths per 100,000 (drug code T40.2). In Panel D, the dependent variable is total deaths from any opioid, including heroin (drug codes T40.1–T40.4). In all panels, the Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. In all panels, the treated states are the 10 that implemented must-access PDMPs from 2010h2 to 2013h2, and the treated sample is balanced in relative periods from -15 to +6. The distant relative periods outside the -15/+6 event time window are trimmed. The control states are the 34 control states that did not implement must-access policies until 2016h2, and the control sample is balanced from 2003h1 to 2016h2. Florida is excluded from the control sample in all panels (see Appendix Section B.3). In all panels, I control for fixed effects for state and half-year, an indicator for whether a state had a pill mill law, the ARCOS measure of pre-reformulation OxyContin use interacted with time dummies, and the following time-varying covariates: the log unemployment rate, share of the population in six age groups (0–14, 15–24, 25–44, 45–64, 65–84, 85+), share non-Hispanic black, share Hispanic, and share other race.

Figure 2.4: Sensitivity of Heroin Estimates to Adding Controls



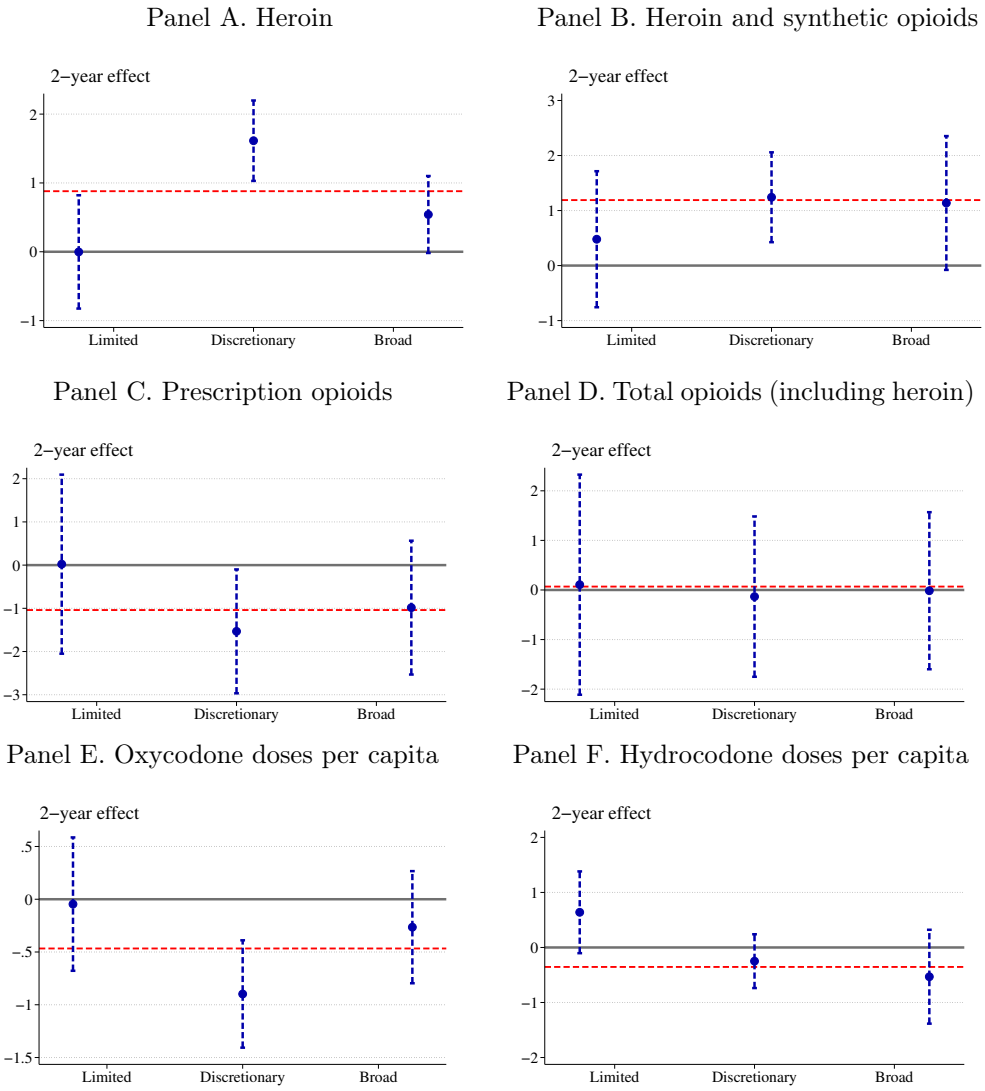
Notes: The figure shows how adding controls affects the heroin estimates by plotting the coefficients on indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) with different sets of controls. The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. In all panels, the dependent variable is the Ruhm-corrected numbers of heroin deaths per 100,000 (drug code T40.1), which are calculated using data from the National Vital Statistics System (NVSS). In Panel A, I only include the fixed effects for state and half-year and the indicators for pre- and post periods. The gray shaded area in Panel A indicates the time of the reformulation, which was introduced in 2010h2. The number of treated states at the time of the reformulation in each event time period is presented in the parentheses below that period. In Panel B, I add an indicator for whether a state had a pill mill law and the following time-varying covariates: the log unemployment rate, share of the population in six age groups (0–14, 15–24, 25–44, 45–64, 65–84, 85+), share non-Hispanic black, share Hispanic, and share other race. In Panels C and D, I add a measure of pre-reformulation OxyContin use interacted with time fixed effects. I use the two alternative measures of pre-reformulation OxyContin use: the NSDUH measure (Panel C, see Section 3.4) and the ARCOS measure (Panel D, see Section 3.4). The estimates presented in Panel D are identical to those in Figure 2.3 Panel A. Observations are weighted by state population. The treated states are the 10 that implemented must-access PDMPs from 2010h2 to 2013h2, and the treated sample is balanced in relative periods from -15 to +6. The distant relative periods outside the -15/+6 event time window are trimmed. The control states are the 34 control states that did not implement must-access policies until 2016h2, and the control sample is balanced from 2003h1 to 2016h2. Florida is excluded from the control sample in all panels (see Appendix Section B.3).

Figure 2.5: Effects of Must-Access PDMPs on the Exclusive Measures of Opioid Deaths



Notes: The figure displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1). The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. Observations are weighted by state population. In Panel A, the dependent variable is illegal-opioid-only deaths per 100,000, which involved drug code T40.1 or T40.4 but not T40.2 or T40.3 at the time of death. In Panel B, the dependent variable is prescription-opioid-only deaths, which involved drug code T40.2, but not T40.1, T40.3 or T40.4. In all panels, Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. In all panels, the analysis sample and controls are identical to those in Figure 2.3. Fixed effects for state and half-year are always included.

Figure 2.6: Heterogeneous Treatment Effects—Three Types of Must-Access Laws



Notes: The figure shows the heterogeneous treatment effects across my three types of must-access laws: (i) limited laws that apply to certain ingredients or require access under limited circumstances (2 states), (ii) discretionary laws that rely on provider suspicion of abuse (2 states), and (iii) broad laws without such restrictions (6 states). Each panel presents the estimates obtained when I interact the indicators for all the post-periods (from event time 0 to +6) from the baseline specification (equation 3.1) with three indicators for limited, discretionary and broad laws. The (horizontal) dashed red line presents the overall estimate, for reference. The dependent variable is heroin deaths per 100,000 (drug code T40.1) in Panel A, illegal opioid death rate (T40.1, T40.4) in Panel B, prescription opioid death rate (T40.2) in panel C, total opioid-related death rate (T40.1–T40.4) in Panel D, oxycodone (morphine equivalent) doses per capita in Panel E, and hydrocodone (morphine equivalent) doses per capita in Panel F. For all the outcomes, I report the trend break estimates summarizing the two-year effect ($\Delta 5 = (\beta_4 * \mathbf{1}(\text{Law Type}) - \beta_{-1}) - (\beta_{-1} - \beta_{-6})$). I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. In all panels, Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. Observations are weighted by state population. In all panels, the analysis sample and controls are identical to those in Figure 2.3. Fixed effects for state and half-year are always included.

Table 2.1: State Laws

State	Any PDMP	Must-Access PDMP		Pill Mill Laws
		Start Date	Alternative	
Alabama	2005m11			
Alaska	2008m9			
Arizona	2007m9			
Arkansas	2013m3			
California	Pre-1990			
Colorado	2005m6			
Connecticut	2006m10	2015m10	2015m10	
Delaware	2011m9	2012m3	2012m3	
District of Columbia	2014m2			
Florida	2010m12			2011m7
Georgia	2011m7		2014m7	
Hawaii	Pre-1990			
Idaho	Pre-1990			
Illinois	Pre-1990			
Indiana	Pre-1990		2014m7	
Iowa	2006m5			
Kansas	2008m7			
Kentucky	1998m7	2012m7	2012m7	2011m7
Louisiana	2006m7	2014m8	2008m1	2005m7
Maine	2004m1			
Maryland	2011m10			
Massachusetts	1992m12	2013m6	2014m7	
Michigan	Pre-1990			
Minnesota	2009m1			
Mississippi	2006m6			2011m9
Missouri				
Montana	2011m7			
Nebraska	2011m8			
Nevada	1996m1	2007m10	2007m10	
New Hampshire	2012m6		2016m1	
New Jersey	2009m8	2015m7	2015m11	
New Mexico	2004m7	2012m10	2012m9	
New York	Pre-1990	2013m9	2013m8	
North Carolina	2006m1			
North Dakota	2006m12			
Ohio	2005m5	2011m11	2012m3	2011m5
Oklahoma	1991m1	2010m11	2011m3	
Oregon	2009m7			
Pennsylvania	Pre-1990			
Rhode Island	Pre-1990	2014m7	2016m6	
South Carolina	2006m6			
South Dakota	2010m3			
Tennessee	2003m1	2013m1	2013m7	2012m1
Texas	Pre-1990			2009m6
Utah	1995m7			
Vermont	2008m6	2013m11	2015m5	
Virginia	2003m9	2015m7	2015m7	
Washington	2011m8			
West Virginia	1995m6	2012m6	2012m6	2014m9
Wisconsin	2010m6			
Wyoming	2003m7			

Notes: The table reports the start dates of state laws enacted until December 31, 2016. Each column reports the dates obtained from a separate source.

Table 2.2: Summary Statistics, 2003h1–2009h2

Outcome (mean, 2003h1–2009h2)	All 44 states	States with must-access PDMPs (10 states)	States having no must-access PDMPs	Source
Per capita legal supply of opioids (morphine-equivalent doses)				
Oxycodone	1.474	1.862	1.362	ARCOS
	(0.767)	(0.758)	(0.733)	
Hydrocodone	0.849	0.892	0.837	ARCOS
	(0.427)	(0.662)	(0.331)	
Ruhm-corrected overdose deaths per 100,000				
Heroin	0.509	0.466	0.521	Vital Statistics
	(0.324)	(0.36)	(0.312)	
Heroin and synthetic opioids	0.979	1.012	0.97	Vital Statistics
	(0.471)	(0.583)	(0.434)	
Prescription opioids	1.545	1.708	1.498	Vital Statistics
	(0.806)	(1.215)	(0.636)	
Total opioids (including heroin)	3.131	3.411	3.05	Vital Statistics
	(1.353)	(1.925)	(1.126)	
Measures of pre-reformulation OxyContin use				
Oxycodone / (oxycodone + hydrocodone)	0.61	0.69	0.59	ARCOS, 2004h1–2009h2
	(0.16)	(0.12)	(0.16)	
OxyContin misuse rate (%)	0.55	0.71	0.5	NSDUH, 2004–2009
	(0.23)	(0.19)	(0.22)	
Population (%)				
0–14	0.21	0.19	0.21	Census
15–24	0.14	0.14	0.15	Census
25–44	0.28	0.28	0.28	Census
45–64	0.25	0.26	0.25	Census
65–84	0.11	0.11	0.1	Census
85+	0.02	0.02	0.02	Census
Race/ethnicity (%)				
Non-Hispanic white	0.67	0.73	0.65	Census
Non-Hispanic black	0.12	0.11	0.12	Census
Hispanic	0.15	0.1	0.16	Census
Other race	0.07	0.06	0.07	Census
Unemployment rate (%)	5.99	5.88	6.02	BLS
Observations	616	140	476	
Number of states	44	10	34	

Notes: Each column describes the balanced panel of state-half-year from 2003h1 to 2009h2. Observations are weighted by state population, and standard deviations are in parentheses. The first column includes all the 44 states included in the analysis sample. The second column includes the ten treated states that implemented must-access PDMPs from 2010h2 to 2013h2. The last column includes the 34 control states that did not implement must-access policies until 2016h2. Florida is excluded from the control sample (see Appendix Section B.3). I combine states having no PDMP and states having a voluntary-access PDMP but not a must-access PDMP into a single control group.

Table 2.3: Effects of Must-Access PDMPs on Heroin Death Rates and Illegal Opioid Death Rates

	Original Estimates (β_k)				Trend Break Estimates (Δn)
	(1)	(2)	(3)	(4)	(5)
Panel A. Heroin deaths per 100,000 population (T40.1)					
1-year effect (β_2 or $\Delta 3$)	0.52**	0.51**	0.41	0.42*	0.36
	(0.25)	(0.24)	(0.26)	(0.21)	(0.34)
2-year effect (β_4 or $\Delta 5$)	1.08***	1.04***	0.87**	0.90***	0.88**
	(0.35)	(0.33)	(0.38)	(0.29)	(0.43)
3-year effect (β_6 or $\Delta 7$)	1.42***	1.39***	1.10*	1.13**	1.10**
	(0.50)	(0.49)	(0.56)	(0.46)	(0.52)
Mean of dependent variable	1.098	1.098	1.098	1.098	1.098
R^2	0.768	0.809	0.834	0.845	0.845
Panel B. Heroin and synthetic opioid deaths per 100,000 population (T40.1, T40.4)					
1-year effect (β_2 or $\Delta 3$)	0.56	0.54	0.36	0.40	0.22
	(0.41)	(0.43)	(0.38)	(0.35)	(0.34)
2-year effect (β_4 or $\Delta 5$)	1.67***	1.63**	1.24**	1.36***	1.19**
	(0.57)	(0.63)	(0.54)	(0.49)	(0.45)
3-year effect (β_6 or $\Delta 7$)	2.72***	2.68**	1.98**	2.08**	1.88**
	(0.99)	(1.07)	(0.95)	(0.90)	(0.83)
Mean of dependent variable	1.799	1.799	1.799	1.799	1.799
R^2	0.712	0.746	0.786	0.807	0.807
Ruhm (2018) correction	X	X	X	X	X
State fixed effects	X	X	X	X	X
Half-year fixed effects	X	X	X	X	X
Time-varying covariates		X	X	X	X
Pill mill laws		X	X	X	X
OxyContin reformulation			X	X	X
Measure of OxyContin use			NSDUH	ARCOS	ARCOS
Observations	1,172	1,172	1,172	1,172	1,172

Notes: The table shows the 1-year effect (the original coefficient β_2 or the trend break estimate $\Delta 3$), 2-year effect ($\beta 4$ or $\Delta 5$), and 3-year effect (β_6 or $\Delta 7$) from the baseline specification (equation 3.1) with different sets of controls. Columns 1–4 report the original estimates (β_k), and column 5 presents the trend break estimates (see equation 2.2). Although each regression includes a full set of indicators for pre- and post-periods, I report the three coefficients above for brevity. The last pre-period is omitted. Observations are weighted by state population. In Panel A, the dependent variable is heroin deaths per 100,000 (drug code T40.1). In Panel B, the dependent variable is the combined deaths from heroin and synthetic opioids per 100,000 (drug codes T40.1, T40.4). In all columns, Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. In column 1, I include only the fixed effects for state and half-year, and the indicators for pre- and post-periods. In column 2, I add an indicator for whether a state had a pill mill law and the following state- and time-varying controls: the log unemployment rate, share of the population in six age groups (0–14, 15–24, 25–44, 45–64, 65–84, 85+), share non-Hispanic black, share Hispanic, and share other race. Columns 3–6 additionally control for the 2010 OxyContin reformulation by including a measure of pre-reformulation OxyContin use interacted with the half-year fixed effects: column 3 uses the NSDUH measure, and columns 4 and 5 use the ARCOS measure of OxyContin use. In all columns, the treatment states are the 10 that implemented must-access PDMPs from 2010h2 to 2013h2, and the treated sample is balanced in relative periods from -15 to +6. Distant event periods outside the -15/+6 window are trimmed. In all columns, the control states are the 34 that did not implement must-access policies until 2016h2, and the control sample is balanced from 2003h1 to 2016h2. Florida is excluded from the control sample in all columns (see Appendix Section B.3). Standard errors clustered at the state level are in parentheses. ***, **, * denotes statistical significance at 1%, 5%, and 10% levels, respectively.

Table 2.4: Effects of Must-Access PDMPs on the Exclusive Measures of Opioid Death Rates

	Heroin Only	Illegal Opioid-Only	Prescription Opioid-Only
	(1)	(2)	(3)
Dependant variable: Drug overdose deaths per 100,000 population			
1-year effect (Δ3)	0.16	0.30	-0.93**
	(0.31)	(0.32)	(0.43)
2-year effect (Δ5)	0.38	1.09**	-1.03
	(0.41)	(0.44)	(0.64)
3-year effect (Δ7)	0.25	1.74**	-0.95*
	(0.43)	(0.76)	(0.53)
Mean of dependent variable	0.836	1.436	1.532
R^2	0.842	0.801	0.863
Ruhm (2018) correction	X	X	X
State fixed effects	X	X	X
Half-year fixed effects	X	X	X
Time-varying covariates	X	X	X
Pill mill laws	X	X	X
OxyContin reformulation	X	X	X
Measure of OxyContin use	ARCOS	ARCOS	ARCOS
Observations	1,172	1,172	1,172

Notes: The table shows the 1-year effect (the trend break estimate Δ3), 2-year effect (Δ5), and 3-year effect (Δ7) from the baseline specification (equation 3.1). In all columns, I report the trend break estimates (see equation 2.2). Although each regression includes a full set of indicators for pre- and post-periods, I report the three statistics above for brevity. The last pre-period is omitted. Observations are weighted by state population. In column 1, the dependent variable is heroin-only deaths per 100,000, which involved drug code T40.1 but not T40.2, T40.3, or T40.4 at the time of death. In column 2, the dependent variable is illegal-opioid-only deaths per 100,000, which involved drug code T40.1 or T40.4 but not T40.2 or T40.3. In column 3, the dependent variable is prescription-opioid-only deaths, which involved drug code T40.2, but not T40.1, T40.3 or T40.4. In all columns, Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. The analysis sample and controls are identical to those in Table 2.3. Fixed effects for state and half-year are always included. Standard errors clustered at the state level are in parentheses. ***, **, * denotes statistical significance at 1%, 5%, and 10% levels, respectively.

Table 2.5: Effects of Must-Access PDMPs on Prescription Opioid Death Rates and Net Effects

	Original Estimates (β_k)				Trend Break Estimates (Δn)
	(1)	(2)	(3)	(4)	(5)
Panel A. Prescription opioid deaths per 100,000 population (T40.2)					
1-year effect (β_2 or $\Delta 3$)	-0.46**	-0.52**	-0.51**	-0.54**	-1.10**
	(0.22)	(0.24)	(0.22)	(0.25)	(0.46)
2-year effect (β_4 or $\Delta 5$)	-0.14	-0.23	-0.23	-0.27	-1.04
	(0.28)	(0.31)	(0.29)	(0.32)	(0.74)
3-year effect (β_6 or $\Delta 7$)	-0.03	-0.14	-0.20	-0.27	-0.96
	(0.29)	(0.33)	(0.31)	(0.35)	(0.62)
Mean of dependent variable	1.984	1.984	1.984	1.984	1.984
R^2	0.818	0.854	0.864	0.861	0.861
Panel B. Total opioid-related deaths per 100,000 population (T40.1–T40.4)					
1-year effect (β_2 or $\Delta 3$)	0.01	-0.04	-0.16	-0.17	-0.85
	(0.47)	(0.50)	(0.42)	(0.42)	(0.54)
2-year effect (β_4 or $\Delta 5$)	1.19**	1.10*	0.80	0.84*	0.07
	(0.55)	(0.63)	(0.52)	(0.46)	(0.76)
3-year effect (β_6 or $\Delta 7$)	2.30**	2.23**	1.60*	1.61*	0.92
	(0.99)	(1.09)	(0.94)	(0.89)	(0.91)
Mean of dependent variable	4.126	4.126	4.126	4.126	4.126
R^2	0.762	0.802	0.824	0.842	0.842
Ruhm (2018) correction	X	X	X	X	X
State fixed effects	X	X	X	X	X
Half-year fixed effects	X	X	X	X	X
Time-varying covariates		X	X	X	X
Pill mill laws		X	X	X	X
OxyContin reformulation			X	X	X
Measure of OxyContin use			NSDUH	ARCOS	ARCOS
Observations	1,172	1,172	1,172	1,172	1,172

Notes: The table shows the 1-year effect (the original coefficient β_2 or the trend break estimate $\Delta 3$), 2-year effect ($\beta 4$ or $\Delta 5$), and 3-year effect (β_6 or $\Delta 7$) from the baseline specification (equation 3.1) with different sets of controls. Columns 1–4 report the original estimates (β_k), and column 5 presents the trend break estimates (see equation 2.2). Although each regression includes a full set of indicators for pre- and post-periods, I report the three coefficients above for brevity. The last pre-period is omitted. Observations are weighted by state population. In Panel A, the dependent variable is prescription opioid deaths per 100,000 (drug code T40.2). In Panel B, the dependent variable is total deaths from any opioid, including heroin, per 100,000 (drug codes T40.1–T40.4). In all columns, Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. The analysis sample and controls are identical to those in Table 2.3. Fixed effects for state and half-year are always included. Standard errors clustered at the state level are in parentheses. ***, **, * denotes statistical significance at 1%, 5%, and 10% levels, respectively.

Table 2.6: Robustness of Heroin Estimates

	Heroin Deaths per 100,000 (T40.1)							
	(1) Baseline	(2) Reported mortality	(3) Voluntary PDMPs	(4) Include FL	(5) Add MMLs	(6) Add NALs	(7) Good Sam laws	(8) Alternative dates
1-year effect (β_2)	0.42*	0.47**	0.40*	0.42*	0.35	0.46**	0.44**	0.53
	(0.21)	(0.22)	(0.21)	(0.23)	(0.26)	(0.20)	(0.21)	(0.34)
2-year effect (β_4)	0.90***	1.02***	0.89***	0.88***	0.87**	1.00***	0.94***	0.98***
	(0.29)	(0.29)	(0.29)	(0.31)	(0.32)	(0.29)	(0.29)	(0.35)
3-year effect (β_6)	1.13**	1.24***	1.11**	1.10**	1.08**	1.13**	1.16**	1.02*
	(0.46)	(0.43)	(0.45)	(0.46)	(0.47)	(0.42)	(0.46)	(0.53)
Ruhm (2018) correction	X		X	X	X	X	X	X
Number of treatment states	10	10	10	10	10	10	10	8
Number of control states	34	34	34	35	34	34	34	31
Observations	1,172	1,172	1,172	1,200	1,172	1,172	1,172	1,044
Mean of dependent variable	1.098	0.872	1.098	1.082	1.098	1.098	1.098	1.114
R^2	0.844	0.831	0.845	0.844	0.850	0.849	0.844	0.845

Notes: The table tests the robustness of my baseline estimates for heroin mortality to alternative explanations. The table shows the 1-year effect (β_2), 2-year effect (β_4), and 3-year effect (β_6), obtained from the baseline specification (equation 3.1). Although each regression includes a full set of indicators for pre- and post-periods, I report the three coefficients above for brevity. The last pre-period is omitted. Observations are weighted by state population. In all columns, the dependent variable is the heroin deaths per 100,000 (drug code T40.1). In column 1, I repeat my baseline heroin estimates from column 4 of Table 2.3. In the subsequent columns, I change or add some factors one by one. In column 2, I use the raw reported numbers of deaths, and the other columns use the Ruhm-corrected numbers of deaths. Both the corrected and reported numbers of deaths are calculated using data from the National Vital Statistics System (NVSS). In column 3, I control for an indicator for whether a state had a voluntary-access PDMP. In column 4, I include Florida in the analysis sample. Florida is excluded from the control sample in the other columns (see Appendix Section B.3). In columns 5–7, I include several other co-occurring opioid-related policies one by one: in column 5, I include a time-varying indicator for whether the state had medical marijuana laws (MMLs) as well as an indicator for whether the state had legal and operational dispensaries; in column 6, I add an indicator for whether the state had naloxone access laws (NALs), and in column 7, I add an indicator for whether the state had Good Samaritan overdose prevention laws. In column 8, I use alternative start dates of must-access PDMPs listed in the third column of Table 2.1, and in this estimation, the treated states are the 8 that implemented must-access PDMPs from 2010h2 to 2013h2, and the control states are the 31 that did not implement must-access policies until 2016h2, excluding Florida. In all columns, the distant event periods outside the -15/+6 window are trimmed. In all columns, I control for fixed effects for state and half-year, an indicator for whether a state had a pill mill law, the ARCOS measure of pre-reformulation OxyContin use interacted with the half-year fixed effects, and the time-varying covariates that are identical to those in column 4 of Table 2.3. Standard errors clustered at the state level are in parentheses. ***, **, * denotes statistical significance at 1%, 5%, and 10% levels, respectively.

Chapter 3

Beyond Opioids: The Effect of Mandatory Access Prescription Drug Monitoring Programs on Non-Opioid Prescribing*

3.1 Introduction

Over the past 25 years, the United States has undergone the most devastating drug crisis in its history. Between 1999 and 2020, drug overdose deaths have increased more than 500 percent, with nearly 92,000 deaths in 2020 alone.[1] Since opioids have been the primary driver of these increases, relatively little attention has been paid to non-opioid drugs. Although in many cases non-opioid drugs are not as fatal as opioids, the ingestion of multiple drugs or simultaneous use along with opioids could dramatically increase the risk of adverse outcomes, such as misuse and overdose (Ruhm, 2017). Unfortunately, overdose deaths involving non-opioid drugs have increased almost as fast as those involving opioids: overdose deaths involving non-opioid drugs rose 274 percent from 1999 to 2016 (Ruhm, 2019). This increase is attributable to a rise in polydrug use; that is, the simultaneous use of multiple drugs for enhanced recreational benefits. Over half of overdose deaths currently involve polydrug use, generally combining opioids with stimulants (e.g., amphetamines) or sedatives (e.g., benzodiazepines) (Ruhm, 2017, 2019).

*This chapter is jointly authored with David Beheshti. The Contribution Statement at the end of the dissertation outlines the specific contributions made by each author. We thank Jori Barash, Marika Cabral, Gue Sung Choi, Seth Neller, Ana Paula Saravia, Jinyeong Son, and Nicole Stedman for their helpful comments. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

[1]Centers for Disease Control and Prevention (`https://www.cdc.gov/drugoverdose/prevention/index.html`).

As drug overdose deaths have continued to soar, policy makers have implemented a myriad of laws and regulations in an attempt to stem the tide of rising deaths. Many of these policies specifically and exclusively target prescription opioid use and misuse.[2] In contrast, other policies such as prescription drug monitoring programs (PDMPs)—state-run databases which allow prescribers to view a patient's prescription history before prescribing controlled substances—target a wide range of prescription drugs. PDMPs track prescriptions of schedule II–V controlled drugs, including stimulants and benzodiazepines. By 2019, all but one state had established a PDMP, and more than 40 states have enacted laws that require prescribers to access the PDMP database before prescribing opioids and/or other controlled substances, commonly called mandatory access (MA) PDMPs. Because prior research has found that MA PDMPs are especially effective at changing prescribing behavior, we also focus our attention on MA PDMPs.[3]

Understandably, the vast majority of prior research has examined the impact of MA PDMPs and other policies with a primary focus on opioid-related outcomes.[4] Unfortunately, the misuse and abuse of other non-opioid substances have become increasingly common. For example, in the 2015-16 wave of the National Survey on Drug Use and Health (NSDUH), approximately 2 percent of individuals over the age of 12 reported misusing benzodiazepines in the past year, with a similar percentage reporting misuse of prescription stimulants. This misuse accounts for approximately 18 percent of all benzodiazepine use and up to 40 percent of all stimulant use. The increasing trend in polydrug misuse is particularly salient with regards to opioids along with benzodiazepines and stimulants, which now contribute to a large fraction of overdose deaths. However, there is limited evidence on the extent to which existing policies have influenced the prescribing of these drugs.

In this paper, we estimate the effect of MA PDMPs on the prescribing of stimulants and benzodiazepines using a difference-in-differences event study framework, exploiting the staggered adoption of MA PDMPs across states over time. Our analysis uses administrative data on the legal supply of stimulants from the Drug Enforcement Administration (DEA)'s

[2]For example, the reformulation of OxyContin and the rescheduling of hydrocodone-combination products.

[3]Prior research has shown that, when not mandated, PDMP engagement among prescribers is low (Haffajee et al., 2015; Kreiner et al., 2014).

[4]See Section 3.2.1 for a discussion of this literature.

Automation of Reports and Consolidated Orders System (ARCOS) and data on the prescribing of stimulants and benzodiazepines from the Medicaid State Drug Utilization Data over the period 2008–2017.[5] Our outcomes of interest are amphetamine-equivalent stimulant grams per 100 population and benzodiazepine prescriptions per 100 Medicaid enrollees.[6]

Overall, our estimates indicate that MA PDMPs led to decreases in the legal supply of stimulants. The results patterns are similar pooling across different stimulants and examining different types of stimulants separately (e.g., amphetamine and lisdexamfetamine). Five years following policy implementation, MA PDMPs were associated with a 16.6 percent decrease in amphetamine-equivalent stimulant grams per 100 population relative to the mean one year before treatment. Our point estimates range between a 15.9–21.5 percent decrease when we examine each type of stimulant separately.[7] We find qualitatively similar results when implementing the method of Sun and Abraham (2021), suggesting that our results are not driven by treatment effects that are heterogeneous over time (i.e., dynamic treatment effects). Likewise, we find qualitatively similar results using a synthetic controls approach, which allows for the construction of control groups for each state that more closely match the pre-treatment dynamics.

Interestingly, we find opposite signed effects when we examine benzodiazepine prescriptions. Using a two-way fixed effects regression framework, we find that the implementation of a MA PDMP leads to an immediate increase in benzodiazepine prescribing. In the year of MA PDMP implementation, we observe a 9.75 percent increase in benzodiazepine prescriptions per 100 Medicaid enrollees relative to the mean one year before

[5]The DEA does not track benzodiazepine shipments.

[6]We also report the results obtained using an alternative measure of stimulant prescribing constructed using data from Medicaid (i.e., stimulant prescriptions per 100 Medicaid enrollees).

[7]Our estimates indicate that stimulant grams per 100 population decrease by 0.91 for amphetamine (16.5% in terms of the mean one year before the treatment, $p-$value=0.006), by 0.51 for methylphenidate (15.9%, $p-$value=0.137), and by 0.10 for lisdexamfetamine (21.5%, $p-$value=0.073).

the treatment.[8] In addition, we find suggestive evidence that five years following policy implementation, MA PDMPs were associated with a 5.97 percent increase in aggregate benzodiazepine prescribing, although the effects are heterogeneous across benzodiazepine types. However, none of these long-run effects are statistically significant.[9] Overall, our results are qualitatively similar using a synthetic controls approach, but are significantly attenuated using the estimator proposed by Sun and Abraham (2021). We therefore view the benzodiazepine results as more suggestive relative to the clear effects we document with stimulants.

MA PDMPs could reduce non-opioid drug prescribing through several different channels. First, PDMPs are designed to affect prescribing by providing information on patients' prescription history, which allows providers to identify inappropriate prescribing trends. Second, Alpert et al. (2020) suggest that the "hassle cost" of required access to the PDMP database could deter physicians from prescribing controlled substances under any circumstances. Our finding that MA PDMPs reduce stimulant prescribing may reflect these two channels. However, there are also mechanisms by which MA PDMPs could actually increase the utilization of certain drugs. For example, reductions in the availability of commonly diverted drugs may increase demand for substitute drugs (such as benzodiazepines). Several studies have shown that MA PDMPs have unintended consequences of shifting users toward illicit opioids, which are often taken in conjunction with benzodiazepines (Meinhofer, 2018b; Kim, 2021b). Similarly, as MA PDMPs limit access to opioids, they could increase the share of patients with opioid withdrawal

[8]Our estimates indicate that the number of prescriptions per 100 Medicaid enrollees increase by 1.58 for alprazolam (15.5% in terms of the outcome mean one year before the treatment, $p-$value=0.011), by 0.91 for clonazepam (9.54%, $p-$value=0.047), by 1.21 for lorazepam (20.2%, $p-$value=0.02), by 0.43 for diazepam (11%, $p-$value=0.063), and by 0.24 for temazepam (19.2%, $p-$value=0.219), in the year of policy implementation.

[9]We find that the number of prescriptions per 100 Medicaid enrollees increase by 1.75 for alprazolam (17.1% in terms of the outcome mean one year before the treatment, $p-$value=0.5), 0.86 for clonazepam (9%, $p-$value=0.7), 1.31 for lorazepam (21.9%, $p-$value=0.371), and 0.37 for temazepam (29.6%, $p-$value=0.634). The number of diazepam prescriptions per 100 enrollees decreased by 0.7 (18%, $p-$value=0.623) five years after the treatment.

symptoms, who may seek benzodiazepines to treat opioid withdrawal.[10]

Despite a large literature on the effect of PDMPs on opioid-related outcomes (e.g., Meinhofer, 2018b; Buchmueller and Carey, 2018), only a handful of papers have examined the effects of MA PDMPs on non-opioid-related outcomes. Consistent with our findings, Meinhofer (2018b) finds that MA PDMPs lead to a decrease in the supply of prescription stimulants. In contrast, several studies have examined the effects of MA PDMPs on benzodiazepine-related outcomes and produced mixed results. Meinhofer (2018b) shows that benzodiazepine-involved deaths decline following MA PDMPs. Other studies in the medical literature find no effect of MA PDMPs on benzodiazepine deaths (Liang and Shi, 2019), benzodiazepines dispensed, dosage, or spending (Liang et al., 2021).[11]

The findings of this paper contribute to this literature in two key ways. First, unlike most prior work, which was only able to investigate the short-term effect of the policy, we use a longer period to analyze how MA PDMPs affect non-opioid prescribing in the medium term. For example, Meinhofer (2018b)—the most closely related paper—only covers PDMPs implemented by 2013. The majority of MA PDMPs have been relatively recently implemented and having both additional states and years of data allows us to better understand their policy effects. While Meinhofer (2018b) presents event study regression estimates of PDMPs up to two years after the implementation of a PDMP, we are able to use our longer sample period to trace out their effects up to five years after implementation. This is especially important for stimulants, which exhibit stronger responses as time passes. Second, while other studies only focus on aggregate measures for the prescribing of stimulants or benzodiazepines, we

[10]Stein et al. (2016) surveyed those who used benzodiazepines in the month prior to initiating inpatient opioid detoxification; among the 176 survey participants, 10.2% reported the reason for benzodiazepine use as 'to decrease opioid withdrawal.'

[11]Liang and Shi (2019) use the Medicaid State Drug Utilization Data to study the impact of PDMP mandates for use of benzodiazepine records on benzodiazepine prescribing. Using an event study design, they find no evidence for the association between the mandates and quantity, dosage, and Medicaid spending of benzodiazepine prescriptions per 100 enrollees in a quarter-period. The key difference between our work and Liang and Shi (2019) is that they focus explicitly on the states having PDMP mandates for use of benzodiazepine records, while we include a broader set of PDMP mandates in our analysis. As discussed above, there are several different channels through which any PDMP mandate (e.g., mandate for use of opioid records only) can affect benzodiazepine prescribing.

investigate the policy effects on the prescribing of each type of drug separately.[12] While drugs belonging to the same class have similar properties, there are important differences that could result in heterogeneous responses to policy shocks. For example, alprazolam and clonazepam are both benzodiazepines and are commonly prescribed to treat anxiety. However, alprazolam has a relatively faster onset and is associated with a better subjective high, making it the preferred drug among most recreational users (Lader, 2011). In contrast, clonazepam has a slower onset but longer-lasting effects, making it more preferred for treating opioid withdrawals (Stein et al., 2016). While we find that most of the different generic drugs within a broader category have similar responses to MA PDMPs, there is some degree of heterogeneity, especially for benzodiazepines.

Our findings inform the policy discussion surrounding MA PDMPs along two key dimensions. First, our results highlight the fact that MA PDMPs impact drug prescribing patterns for a variety of non-opioid drugs. This is important in light of the complicated interrelationships between various drugs. The effect of PDMPs on drug prescribing depends not only on the direct effects of the PDMP on physician behavior, but also on the demand response which is a function of the substitutability or complementarity of a myriad of different drugs. Second, these effects are not uniform across drug types. While we find decreases in stimulant prescribing, our results suggest increases in benzodiazepine prescriptions in response to MA PDMPs. This heterogeneity could be the result of differences in regulatory scrutiny for these different drug types (e.g., stimulants are typically Schedule II drugs, while benzodiazepines are Schedule IV), or they may reflect important differences in how these drugs relate to each other. For example, if MA PDMPs reduce access to certain drugs, then we may expect to see increases in demand for substitutes. This would be true even if the substitute drug is also covered by the PDMP. Therefore, it is unlikely that PDMPs will uniformly decrease prescribing of all commonly misused drugs.

Our paper proceeds as follows: in section 3.2 we discuss the institutional details of PDMPs as well as some of the most closely related literature. We also provide background information on stimulants and benzodiazepines, the two drug classes of interest in this paper.

[12] We analyse amphetamine, methylphenidate, and lisdexamfetamine separately in our stimulant analysis. Likewise, we analyze alprazolam, clonazepam, lorazepam, diazepam, and temazepam separately in our benzodiazepine analysis.

In section 3.3, we describe our identification strategy. We describe our data in section 3.4. We present our main results in section 3.5, and conclude in section 3.6.

3.2 Background

3.2.1 Prescription Drug Monitoring Programs and Related Literature

PDMPs A PDMP is a state-level database that collects information on patients' scheduled prescription medications at the point of prescribing or dispensing.[13] PDMPs are designed to help providers identify inappropriate use of scheduled prescription medications. Authorized providers are able to access the database for patients' controlled substance prescription histories before prescribing. By 2017, all states but Missouri had a modern electronic PDMP system in operation (Horwitz et al., 2021).[14] However, when providers are not required to access the database before prescribing, provider participation rates are low (Haffajee et al., 2015).[15] In response to the low participation rates, 26 states implemented a mandatory access provision between 2007 and 2017.[16] MA PDMPs legally require providers to use the PDMP database before controlled substance prescribing under certain conditions. Provider utilization has substantially increased following the implementation of MA PDMPs. For example, the number of active users in New York reached 67,779 in the first six months of policy implementation, while it had only 5,087 users prior to the mandate (PDMP Center of Excellence, 2014).

Although PDMPs have historically been considered as a means to combat prescription opioid diversion and misuse, they typically encompass a variety of different drugs. For example, of the 26 mandatory access PDMPs that were implemented between 2007–2017, 12 of them require the prescriber to query the PDMP prior to prescribing any

[13] Controlled substances are placed into one of 5 "schedules" reflecting their medical efficacy and potential for misuse. Schedule I drugs are federally illegal, while Schedule II-V drugs are available only via prescription, with lower numbered schedules reflecting higher potential for misuse.

[14] See Horwitz et al. (2021) for more information.

[15] The utilization rate among healthcare providers in states without the mandates is about 14 to 25 percent (Alexander, 2015).

[16] See Table 3.1 and Appendix Figure C.3.

Schedule II substances (e.g., stimulants).[17] Likewise, 17 explicitly require the prescriber to query the PDMP to prescribe benzodiazepines (schedule IV). In our primary specification, we construct our treatment variable as an indicator for whether the state has a mandatory access provision for any drug. Prior research has highlighted hassle costs as an important mechanism by which PDMPs reduce drug prescribing, even if the information provided by the PDMP does not necessarily warrant the reduction (Alpert et al., 2020). Our results are consistent with a large role for hassle costs, with drug prescriptions falling as a result of PDMPs even for drugs that are not explicitly included.[18]

Related Literature A rapidly expanding literature has documented the effects of PDMPs on a variety of outcomes. Early work in this area that did not distinguish between voluntary and mandatory access programs produced mixed results on an impact of PDMPs on opioid-related outcomes. For example, Meara et al. (2016) find no statistically significant effect of PDMPs on various opioid-prescribing outcomes among Medicare beneficiaries. In contrast, other work shows that PDMPs were associated with reduced opioid-related mortality (Kilby, 2016) and reduced rates of opioid prescribing in ambulatory care settings (Bao et al., 2016).

Studies examining states where prescribers are required to query the PDMP prior to prescribing—commonly referred to as mandatory access PDMPs—have shown significant reductions in prescription opioid misuse (Buchmueller and Carey, 2018; Grecu et al., 2019; Kim, 2021b; Mallatt, 2018; Meinhofer, 2018b; Wen et al., 2019). For example, Buchmueller and Carey (2018) show that MA PDMPs reduce measures of excessive opioid consumption and doctor shopping among Medicare beneficiaries. Likewise, Mallatt (2018) finds that the implementation of a MA PDMP reduces oxycodone shipments by 8 percent. Consistent with this reduction in opioid prescribing, Grecu et al. (2019) find a 20–26 percent decline

[17] Data on what drugs are included in the mandate are from the Pew Charitable Trusts. For more details, see: `https://www.pewtrusts.org/en/research-and-analysis/data-visualizations/2018/when-are-prescribers-required-to-use-prescription-drug-monitoring-programs`, last accessed April 24, 2022.

[18] In results not presented here, we find little evidence of heterogeneity by whether the law explicitly requires prescribers to check the PDMP prior to prescribing stimulants or benzodiazepines. One potential explanation for this is that prescribers are unaware of the finer details of the law and mistakenly believe that all controlled substances are covered.

in admissions to drug treatment facilities following the implementation of a MA PDMP. In addition, these reductions in prescribing have resulted in fewer overdose deaths involving prescription opioids. For example, Meinhofer (2018b) shows that prescription opioid-related deaths decrease by 9 percent following MA PDMP implementation.

Later work has considered the impact of mandatory access PDMPs beyond opioid prescribing and overdose deaths. Several recent papers have examined substitution toward illicit substances, especially heroin and fentanyl, in response to reduced prescription opioid access as a result of mandatory access PDMPs. Meinhofer (2018b) and Kim (2021b) find that mandatory access PDMPs led to increases in heroin overdose deaths, offsetting reductions in prescription opioid overdose deaths. Likewise, Mallatt (2018) shows that PDMPs led to increases in heroin-related crime in counties with high levels of pre-PDMP prescription opioid use.

Given the broad scope of PDMPs and their impacts on opioid prescribing, it is plausible that they could alter prescription patterns for other drugs as well. There is, however, a dearth of evidence on the effects of PDMPs on non-opioid prescriptions. Meinhofer (2018b) shows that MA PDMPs lead to a decrease in the supply of prescription stimulants. In contrast, several studies have examined the effects of MA PDMPs on benzodiazepine-related outcomes and produced mixed results: Meinhofer (2018b) shows that benzodiazepine-involved deaths decline following MA PDMPs; Winstanley et al. (2018) find that Ohio's mandate led to a statistically significant decrease in benzodiazepines dispensed; other studies in the medical literature find no effect of MA PDMPs on overdose deaths involving benzodiazepines (Liang and Shi, 2019), benzodiazepines dispensed, dosage, or spending (Liang et al., 2021).

3.2.2 Benzodiazepines and Stimulants

Benzodiazepines Benzodiazepines are a class of drugs that are most commonly prescribed to treat anxiety and panic disorders, although they are widely used to treat other ailments. Benzodiazepines are commonly referred to as "benzos", and include drugs such as alprazolam (brand name "Xanax"), diazepam (brand name "Valium"), and clonazepam (brand name "Klonopin"). These drugs work by suppressing the activity of nerves in the brain, relieving symptoms of various psychological problems. While benzos do not typically

produce euphoric effects common in recreational drugs, they are frequently misused for their calming and sedative properties. Fatal overdoses are uncommon when using benzos in isolation. However, benzos interact strongly with depressants such as alcohol and opioids. These interactions amplify the recreational properties of the drugs, but they also greatly increase the probability of respiratory depression and death. In fact, opioids were involved in the vast majority of benzodiazepine overdose deaths (Ruhm, 2019).

Stimulants Stimulants refer to a broad class of legal and illegal drugs that act on the central nervous system to increase alertness and energy. Stimulants range from ubiquitous drugs such as caffeine to prescription drugs including amphetamine (brand name "Adderall") and methylphenidate (brand name "Ritalin") to Schedule I drugs such as MDMA. Prescription stimulants are commonly used to treat attention deficit hyperactivity disorder (ADHD), but are also used for their recreational effects. Taken in high doses, stimulants can produce intense feelings of euphoria. Stimulants are also used as appetite suppressants and as "study-drugs", enhancing the user's ability to focus for long periods of time. However, stimulants use can also lead to agitation and anxiety, among other adverse behavioral effects. Physically, stimulants can elevate blood pressure to dangerous levels and lead to heart attack or stroke.

Benzodiazepines and stimulants are widely consumed. Figure 3.1 displays the rates of use and misuse for various drugs from the 2015-2016 wave of the National Survey on Drug Use and Health (NSDUH). White bars represent any use of the drug (including legitimate medical use), while gray bars represent misuse. Approximately 11 percent of respondents reported using benzodiazepines at some point in the previous year. A little under one-fifth of these users reported misusing benzodiazepines, that is, use without a legitimate prescription or for the sole purpose of recreation. The most commonly used and abused benzodiazepine was alprazolam, commonly sold under the brand name Xanax. Stimulant use, on the other hand, was reported by about 5 percent of respondents, a little under half the fraction of benzodiazepine use. However, despite the lower overall prevalence, stimulants were misused at nearly the exact same rate as benzodiazepines. For the sake of comparison, this figure also includes analogous numbers for the two most commonly prescribed opioids, oxycodone and hydrocodone. Over 35 percent of respondents reported consuming these opioids at some point in the last year, with about 5 percent reporting misuse. Therefore, while opioid use

and misuse is more prevalent than the use and misuse of benzodiazepines or stimulants, the fraction of users who misuse the drug is higher for these latter drug classes.

3.3 Research Design

Our empirical strategy for estimating the causal impact of MA PDMPs exploits variation in the timing of adoption across states. Specifically, we estimate event study difference-in-differences regressions of the form:

$$Y_{st} = \alpha_s + \beta_t + \sum_{k \neq -1} \gamma_k \mathbf{1}(MA\ PDMP_{sk}) + X_{st}\delta + \varepsilon_{st}, \tag{3.1}$$

where Y_{st} is the outcome variable measured at the state-by-year level, and α_s and β_t are state and year fixed effects, respectively. The indicator variable $\mathbf{1}(MA\ PDMP_{sk})$ is set equal to 1 if state s enacted a MA PDMP k years ago. The coefficients of interest, γ_k, indicate the difference in outcome between treatment and control states in period k, relative to the last pre-policy period, conditional on the other control variables. We trim all post-periods after the fifth ($k > 5$) and all pre-periods more than nine years prior ($k < -9$). X_{st} is a vector of time-varying covariates. In our regressions for stimulant distribution outcomes, we control for race/ethnicity composition (the share of the population that is non-Hispanic white, non-Hispanic Black, Hispanic, other) and age composition (the share of ages under 15, 15–24, 25–44, 45–64, 65–84, over 85).[19] Observations are weighted by state population (Medicaid enrollment) in our regressions for stimulant distribution (Medicaid prescribing) outcomes. Standard errors are clustered at the state level. Our analysis sample consists of 24 treatment states that implemented MA PDMPs between 2009 and 2017 and 22 control states that did

[19] We do not include any time-varying covariates in our regressions for Medicaid prescribing outcomes, but our results are nearly identical when we control for them.

not implement the policy until 2018.[20] Since states implemented the policy with different timing, our sample of states and years is unbalanced in relative periods.

The key identifying assumption in this model is that, absent the implementation of a MA PDMP, control and treatment states would have trended in parallel conditional on the covariates. We assess the plausibility of this assumption by plotting the γ_k coefficients which allows us to examine whether treatment and control states were trending in parallel prior to treatment. As we will show in section 3.5, this assumption appears reasonable for many of our outcome variables. However, for certain outcomes we find evidence of pre-existing trends, casting doubt on this assumption. Therefore, we supplement our event study approach with a synthetic controls analysis.

3.3.1 Synthetic Control Analysis

We complement our event study regressions with a synthetic control analysis. The idea is to construct a comparable synthetic control state for each treated state based on pre-period data in such a way that the synthetic control state has similar trends in outcomes to the treated state prior to policy implementation. If the results from our synthetic control analysis are similar to the baseline results, it will imply that pre-treatment differences between the treated and control groups are not likely to be responsible for our results.

While a synthetic control approach has been more widely conducted for a single treated unit or multiple units with the same treatment timing, this method has recently been adopted for the case of multiple units with differential treatment timing (e.g., Kleven, 2019; Acemoglu et al., 2016). To conduct a synthetic control analysis, we first construct a synthetic control state for each treated state and then create a sample so that both the treated and synthetic control samples are strongly balanced in relative periods.

[20] As shown in Table 3.1, 29 states implemented a MA PDMP until 2018. Since our data covers 2008–2017, our analysis focuses on states that adopted a mandate during our sample period. We drop four states that enacted the law outside the period 2008–2017 (i.e., either pre-2008 or 2018), but our results are similar if we include these "already treated" or "not yet treated" units. In addition, we drop one treatment state which implemented a MA PDMP in 2008, for which we do not observe any pre-treatment period in our data, to be consistent with our synthetic control analysis in Section 3.3.1. Our results are robust to including this state. Our final analysis sample consists of 24 treatment states and 22 control states.

For each of these treated states, we construct a synthetic control state from the 22 control states included in the baseline sample, matching on an outcome variable measured in each of the pre-treatment periods.[21] Note that the number of pre-treatment periods differs across states, and we use all available pre-treatment periods to construct a synthetic control. Each synthetic control state is composed of a weighted average of observations from the subset of the 22 control states.[22][23]

Using observations from the treated and synthetic control groups, we create a sample so that each treated and the matched synthetic control sample are strongly balanced from relative period -x_{1s} to +x_{2s}, where $-x_{1s}$ ($+x_{2s}$) is the earliest (latest) relative period available for the state.[24] For each treated state and each relative period, we calculate the difference in the outcome variable between the treated state and matched synthetic control. Finally, for each relative period, we take the average difference in the outcome between the treated states and synthetic controls, weighting by state population (or Medicaid enrollment for Medicaid prescribing outcomes) measured in 2008 (i.e., the baseline period). In the results section, we show how the average difference in the outcome between the treated and synthetic control groups change around the time of policy implementation.

3.4 Data

3.4.1 Prescribing Data

ARCOS Our primary dataset measuring the distribution of various stimulants is the Automated Reports and Consolidated Ordering System (ARCOS). These data are reported

[21] We use the Stata command `synth` to construct a synthetic control. For more details, see: https://fmwww.bc.edu/RePEc/bocode/s/synth.html.

[22] Tables reporting the makeup of the synthetic state for each treated state and for each outcome are available upon request.

[23] As we describe in Section 3.4, we only include in our sample the generic type-state-year observations that consistently report in all four quarters (around 96.5% of all generic type-state-year observations). Since we construct a synthetic control unit by matching on an outcome variable measured in each of the pre-treatment periods, requiring consistent observations over the pre-periods, we use the linear interpolation and/or extrapolation methods to impute the dropped values.

[24] For example, if a treated state implemented the policy in 2011, $-x_{1s}$ is equal to -3 and $+x_{2s}$ is equal to $+6$, since our sample period is 2008–2017.

at the state-by-quarter level by the Drug Enforcement Agency (DEA). They are constructed from reports sent to the DEA by distributors and manufacturers, who are required by law to report all transactions of certain controlled substances.[25]

We obtain information about the weight in grams of amphetamine, methylphenidate, and lisdexamfetamine distributed to each state for each quarter from 2008-2018.[26] Although these data do not directly measure the amount of each substance consumed in each period, prior research has shown that measures of drug distribution from ARCOS are highly correlated with measures of consumption from other datasets (Beheshti, 2022). We also create an aggregate measure of stimulant supply by pooling together each stimulant, weighted by potency. Specifically, we create a measure of amphetamine-equivalent milligrams using the conversion factors listed in Appendix Table C.1.

We display the aggregate distribution of each stimulant in Appendix Figure C.1. From 2008 to 2017, the per capita supply of amphetamine and lisdexamfetamine more than doubled. In contrast, the quantity of methylphenidate distributed in each quarter remained relatively constant over this period. Since the DEA does not track benzodiazepine sales, we are unable to examine trends in benzodiazepine shipments over time.

Medicaid We use the Medicaid State Drug Utilization Data from the Centers for Medicare and Medicaid Services (CMS) over the period 2008–2017. The data provide state-quarter level counts of prescriptions reimbursed by Medicaid (both fee-for-service and managed care) separately by National Drug Code (NDC). We first categorize NDCs into generic types using the product name and then collapse the NDC-state-quarter aggregate prescription records

[25]Title 21, United States Code, Section 827(d)(1), and Title 21, Code of Federal Regulations, Section 1304.33.

[26]There exist ARCOS reports back to 2000, although prior to 2008 different forms of amphetamines are reported separately, making comparisons prior to 2008 difficult.

into generic type-state-year level data.[27][28]

For benzodiazepines, we include in our analysis the generic types alprazolam, clonazepam, lorazepam, diazepam, and temazepam; for stimulants, we include amphetamine, methylphenidate, and lisdexamfetamine. Our outcome of interest is the number of prescriptions per 100 Medicaid enrollees for each generic type of benzodiazepine and stimulant.[29] We also create an aggregate measure of benzodiazepine prescribing by adding together the number of each type of benzodiazepine prescription.[30] Data on Medicaid enrollment are obtained from the Kaiser Family Foundation.[31] We show time series figures of the rates of stimulant and benzodiazepine prescriptions in panels (a) and (b) of Figure C.2, respectively.

[27]For each NDC-state-quarter record, the Medicaid State Drug Utilization Data provide the first 10 characters of product name that is approved by the Food and Drug Administration (FDA). A product name contains either a generic name or a brand name. Using this product name, we categorize NDCs into generic types. In Appendix Table C.2, we list brand names for each generic type that we use for our categorization. The list of brand names is adapted from FDA and several other sources. We do not list the brand names if no corresponding records are included in the 2008–2017 Medicaid State Drug Utilization Data. Note that we do a partial string matching, so any product names that contain a given brand name are included in our sample. For example, both the product names "XANAX XR" and "XANAX .25M" are identified by the brand name "XANAX" and thus included in our sample.

[28]For each generic type, we only include state-years that consistently report in all four quarters (around 96.5% of all generic-state-year observations). CMS suppresses NDC-state-quarter observations if there are less than eleven counts. We replace suppressed observations with zero, but results are similar if we set these values to be five instead.

[29]Liang and Shi (2019) analyze the impact of MA PDMPs on the prescribing of benzodiazepine and find similar patterns across number of prescriptions, dosage of prescriptions, and spending on benzodiazepine prescriptions. In our analysis, we focus on the number of prescriptions.

[30]We set the value of the aggregate measure as missing if information on any of these types is missing.

[31]We use monthly Medicaid and CHIP enrollment measured in June. Data on total monthly Medicaid and CHIP enrollment over the period June 2014–June 2017 are taken from: `https://www.kff.org/health-reform/state-indicator/total-monthly-medicaid-and-chip-enrollment/?currentTimeframe=0&sortModel=%7B%22colId%22:%22Location%22,%22sort%22:%22asc%22%7D`, last accessed March 7, 2022. Data on total monthly Medicaid enrollment over the period June 2008–June 2013 are taken from: `https://www.kff.org/medicaid/issue-brief/medicaid-enrollment-june-2013-data-snapshot/view/print/`, last accessed March 7, 2022. Data on total monthly CHIP enrollment over the period June 2008–June 2013 are taken from: `https://www.kff.org/medicaid/issue-brief/chip-enrollment-june-2013-data-snapshot/view/print/`, last accessed March 7, 2022.

We present summary statistics on each of our primary outcome variables in Table 3.2. The odd-numbered columns display the average value across all years from 2008 to 2017, while the even-numbered columns display the associated standard deviations. The first two columns use data from the entire sample, while columns (3) and (4) show only those states that adopted a MA PDMP at some point in our sample period. Likewise, columns (5) and (6) present summary statistics for states which did not adopt a MA PDMP until 2018. This table also includes demographic information such as age and race compositions, which we include as control variables.

NSDUH We obtain data on the use and misuse of benzodiazepines and stimulants from the National Survey on Drug Use and Health (NSDUH). NSDUH has collected nationally representative data on prescription drug use and misuse, among the randomly sampled non-institutionalized US civilians aged 12 or older. We construct the measures for the overall use and misuse of stimulants and benzodiazepines among NSDUH respondents over age 12 in the 2015 and 2016 survey years (N=114,043).[32]

We display these rates in Figure 3.1, along with rates of opioid (mis)use for comparison.[33] The light bars show the fraction of respondents over the age of 12 who report any use of the drug, including legitimate medical use. The dark bars indicate the fraction who explicitly report misusing the drug. The second column indicates that around 11.3 percent of individuals used benzodiazepines in 2015-2016. Approximately 18.3 percent of those reported misusing the drug. The next four columns break out this estimate by the four most common types of benzodiazepines. Rates of stimulant use are much lower, around five percent. However, nearly 36.5 percent of this was misuse, putting overall stimulant

[32]We focus on the 2015–2016 data to construct the consistent measures. NSDUH survey was partially redesigned in 2015 to collect more detailed and complete information on the use and misuse of prescription drugs, including stimulants and benzodiazepines. Prior to 2015, NSDUH definition of prescription drug misuse was limited to "nonmedical use," but the 2015 definition of misuse was revised to use a drug "in any way a doctor did not direct." For more details, see: `https://www.samhsa.gov/data/sites/default/files/NSDUH-TrendBreak-2015.pdf`, last accessed April 24, 2022.

[33]We proxy for this with (mis)use of either oxycodone or hydrocodone. Inconsistency across questions for different drugs prevents us from including a broader set of opioids. These two drugs make up the majority of opioid prescriptions in the United States, and are the most commonly misused prescription opioids.

misuse almost identical to the overall rate of benzodiazepine misuse. Both of these are lower than the corresponding rates for opioids, consistent with the larger research focus on opioid misuse.

3.4.2 PDMPs

Table 3.1 shows the effective dates of the laws used in this paper, taken from Sacks et al. (2021). Appendix Figure C.3 presents the trends in the total number of states with MA PDMPs. By the end of 2017, 26 states had passed MA PDMP laws.

3.5 Results

A rapidly growing literature has considered the effect of MA PDMPs on opioid prescribing and related outcomes. Although voluntary access PDMPs had limited efficacy in reducing prescriptions, studies focusing on MA PDMPs have shown stronger effects.[34] Given the consistent finding of this prior work, we do not discuss our replication of this finding here.[35] We instead focus our discussion on stimulants and benzodiazepines, drug categories that have not been considered to the same extent as opioids.

Stimulants We first consider the effect of MA PDMPs on stimulant prescribing. There are three different types of stimulants included in the ARCOS dataset: amphetamine, methylphenidate, and lisdexamfetamine. We present the regression coefficients from equation 3.1 for each of these outcomes, as well as our measure aggregating across these three types, in Figure 3.2.[36] We consider our aggregate measure in panel (a). Prior to the implementation of a MA PDMP, each of the coefficients is small in magnitude and statistically indistinguishable from zero. This pattern of coefficients lends plausibility to our identifying assumption, that treated states would have trended in parallel to untreated states in the absence of treatment. Immediately after the implementation of a MA PDMP, however, the coefficients become negative and continue to grow in magnitude as time passes. After five years, the coefficient is equal to -1.52. Relative to the mean of 9.179 one year

[34] See Maclean et al. (2020) for a review.

[35] These results are available upon request, and fall within the range of estimates in the existing literature.

[36] The coefficients are listed in table form in Table 3.3.

before treatment, this is a decrease of 16.6 percent. In the remaining panels, we present the results for each type of stimulant separately. We consider the number of grams of amphetamine per 100 individuals in panel (b). The pattern of coefficients is nearly identical to panel (a), revealing no evidence of pre-existing trends. After five years, the coefficient is equal to -0.91, a decrease of 16.5 percent. In panel (c), we turn our attention to methylphenidate. Five years after the treatment begins, the point estimate of -0.51 indicates a reduction of 15.9 percent, very similar to what we observed for amphetamine. Finally, we consider lisdexamfetamine in panel (d). The pattern is again nearly identical to what we observed in panels (b) and (c), and indicates a reduction of about 21.5 percent five years after the implementation of a MA PDMP.[37]

To probe the sensitivity of these results, we also employ a synthetic controls approach. As discussed in Section 3.3.1, we construct a synthetic version of each state consisting of a convex combination of other states which never adopted MA PDMPs over our sample period. The exact convex combination is chosen to mimic the treated state's outcome dynamics prior to treatment. We then compute the difference between each treated state's actual and synthetic counterpart and present the average difference in each period in Figure C.4.[38] Similarly to Figure 3.2, we present the results for our aggregate measure in panel (a), followed by amphetamine, methylphenidate, and lisdexamfetamine separately in panels (b)-(d), respectively. In panels (b)-(d), the pre-period coefficients are (by construction) close to zero. In the post-period, we observe very similar dynamics to those from our primary regressions. Furthermore, the point estimates are quite similar using the two different methodologies, though the magnitudes of point estimates for the last post-period are larger in our synthetic control analysis than the corresponding regression estimates. For example, in panel (b) we estimate that five years after the adoption of a MA PDMP, adopting states amphetamine prescribing (measured as grams distributed per 100 population) is about -1.46, compared to -0.91 from the event study. Similarly in panels (c)

[37] In comparison, Meinhofer (2018b) finds that stimulant grams decrease by 10 percent in the first two years of MA PDMP. Our results indicate that these effects continue to grow up to five years after MA PDMP implementation, highlighting the benefits of using a longer panel.

[38] Coefficients are listed in table form in Table C.3. Results for each individual state are available upon request.

and (d), the point estimates are larger than the regression estimates. Qualitatively, however, the synthetic control analysis confirms what we observe using the regression approach.[39]

Next, we consider an alternate measure of prescribing using data from Medicaid. This measure captures the number of prescriptions written per 100 enrollees. By measuring the number of prescriptions per enrollee as opposed to the weight of the drug distributed per capita, we complement our measure of intensive margin prescribing with a measure focusing on extensive margin prescribing. However, since we are now examining Medicaid enrollees as opposed to the general population, we cannot rule out any differences in our results being due to differences in the sample composition rather than the difference in the intensive versus extensive margin. The results from this exercise are shown in Figure C.5.[40] Beginning with panel (b), we observe a point estimate of -3.75 five years after adoption, relative to a mean of 5.49, a 68 percent decrease. This is somewhat larger than what we observe when using our main measure of prescribing. Examining panel (c), however, we find results that are qualitatively different. Our last post-period coefficient is a positive 1.92, suggesting an increase in methylphenidate prescriptions. However, this five-year effect is statistically insignificant at the 5 percent significance level. In addition, examining the pattern of coefficients in the pre-period suggests that this may simply be the continuation of trends that existed prior to treatment, rather than an effect of MA PDMPs. For these reasons, we are hesitant to draw strong conclusions about this outcome variable. In panel (d), we see results that are qualitatively similar to those using the ARCOS measure. Our last coefficient is -1.05, indicating a 19 percent decrease relative to the mean of 5.4. This is nearly identical to what we observe in the ARCOS data. Since methylphenidate has different signed effects than amphetamine and lisdexamfetamine, pooling them together in panel (a) results in somewhat muted results. This highlights the importance of reporting each drug separately.

Lastly, we also consider synthetic control estimates using our prescribing measure from Medicaid. The results from this exercise are shown in Figure C.6.[41] Qualitatively, these results mimic the regression results from Figure C.5. For amphetamine and lisdexamfetamine (panels

[39] In panel (a), the coefficients are shifted up relative to the remaining panels. This is driven by poor synthetic matches for a few states. However, the dynamics look quite similar.

[40] We report the coefficients in table form in Table C.4.

[41] We include the coefficients in table form in Table C.5.

(b) and (d)), we find a decrease of about three prescriptions per 100 enrollees five years after policy implementation for each drug. This is similar in magnitude to what we observe in Figure C.5 for amphetamine, although larger in magnitude for lisdexamfetamine. In panel (c), we again observe an increase in methylphenidate. Combining these drugs in panel (a), we see an overall reduction in stimulant prescribing of about 20.6 percent five years after MA PDMP implementation.

Overall, these results demonstrate a consistent reduction in stimulant prescribing after the implementation of MA PDMPs, similar to what is typically reported in studies that examine opioid prescribing. This is consistent with either information provision— prescribers learning about potential misuse or diversion— or hassle costs— prescribers simply not wanting to engage with the PDMP. The overall welfare effects are unclear, however, as we cannot separately identify reductions in unnecessary prescribing from reductions in appropriate prescribing.

Benzodiazepines Next, we turn our attention to benzodiazepines. We consider these drugs for three reasons. First, we are inherently interested in benzodiazepines due to the increased frequency of overdose deaths involving benzodiazepines. Second, given their less stringent regulatory status, we are interested in whether MA PDMPs have differential effects relative to opioids and stimulants. Finally, benzodiazepines act as both a complement to other recreational drugs (e.g, enhancing the euphoric effects of opioids) as well as a substitute (e.g., alleviating the negative symptoms of withdrawals). There are therefore ambiguous theoretical effects of MA PDMPs.

Since benzodiazepines are Schedule IV drugs, benzodiazepine shipments are not tracked by the DEA. We therefore only consider prescriptions per 100 Medicaid enrollees. We present the event study coefficients from equation 3.1 for all benzodiazepines pooled together in panel (a) of Figure 3.3, followed by alprazolam, clonazepam, lorazepam, diazepam, and temazepam separately in panels (b) through (f), respectively.[42] In panel (a) we observe relatively flat pre-trends, followed by positive coefficients in the post-period. Panel (b) also shows positive coefficients in the post-period, although there is some evidence of pre-existing trends. The remainder of the panels, however, reveal relatively flat

[42]Coefficients are shown in table form in Table 3.4.

pre-trends, consistent with our identifying assumption. In all columns in Table 3.4, we observe a sudden increase in prescriptions in the first post-treatment period, and the coefficients are statistically significant in almost all columns. The effect sizes in the year of treatment range from a 9.5 percent increase (clonazepam) to a 20.2 percent increase (lorazepam). The effect sizes are stable over time, though the coefficients in the later post-periods are statistically indistinguishable from zero. The effect sizes five years after treatment range from a 17.9 percent decrease (diazepam) to a 29.6 percent increase (temazepam) in prescriptions per 100 enrollees. Overall, these sub-figures show that there was a clear increase in prescriptions following the implementation of a MA PDMP, contrary to what we observed for stimulants and what has commonly been found for opioids.

Next, we present the results from our synthetic controls approach in Figure C.7.[43] This is important in light of the apparent pre-trends in panel (b) of the previous figure. Here, panel (a) shows that we are generally able to find synthetic controls which closely match the pre-period dynamics for each state. This panel reveals an increase of 5.61 total benzodiazepine prescriptions per 100 enrollees. Relative to a pre-period mean of 33.91, this indicates an increase of 16.5 percent. When we consider each type of benzodiazepine separately, the estimates are qualitatively similar and generally fall within the confidence intervals of the regression estimates. The only exceptions are lorazepam and temazepam, which show somewhat muted effects relative to the regression results.

3.5.1 Additional Robustness Tests and Analyses

Alternate Econometric Specifications Recent literature has highlighted that traditional difference-in-differences estimates identified on staggered treatment timing can be biased as a result of treatment effect heterogeneity (Goodman-Bacon, 2021). Likewise, Sun and Abraham (2021) show that event study difference-in-differences can suffer from a similar problem, in which treatment effect heterogeneity can induce apparent pre-trends. In this section, we

[43] Estimates shown in table form in Table C.6.

examine the robustness of our results to their proposed estimator.[44]

We present the results for stimulants in Appendix Figure C.8. The event studies shown in this figure are nearly identical to the standard event studies shown in Figure 3.2. This suggests that our findings of decreased stimulant prescribing are not an artifact of treatment effect heterogeneity. The consistent findings across this and the synthetic controls approach all point towards a reduction in stimulant prescribing as a result of MA PDMPs.

Next, we repeat this exercise for benzodiazepines and show the results in Figure C.9. Interestingly, the effects are more muted using this specification relative to Figure 3.3. The post-period point estimates are generally positive, but the magnitudes are notably smaller. In conjunction with the synthetic controls results, our overall takeaway is that there is some evidence of an increase in benzodiazepine prescribing following an MA PDMP, although the results are somewhat sensitive to alternate specifications.[45]

Additional Controls In Appendix Figures C.11–C.13, we test the robustness of our results to adding controls for other co-occurring opioid-related policies. The solid red line indicates the baseline estimates and their 95 percent confidence intervals, and the dashed blue line presents the point estimates and associated 95 percent confidence intervals obtained by adding the following controls to the baseline model (equation 3.1): (i) an indicator for whether the state has a naloxone access law (NAL), (ii) an indicator for having a Good Samaritan overdose prevention law, and (iii) an indicator for a pain management clinic law.[46] We obtain nearly identical results when we add these controls, suggesting that our results are not driven by

[44]Sun and Abraham (2021) propose the interaction-weighted estimator, which is calculated using a three step procedure. First, cohort-time specific treatment effect is estimated by using a linear two-way fixed effects specification with interactions of relative time dummies with cohort dummies (where cohort is defined based on their initial treatment timing). Second, the weights are estimated by sample shares of each cohort in a given period. Finally, the interaction-weighted estimator is estimated by taking the weighted average over all estimates for cohort-time specific effect obtained from step 1 multiplied by the weight estimates from step 2.

[45]For completeness, we also include the results for stimulant prescriptions in Medicaid in Appendix Figure C.10.

[46]The dates of these laws are taken from Prescription Drug Abuse Policy System (PDAPS). For more details, see: `https://pdaps.org/datasets/laws-regulating-administration-of-naloxone-1501695139`, `https://pdaps.org/datasets/good-samaritan-overdose-laws-1501695153`, and `https://pdaps.org/datasets/pain-management-clinic-laws`, last accessed March 7, 2022.

other opioid-related policies implemented around the time of MA PDMPs.

Mortality Given the changes in stimulant and benzodiazepine prescribing behavior documented above, a natural follow-up question is what happens to overdose deaths associated with these drugs? However, there are several factors that complicate this analysis. First, the vital statistics data do not report prescription stimulant deaths separately from other stimulant deaths. This is especially concerning given the high prevalence of methamphetamine use over our study period. Virtually all recreational methamphetamine is produced illicitly, and illicit methamphetamine use accounts for at least 85 to 90 percent of stimulant overdose deaths (Drug Enforcement Agency, 2018).[47] Since MA PDMPs do not directly affect illicit methamphetamine production, this biases us against detecting any mortality changes.[48]

We run into similar complications when examining benzodiazepine overdose deaths. Specifically, overdose deaths solely from benzos are incredibly rare. Almost all benzodiazepine deaths involve other drugs, in particular depressants such as opioids. Since opioid availability is directly affected by MA PDMPs, and moves in the opposite direction as benzodiazepine prescriptions, this makes it difficult to interpret any changes in benzodiazepine overdose deaths. In results not presented here, we separately estimate the effects of MA PDMPs on benzodiazepine overdose deaths that include as well as exclude opioid use, and find somewhat conflicting results. We observe a slight increase in total benzodiazepine-involved mortality, but a decrease in benzodiazepine-only mortality, although both sets of results exhibit notable pre-trends, making it difficult to draw strong conclusions.

3.6 Conclusion

Prescription drug monitoring programs have emerged as one of the key tools that policy makers have used to combat surging drug overdose death rates. A rapidly growing literature has examined the effectiveness of PDMPs on opioid prescribing, misuse, and overdose deaths.

[47]The ICD-10 code for these deaths is T43.6, "psychostimulants with abuse potential."

[48]In results not presented here, we examine deaths related to stimulants and find no effects within four years of a MA PDMP being implemented.

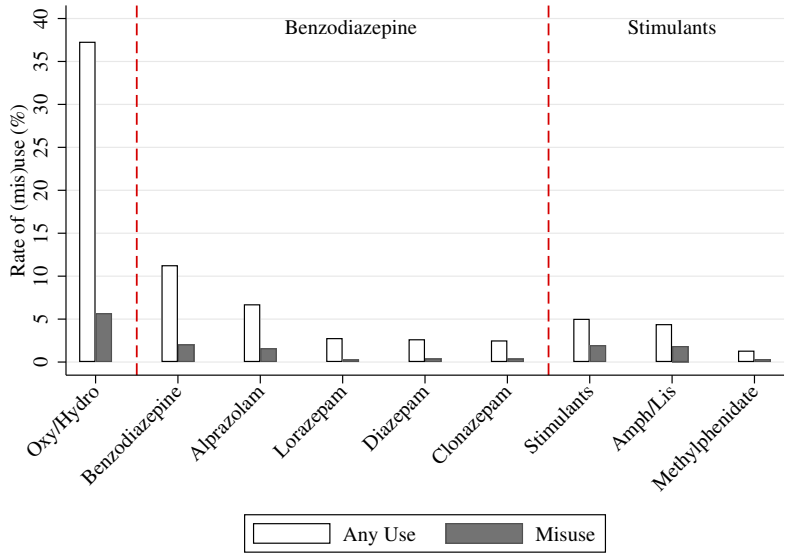
Other work has considered downstream effects including heroin-related crime and overdose deaths, as well as labor market conditions. However, the literature considering the effect of these programs on the consumption of other drugs is still limited.

In this paper, we expand upon this literature by considering how mandatory access PDMPs have affected the consumption of prescription stimulants and benzos. Using a variety of econometric specifications, we find robust evidence that MA PDMPs led to decreases in the availability of prescription stimulants. In contrast, we find some evidence of increased consumption of benzos, although these findings are more sensitive to different empirical specifications.

Our paper highlights two important aspects of mandatory access PDMPs. First, we show that PDMPs have effects on non-opioid drugs. These effects exist even for drugs that are not explicitly included in the PDMP. Next, this paper shows that the effects differ across drug types. We find qualitatively different responses for stimulants and benzodiazepines. This is consistent with important interaction effects and substitution patterns across drug types.

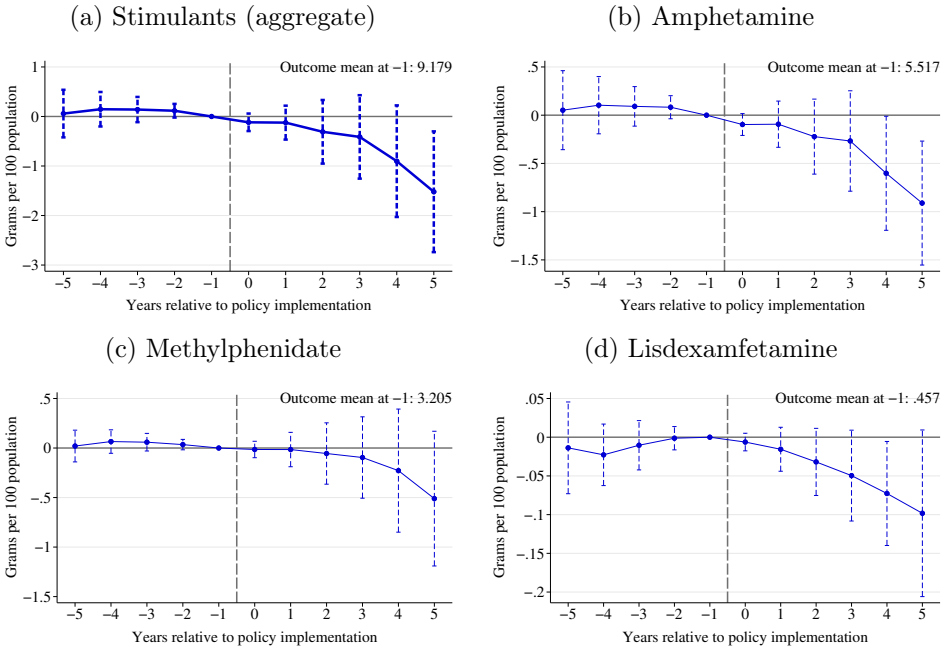
3.7 Figures and Tables

Figure 3.1: Benzodiazepine and Stimulant Use and Misuse



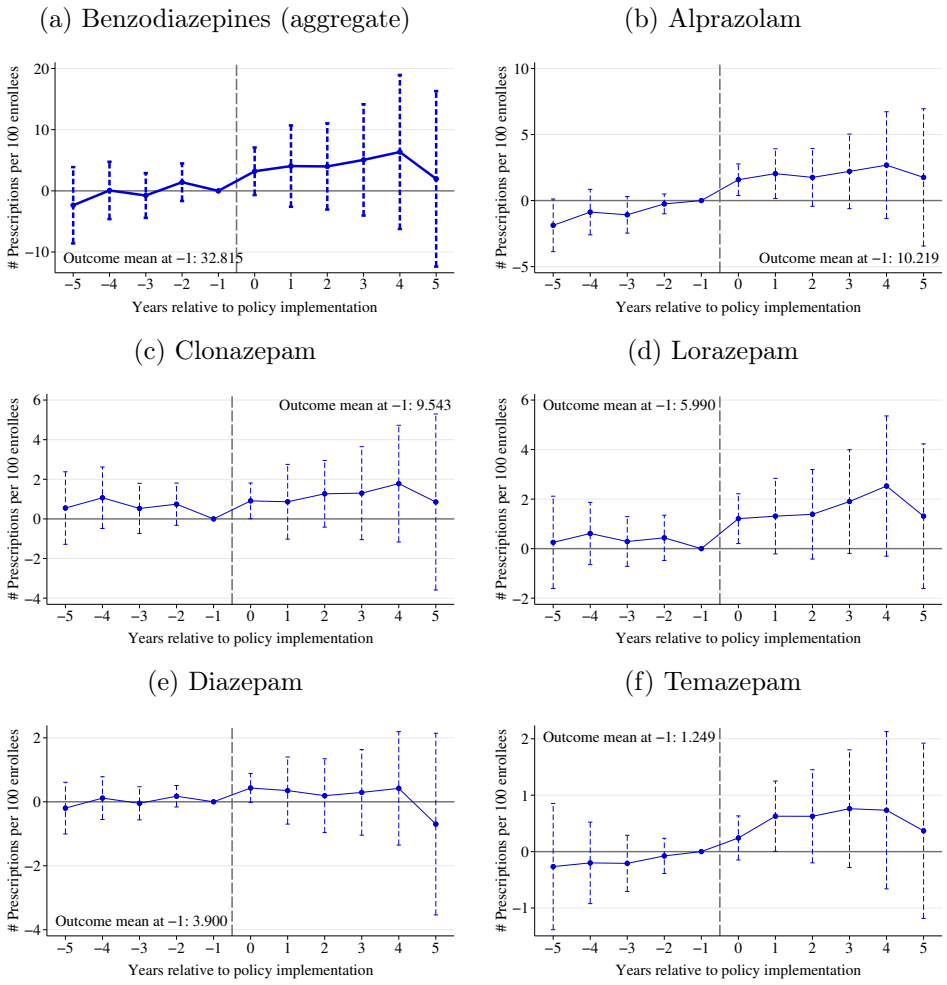
Notes: The figure presents the prevalence of use and misuse of prescription benzodiazepines and stimulants among 2015 and 2016 NSDUH respondents over age 12 (N=114,043).

Figure 3.2: Effects of MA PDMPs on Stimulant Distribution (ARCOS Data)



Notes: These figures present the coefficients and 95% confidence intervals on the interactions between the indicator for treated states and the indicators for each of the years before and after policy implementation obtained from estimation of equation (3.1). The year before the policy implementation is the omitted category. The regressions include state and year fixed effects. Although each regression includes a full set of indicators for event time periods -9 through 5, we only present estimates for event time periods -5 through 5 for brevity. Standard errors are clustered at the state level. Each dependent variable is measured in amphetamine-equivalent grams per 100 population.

Figure 3.3: Effects of MA PDMPs on Benzodiazepine Prescribing (Medicaid Data)



Notes: These figures present the coefficients and 95% confidence intervals on the interactions between the indicator for treated states and the indicators for each of the years before and after policy implementation obtained from estimation of equation (3.1). The year before the policy implementation is the omitted category. The regressions include state and year fixed effects. Although each regression includes a full set of indicators for event time periods -9 through 5, we only present estimates for event time periods -5 through 5 for brevity. Standard errors are clustered at the state level. Each dependent variable is the number of prescriptions per 100 Medicaid enrollees.

Table 3.1: State Laws

State	Effective Date
Alabama	
Alaska	2017m7
Arizona	2017m10
Arkansas	2017m1
California	2018m4
Colorado	
Connecticut	2015m10
Delaware	2012m3
District of Columbia	
Florida	
Georgia	2014m7
Hawaii	
Idaho	
Illinois	2018m1
Indiana	2014m7
Iowa	
Kansas	
Kentucky	2012m7
Louisiana	2008m1
Maine	
Maryland	2018m7
Massachusetts	2014m7
Michigan	
Minnesota	2017m1
Mississippi	
Missouri	
Montana	
Nebraska	
Nevada	2007m10
New Hampshire	2016m1
New Jersey	2015m11
New Mexico	2012m9
New York	2013m8
North Carolina	
North Dakota	
Ohio	2012m3
Oklahoma	2011m3
Oregon	
Pennsylvania	2017m1
Rhode Island	2016m6
South Carolina	2017m5
South Dakota	
Tennessee	2013m7
Texas	
Utah	2017m5
Vermont	2015m5
Virginia	2015m7
Washington	
West Virginia	2012m6
Wisconsin	
Wyoming	

Notes: This table reports the start dates of state laws enacted until December 31, 2018. The dates are obtained from Sacks et al. (2021).

Table 3.2: Summary Statistics

	All States		Treated States		Control States	
	Mean	SD	Mean	SD	Mean	SD
Outcome (mean, 2008–2017)	(1)	(2)	(3)	(4)	(5)	(6)
The legal supply of stimulants (amphetamine equivalent grams) per 100 population						
Stimulants (aggregate)	8.543	(2.538)	8.637	(2.632)	8.435	(2.429)
Amphetamine	5.041	(1.856)	5.075	(1.913)	5.002	(1.792)
Methylphenidate	3.091	(0.870)	3.171	(0.878)	3.001	(0.854)
Lisdexamfetamine	0.410	(0.197)	0.392	(0.203)	0.431	(0.188)
The number of prescriptions per 100 Medicaid enrollees						
Stimulants (aggregate)	19.790	(9.629)	18.560	(9.593)	21.505	(9.441)
Amphetamine	5.249	(3.713)	4.952	(3.523)	5.635	(3.923)
Methylphenidate	9.197	(4.426)	9.134	(4.461)	9.280	(4.390)
Lisdexamfetamine	4.947	(3.645)	4.332	(3.525)	5.809	(3.645)
Benzodiazepine (aggregate)	32.936	(17.460)	33.895	(17.250)	31.745	(17.688)
Alprazolam	10.542	(6.341)	10.406	(5.950)	10.716	(6.817)
Clonazepam	9.259	(5.193)	9.918	(5.574)	8.393	(4.515)
Lorazepam	6.757	(4.890)	6.766	(5.025)	6.745	(4.720)
Diazepam	3.972	(2.426)	3.947	(2.535)	4.004	(2.283)
Temazepam	1.617	(1.601)	1.407	(1.316)	1.878	(1.866)
Age and race/ethnicity compositions						
0–14	0.197	(0.018)	0.195	(0.016)	0.200	(0.019)
15–24	0.139	(0.007)	0.139	(0.006)	0.139	(0.007)
25–44	0.261	(0.014)	0.260	(0.011)	0.262	(0.017)
45–64	0.261	(0.015)	0.264	(0.015)	0.257	(0.014)
65–84	0.123	(0.018)	0.123	(0.014)	0.123	(0.022)
85+	0.019	(0.004)	0.019	(0.004)	0.018	(0.004)
Non-Hispanic White	0.667	(0.135)	0.691	(0.115)	0.640	(0.151)
Non-Hispanic Black	0.130	(0.077)	0.134	(0.074)	0.125	(0.080)
Hispanic	0.142	(0.112)	0.113	(0.084)	0.175	(0.130)
Observations	460		240		220	
Number of states	46		24		22	

Notes: This table presents average characteristics for all states (columns 1–2), treated states (columns 3–4), and control states (columns 5–6) included in our baseline analysis. The table reports the mean and standard deviation. Each panel describes the balanced panel of state-years from 2008 to 2017. For stimulant supply outcomes and state demographic characteristics, observations are weighted by state population. For Medicaid prescribing outcomes, observations are weighted by Medicaid enrollment. The first two columns include all states, columns 3–4 includes the 24 treated states that implemented a MA PDMP between 2009–2017. The last two columns include the 22 control states that did not implement a MA PDMP until December 2018.

Table 3.3: Effects of MA PDMPs on Stimulant Distribution

	Aggregate (1)	Amphetamine (2)	Methyl. (3)	Lisdexamf. (4)
Dependent variable: amphetamine equivalent stimulant grams per 100 population				
Immediate effect	-0.12	-0.10*	-0.01	-0.01
	(0.09)	(0.06)	(0.04)	(0.01)
1-year effect	-0.12	-0.09	-0.02	-0.02
	(0.17)	(0.12)	(0.09)	(0.01)
2-year effect	-0.31	-0.22	-0.06	-0.03
	(0.32)	(0.19)	(0.15)	(0.02)
3-year effect	-0.41	-0.27	-0.10	-0.05*
	(0.42)	(0.26)	(0.20)	(0.03)
4-year effect	-0.90	-0.60**	-0.23	-0.07**
	(0.56)	(0.29)	(0.31)	(0.03)
5-year effect	-1.52**	-0.91***	-0.51	-0.10*
	(0.61)	(0.32)	(0.34)	(0.05)
State fixed effects	Y	Y	Y	Y
Year fixed effects	Y	Y	Y	Y
Time-varying covariates	Y	Y	Y	Y
Mean at -1	9.179	5.517	3.205	0.457
Observations	459	459	459	459
R^2	0.969	0.963	0.961	0.952

Notes: This table shows the immediate effect, 1-year effect, 2-year effect, 3-year effect, 4-year effect, and 5-year effect from equation (3.1). Although each regression includes a full set of indicators for pre- and post-periods, we only report the coefficients on the post-periods above for brevity. The year before the policy implementation is the omitted category. Observations are weighted by state population. In column (1), the dependent variable is aggregate amphetamine equivalent stimulant grams per 100 population. In columns (2)-(4), the dependent variables are the amphetamine equivalent grams of amphetamine, methylphenidate, and lisdexamfetamine per 100 population, respectively. We include state and year fixed effects as well as time-varying covariates (age and race compositions) in each regression. The mean of each dependent variable is calculated using observations from the treated sample measured at the last pre-policy period. Standard errors are clustered at the state level are in parentheses. ***, **, * denotes statistical significance at 1%, 5%, and 10% levels, respectively.

Table 3.4: Effects of MA PDMPs on Benzodiazepine Prescribing

	Aggregate	Alprazolam	Clonazepam	Lorazepam	Diazepam	Temazepam
	(1)	(2)	(3)	(4)	(5)	(6)
Dependent variable: Number of benzodiazepine prescriptions per 100 enrollees						
Immediate effect	3.20	1.58**	0.91**	1.21**	0.43*	0.24
	(1.93)	(0.59)	(0.45)	(0.50)	(0.23)	(0.19)
1-year effect	4.05	2.04**	0.86	1.31*	0.35	0.63**
	(3.30)	(0.93)	(0.94)	(0.76)	(0.52)	(0.31)
2-year effect	3.99	1.75	1.27	1.39	0.19	0.63
	(3.50)	(1.09)	(0.84)	(0.90)	(0.57)	(0.41)
3-year effect	5.06	2.21	1.30	1.90*	0.29	0.76
	(4.51)	(1.40)	(1.16)	(1.04)	(0.66)	(0.52)
4-year effect	6.35	2.68	1.78	2.53*	0.42	0.73
	(6.24)	(2.01)	(1.46)	(1.41)	(0.88)	(0.69)
5-year effect	1.96	1.75	0.86	1.31	-0.70	0.37
	(7.12)	(2.58)	(2.21)	(1.45)	(1.41)	(0.77)
State fixed effects	Y	Y	Y	Y	Y	Y
Year fixed effects	Y	Y	Y	Y	Y	Y
Time-varying covariates						
Mean at -1	32.815	10.219	9.543	5.990	3.900	1.249
Observations	431	443	450	449	448	433
R^2	0.848	0.833	0.838	0.868	0.826	0.815

Notes: This table shows the immediate effect, 1-year effect, 2-year effect, 3-year effect, 4-year effect, and 5-year effect from equation (3.1). Although each regression includes a full set of indicators for pre- and post-periods, we report the coefficients on the post-periods above for brevity. The year before the policy implementation is the omitted category. Observations are weighted by the number of Medicaid enrollees. In column (1), the dependent variable is the total number of benzodiazepine prescriptions per 100 Medicaid enrollees. In columns (2)–(6), we examine each type of benzodiazepine separately. The regressions include state and year fixed effects, as well as time-varying covariates (age and race compositions). The mean of each dependent variable is calculated using observations from the treated sample measured at the last pre-policy period. Standard errors clustered at the state level are in parentheses. ***, **, * denotes statistical significance at 1%, 5%, and 10% levels, respectively.

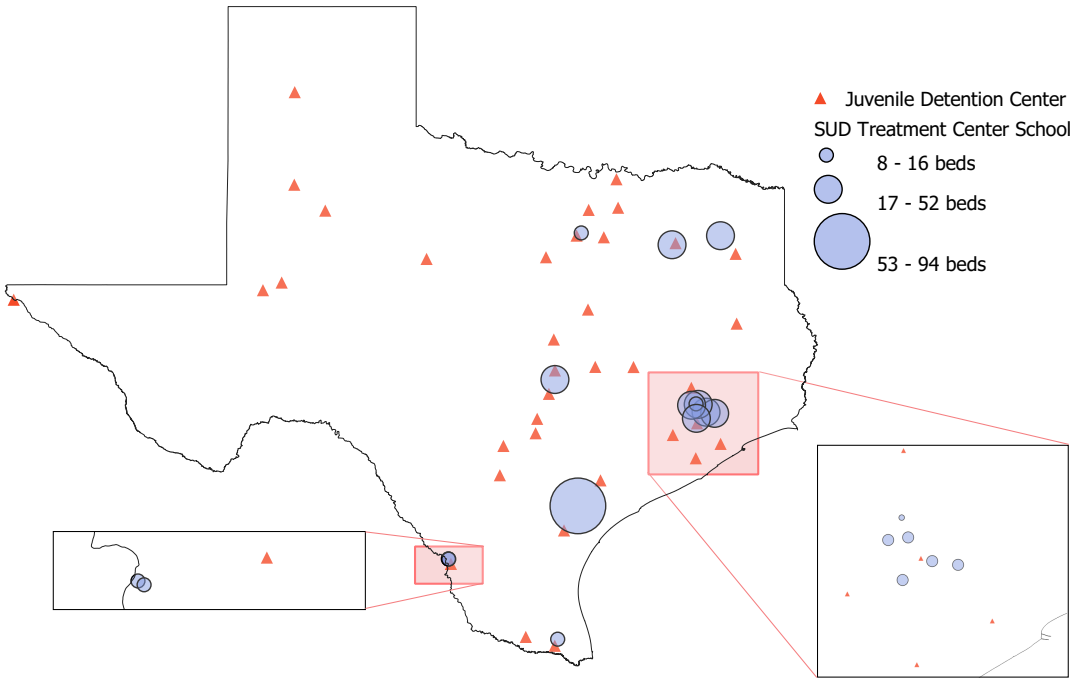
Appendices

Appendix A

Appendix to Chapter 1

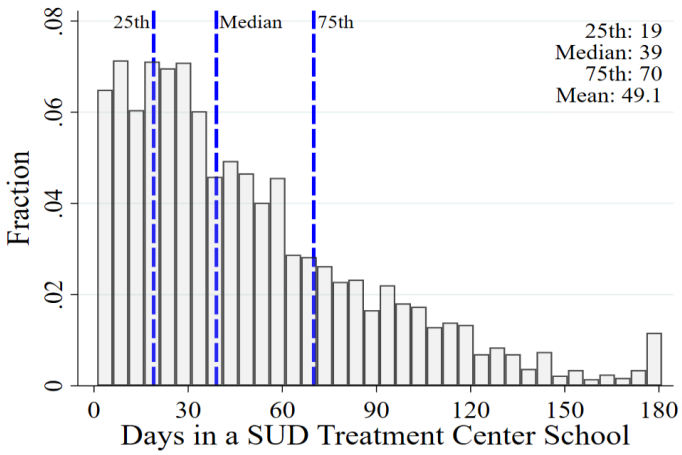
A.1 Appendix Figures

Figure A.1: Location of Treatment Center Schools and the Number of Beds



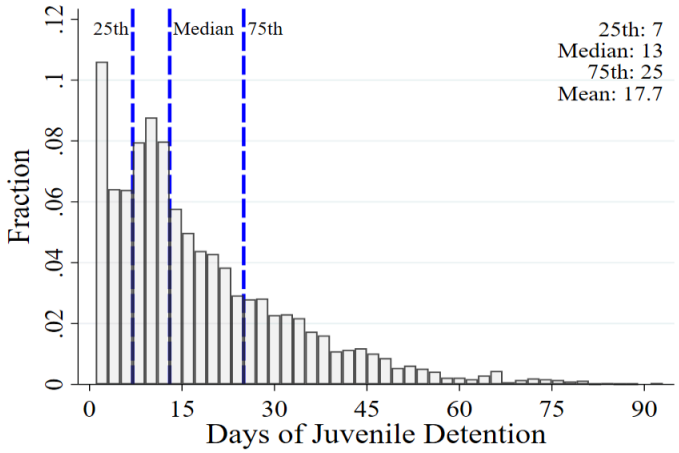
Notes: The figure presents the location of 14 treatment center schools and juvenile detention centers that are included in my analysis. Note that the map only presents juvenile detention centers that are active between 2019 and 2020.

Figure A.2: Distribution of Days in a SUD Treatment Center School



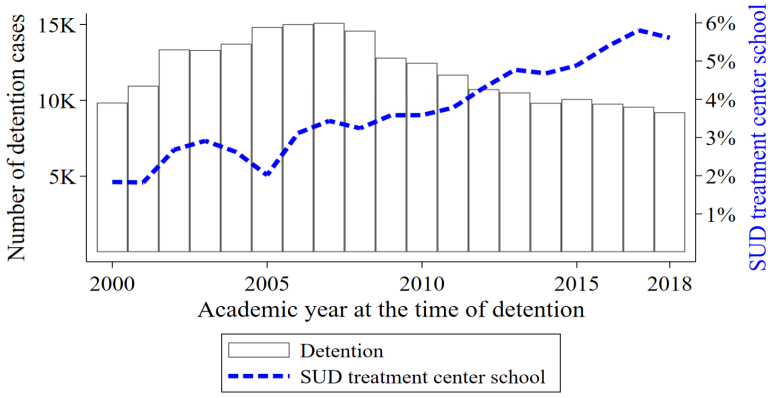
Notes: The figure presents the distribution of the length of stay within a SUD treatment center school among my analysis sample.

Figure A.3: Distribution of Days in a Juvenile Detention Center



Notes: The figure presents the distribution of the length of juvenile detention among my analysis sample.

Figure A.4: Number of Juvenile Detention Cases and Share of Cases Entering a SUD Treatment Center School

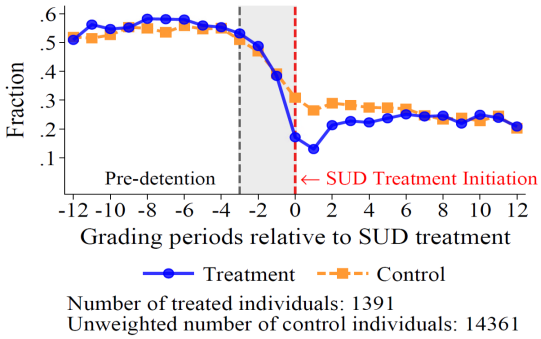


Unique individuals entering a SUD treatment center school: 6,302

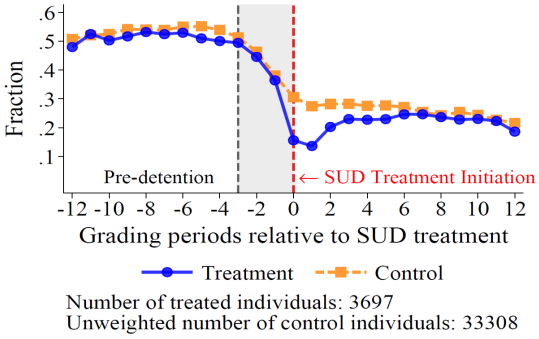
Notes: The figure presents trends in the number juvenile detention cases and the percentage of cases in which a juvenile enters a SUD treatment center school within a year following detention.

Figure A.5: Any Disciplinary Action: Sample with Substance-related Discipline History vs. Full Sample

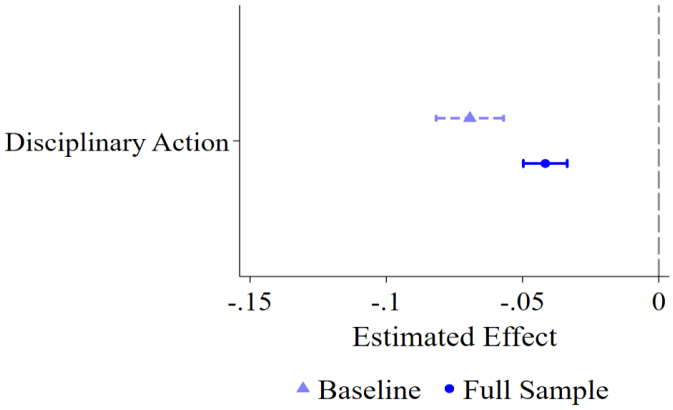
(a) Raw Plot: Individuals with Substance-related Discipline History



(b) Raw Plot: Full sample

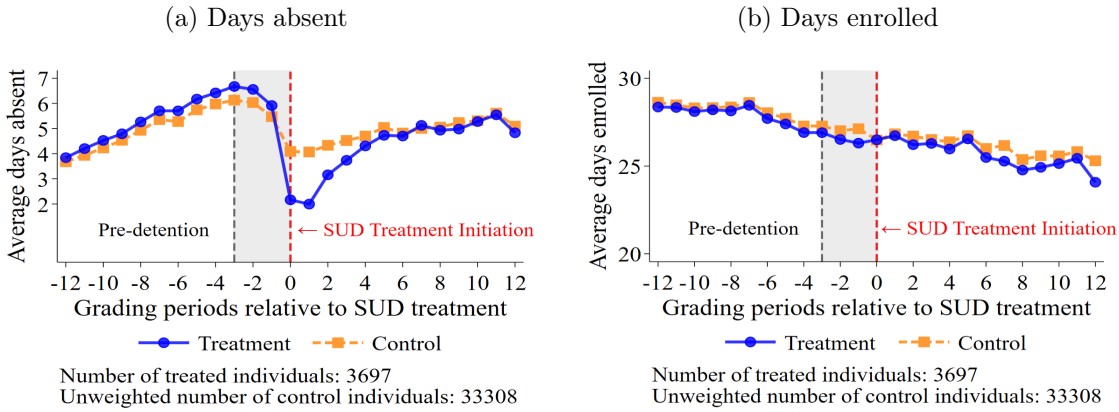


(c) Effect on Disciplinary Action: Individuals with Substance-related Discipline History vs. Full Sample



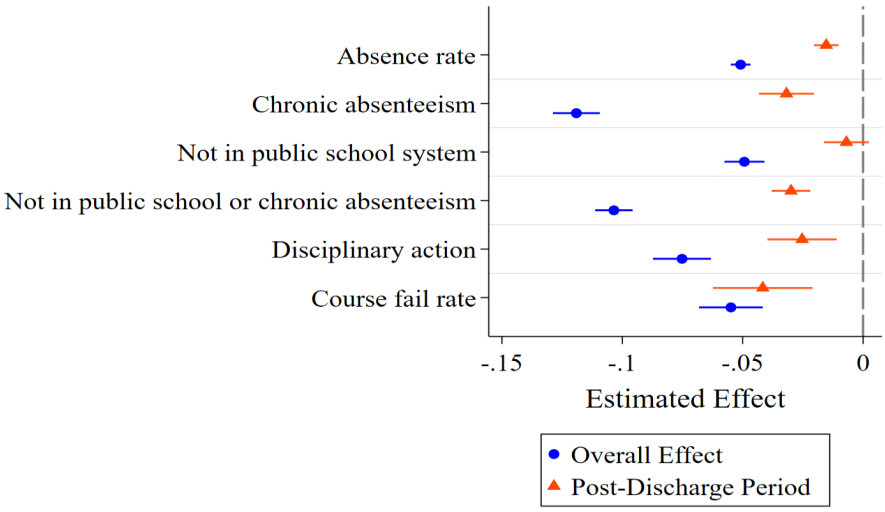
Notes: The figure compares raw data trends and regression results across individuals with substance-related discipline history and the full sample—individuals with and without such history.

Figure A.6: Raw Trends in Days Absent and Days Enrolled in Public School or Juvenile Detention Centers



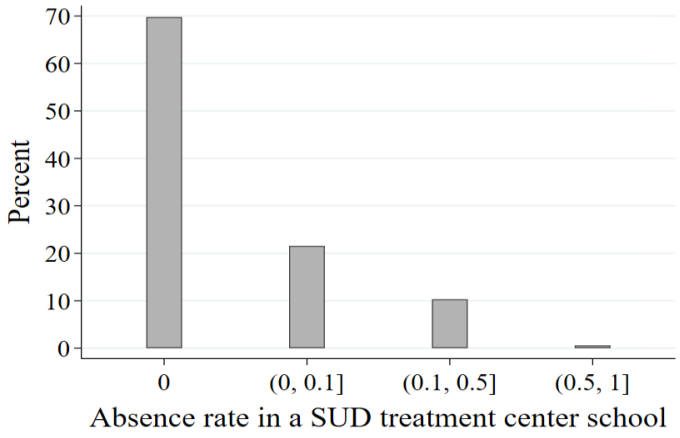
Notes: The figure plots raw data trends and event study results. I present raw data trends in the outcomes from 12 six-week grading periods before (i.e., about two academic years) to 13 grading periods after the time of SUD treatment initiation, separately for treated and matched control individuals.

Figure A.7: Impacts of SUD Treatment Center School Attendance on Short-Run Outcomes: Post-Discharge Period



Notes: The figure presents the impact of SUD treatment center school attendance on short-run outcomes during the post-discharge period.

Figure A.8: Distribution of Absence Rate in a SUD Treatment Center School

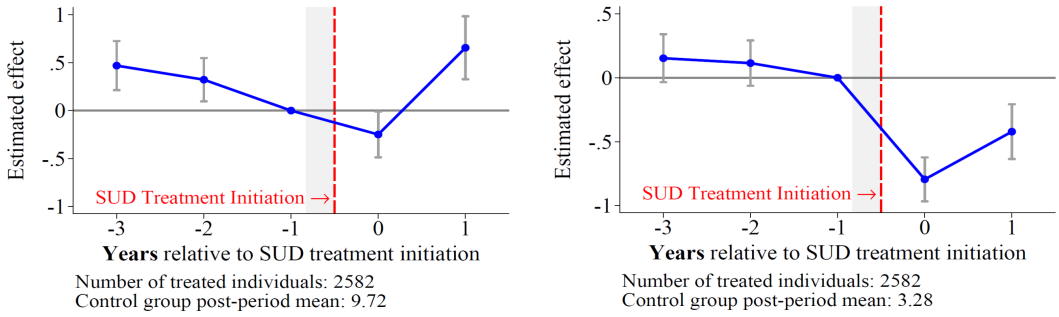


Notes: The figure presents the distribution of absence rate within a treatment center school measured as the total days absent from a SUD treatment center school relative to the days enrolled in the same center school. For almost 70 percent of the treatment individuals in my sample, the absence rate within a SUD treatment center school is zero, but the other 30 percent are absent from a SUD center school for at least one school day.

Figure A.9: Impacts on Courses Taken and Failed

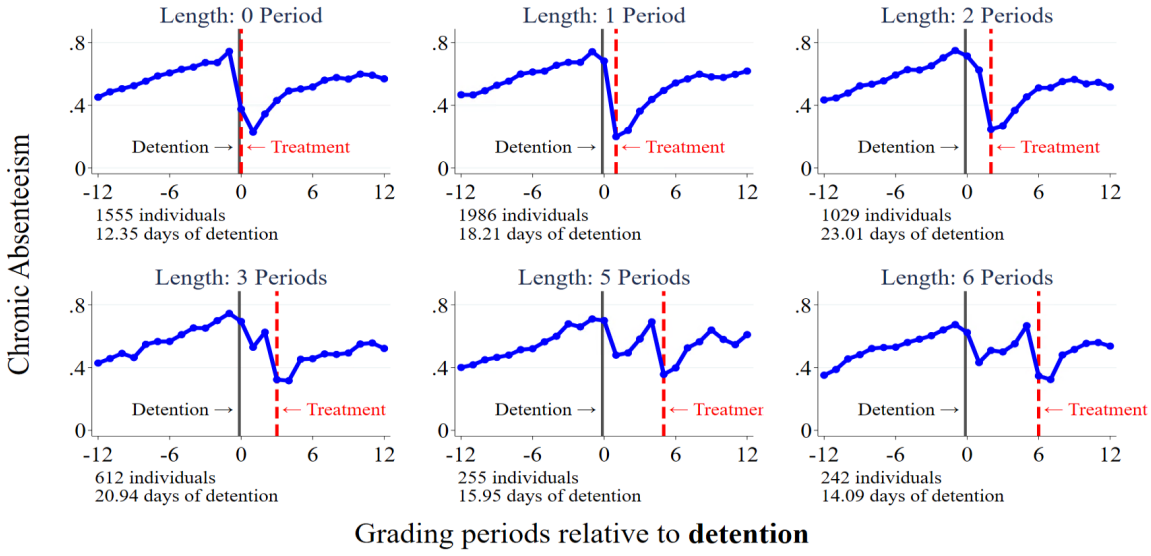
(a) Number of Courses Taken

(b) Number of Courses Failed



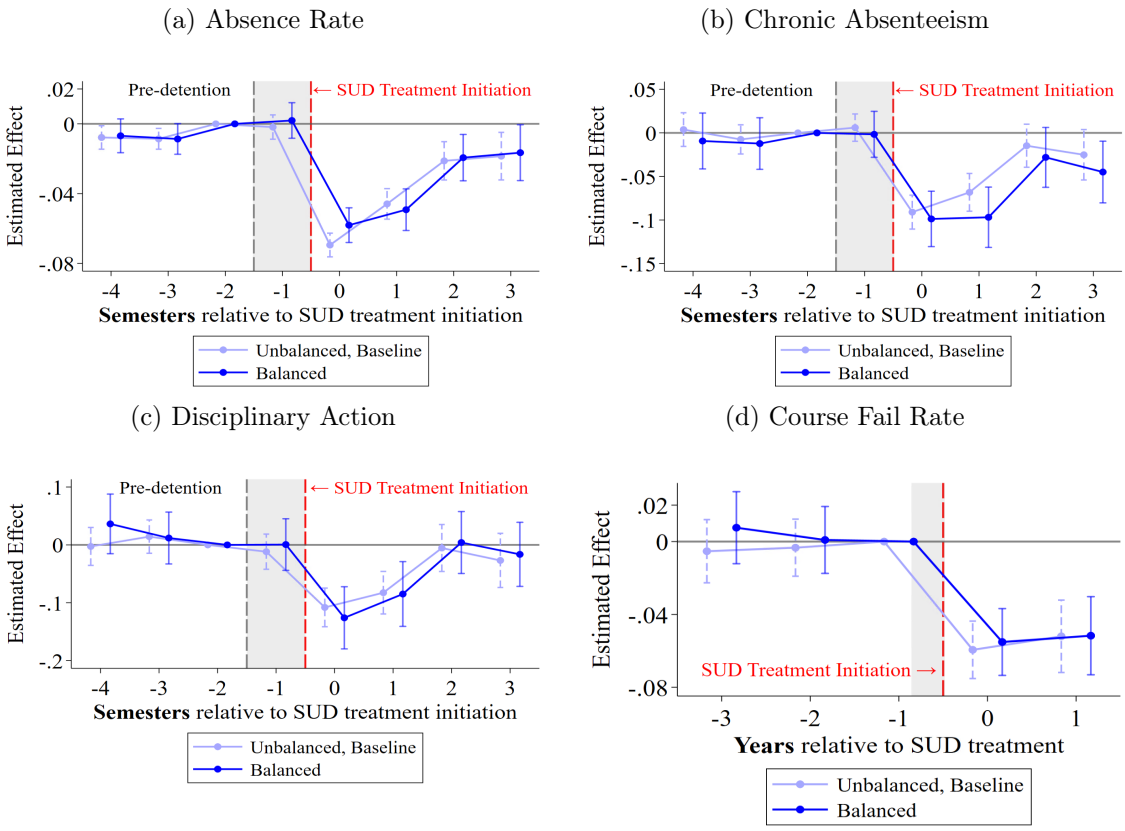
Notes: The figure shows the event study results using a balanced sample. I plot the coefficients and 95% confidence intervals on the interactions between the indicator for a treatment individual and the indicators for the years around the time of SUD treatment initiation from equation (1.2). Standard errors are clustered at the individual level.

Figure A.10: Raw Trends in Chronic Absenteeism by the Length of Intermediate Pre-Period



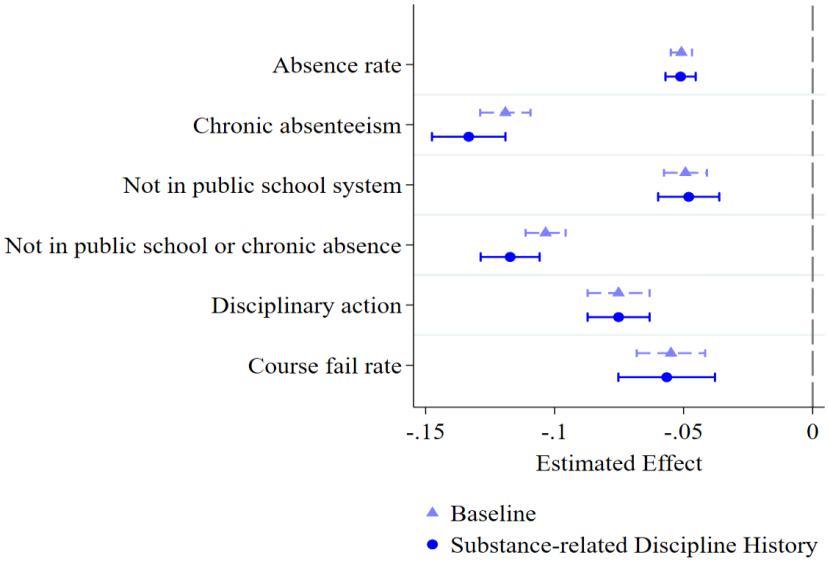
Notes: The figure plots raw trends in the likelihood of chronic absenteeism separately for six sub-groups that are defined based on the length of the intermediate pre-period.

Figure A.11: Short-Run Effects of SUD Treatment Center Schools: Unbalanced and Balanced Sample



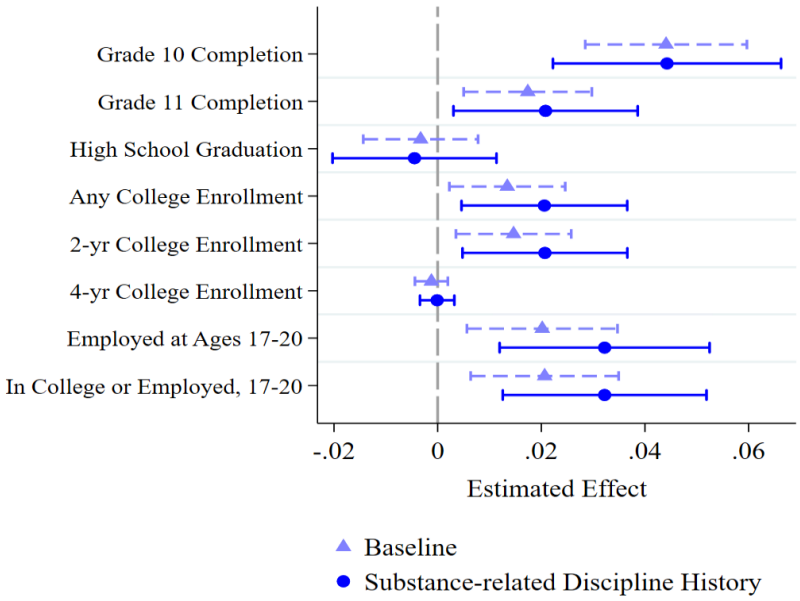
Notes: The figure shows the event study results using a balanced sample. I plot the coefficients and 95% confidence intervals on the interactions between the indicator for a treatment individual and the indicators for the periods around the time of SUD treatment initiation from equation (1.2). Standard errors are clustered at the individual level.

Figure A.12: Robustness of Short-Run Analysis Results: Restricting Sample to Adolescents Disciplined for Substance-related Reasons in the Pre-Detention Period



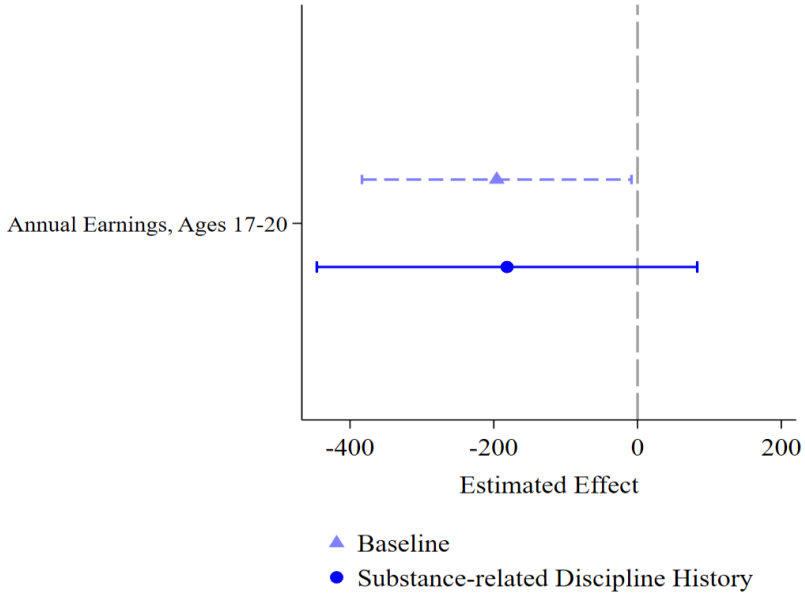
Notes: The figure shows the short-run analysis results by only including match groups in which both the treatment and matched control individuals were ever disciplined for substance-related reasons prior to detention.

Figure A.13: Robustness of Long-Run Analysis Results: Restricting Sample to Adolescents Disciplined for Substance-related Reasons in the Pre-Detention Period



Notes: The figure shows the long-run analysis results obtained when I only include match groups where both the treatment and matched control individuals were ever disciplined for substance-related reasons prior to detention.

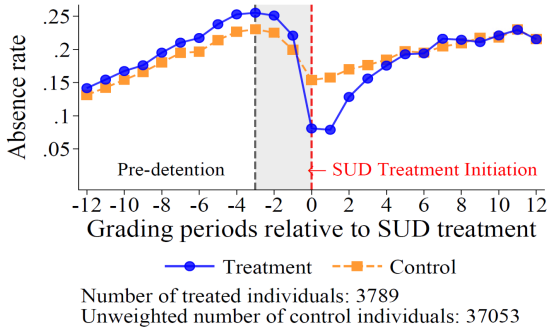
Figure A.14: Robustness of Long-Run Analysis Earnings Results: Restricting Sample to Adolescents Disciplined for Substance-related Reasons in the Pre-Detention Period



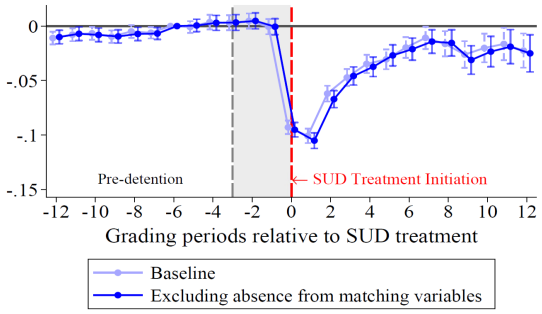
Notes: The figure shows the long-run analysis results obtained when I only include match groups where both the treatment and matched control individuals were ever disciplined for substance-related reasons prior to detention.

Figure A.15: Robustness of Absence Results: Excluding Absence from the Matching

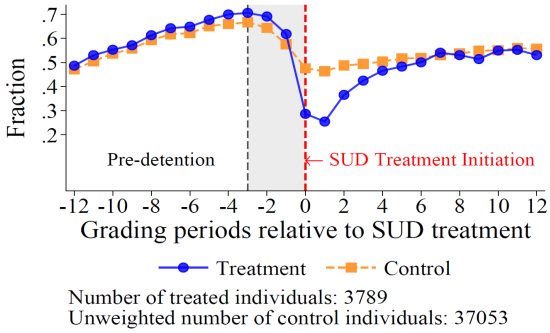
(a) Absence Rate: Raw Trends



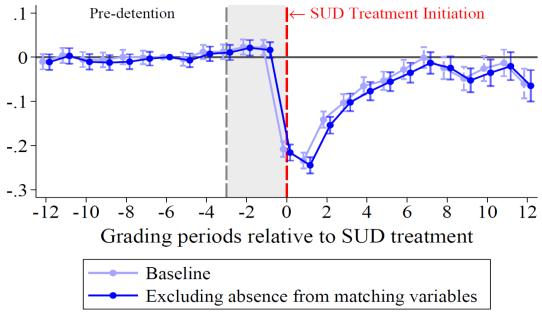
(b) Absence Rate: Event Study



(c) Chronic Absenteeism: Raw Trends



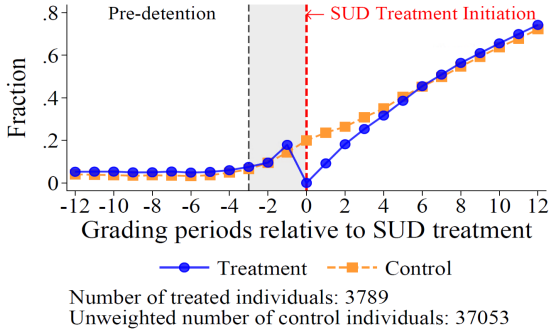
(d) Chronic Absenteeism: Event Study



Notes: The figure shows raw data trends and the short-run analysis results obtained when I exclude absence from the fuzzy matching.

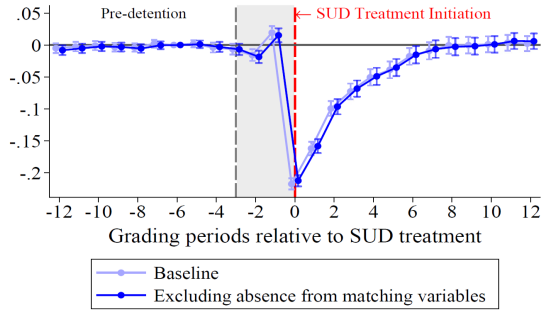
Figure A.16: Robustness of Schooling Results: Excluding Absence from the Matching

(a) Not in Public School System: Raw Trends

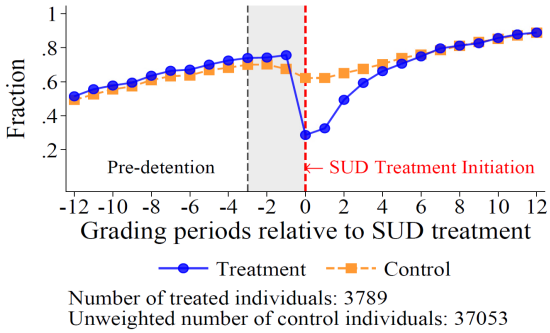


Number of treated individuals: 3789
Unweighted number of control individuals: 37053

(b) Not in Public School System: Event Study

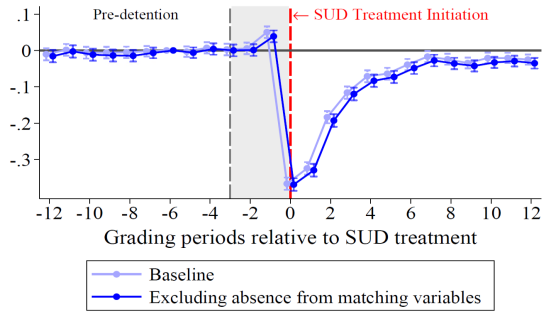


(c) Not in Public School Or Chronic Absenteeism: Raw Trends



Number of treated individuals: 3789
Unweighted number of control individuals: 37053

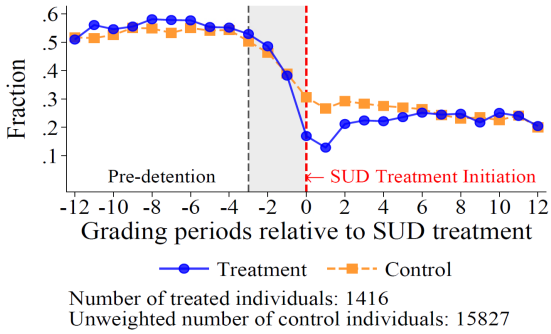
(d) Not in Public School Or Chronic Absenteeism: Event Study



Notes: The figure shows raw data trends and the short-run analysis results obtained when I exclude absence from the fuzzy matching.

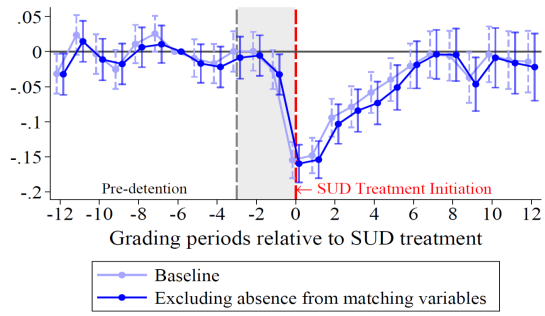
Figure A.17: Robustness of Disciplinary Action Results: Excluding Absence from the Matching

(a) Any Disciplinary Action: Raw Trends



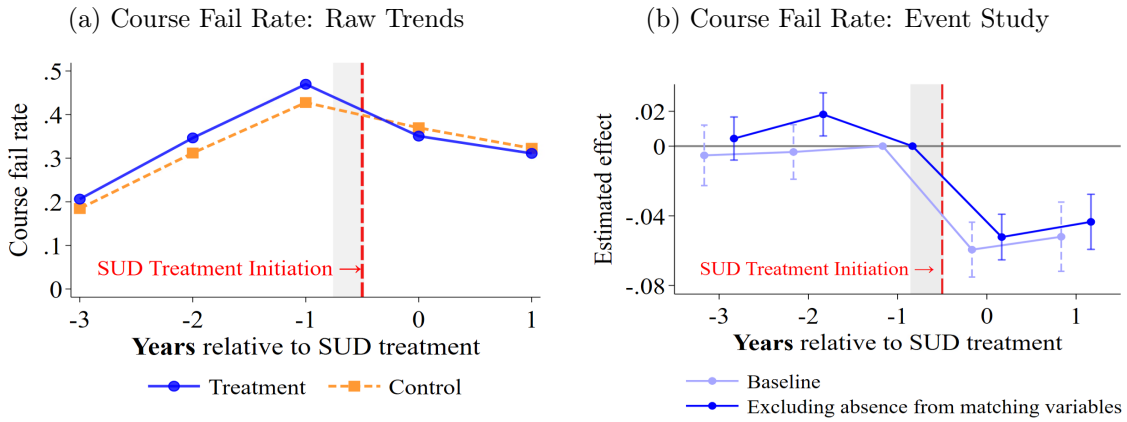
Number of treated individuals: 1416
Unweighted number of control individuals: 15827

(b) Any Disciplinary Action: Event Study



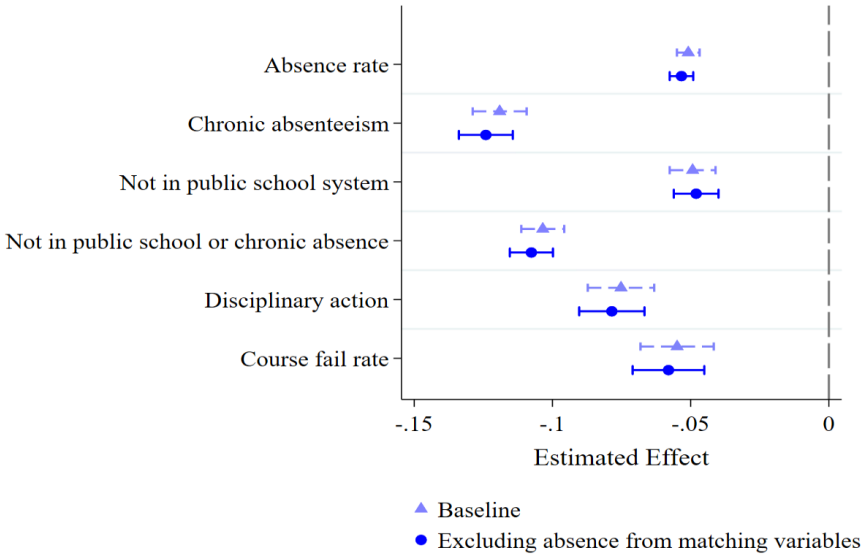
Notes: The figure shows raw data trends and the short-run analysis results obtained when I exclude absence from the fuzzy matching.

Figure A.18: Robustness of Course Fail Rate Results: Excluding Absence from the Matching



Notes: The figure shows raw data trends and the short-run analysis results obtained when I exclude absence from the fuzzy matching.

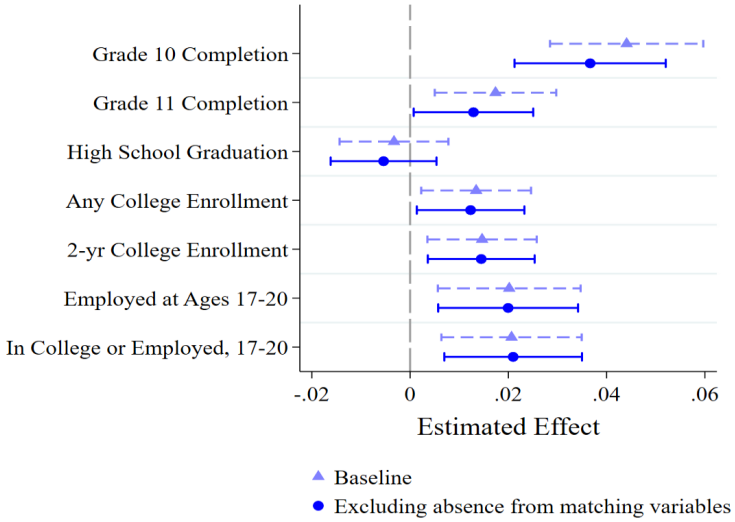
Figure A.19: Robustness of of Short-Run Analysis Results: Excluding Absence from the Matching



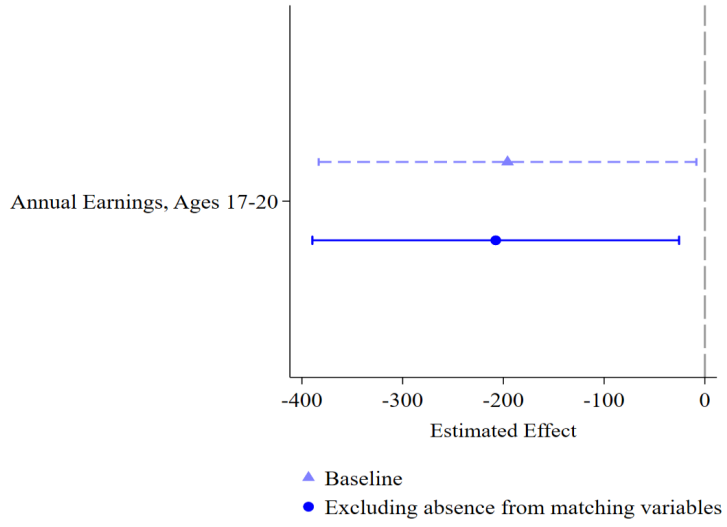
Notes: The figure shows the short-run analysis results obtained when I exclude absence from the fuzzy matching.

Figure A.20: Robustness of Long-Run Analysis Results: Excluding Absence from the Matching

(a) Educational Outcomes and Employment



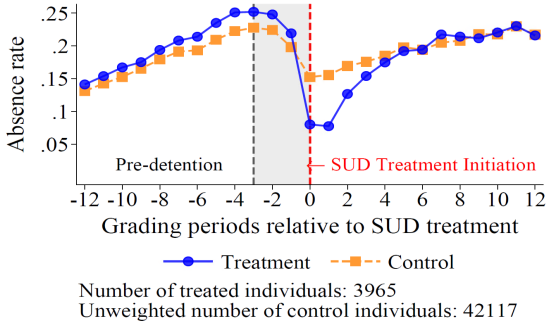
(b) Earnings



Notes: The figure shows the long-run analysis results obtained when I exclude absence from the fuzzy matching.

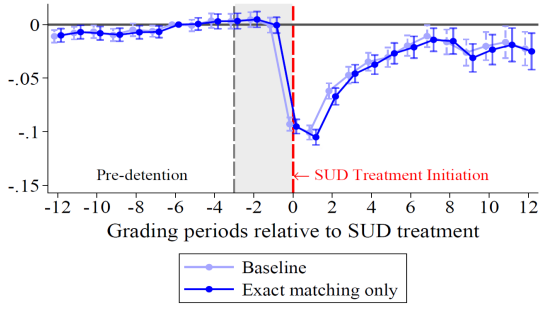
Figure A.21: Robustness of Absence Results: Exact Matching Only

(a) Absence Rate: Raw Trends

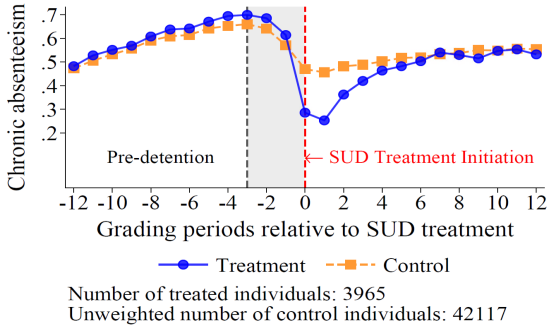


Number of treated individuals: 3965
Unweighted number of control individuals: 42117

(b) Absence Rate: Event Study

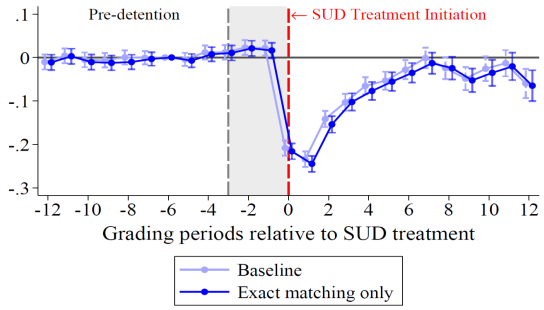


(c) Chronic Absenteeism: Raw Trends



Number of treated individuals: 3965
Unweighted number of control individuals: 42117

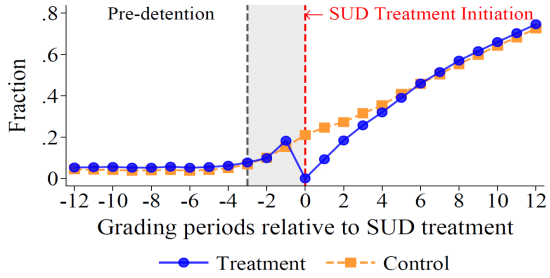
(d) Chronic Absenteeism: Event Study



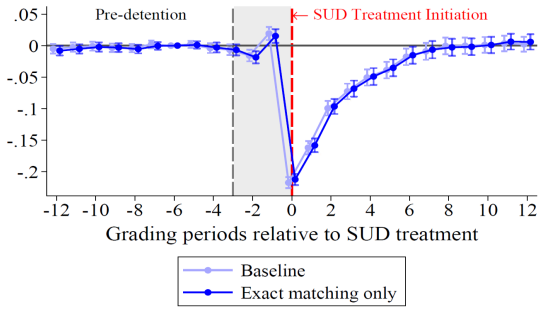
Notes: The figure shows raw data trends and the short-run analysis results obtained when I do the exact matching omitting the fuzzy matching.

Figure A.22: Robustness of Schooling Results: Exact Matching Only

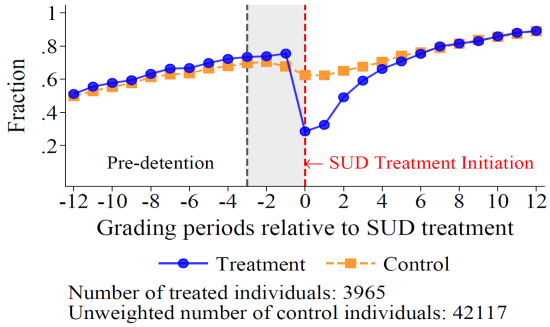
(a) Not in Public School System: Raw Trends



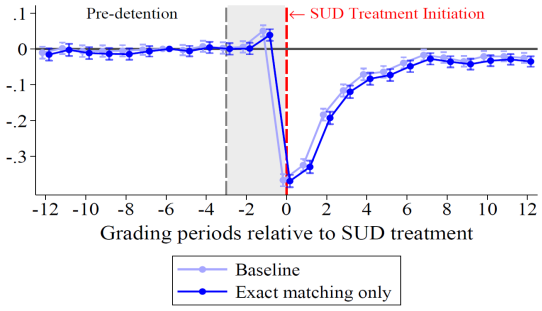
(b) Not in Public School System: Event Study



(c) Not in Public School Or Chronic Absenteeism: Raw Trends



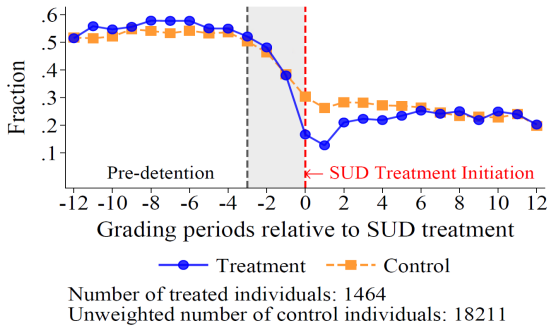
(d) Not in Public School Or Chronic Absenteeism: Event Study



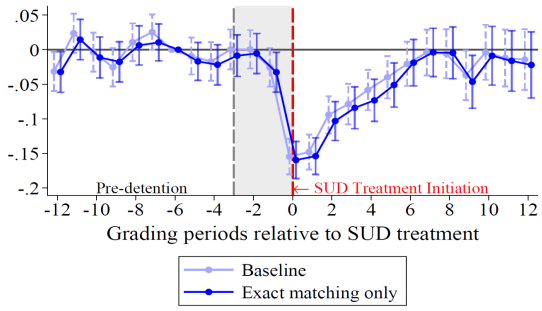
Notes: The figure shows raw data trends and the short-run analysis results obtained when I do the exact matching omitting the fuzzy matching.

Figure A.23: Robustness of Disciplinary Action Results: Exact Matching Only

(a) Any Disciplinary Action: Raw Trends

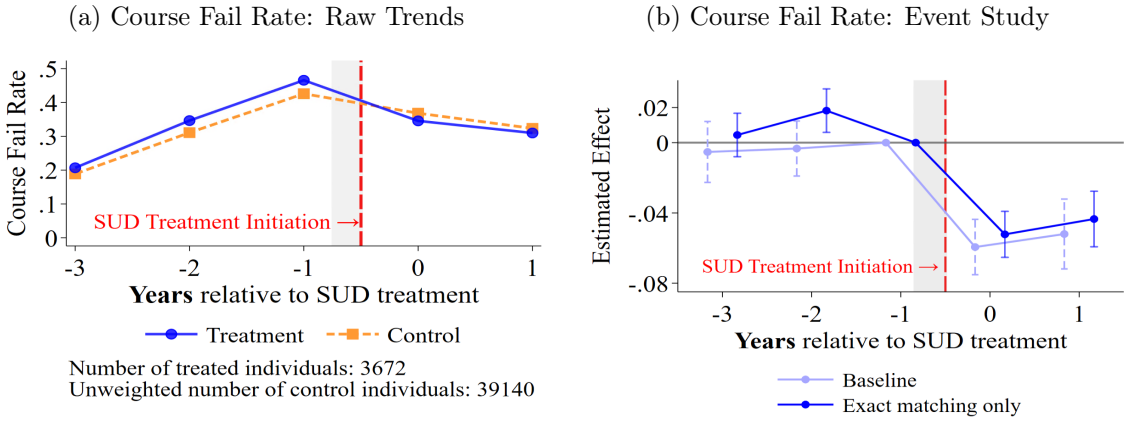


(b) Any Disciplinary Action: Event Study



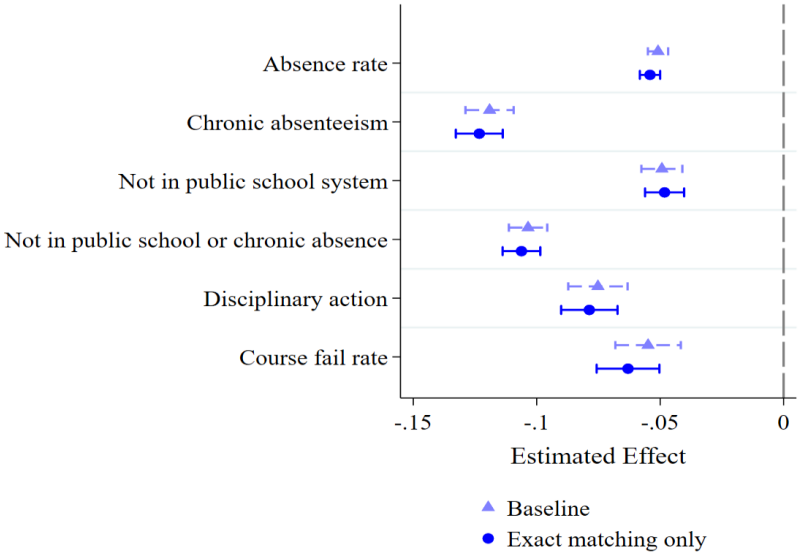
Notes: The figure shows raw data trends and the short-run analysis results obtained when I do the exact matching omitting the fuzzy matching.

Figure A.24: Robustness of Course Fail Rate Results: Exact Matching Only



Notes: The figure shows raw data trends and the short-run analysis results obtained when I do the exact matching omitting the fuzzy matching.

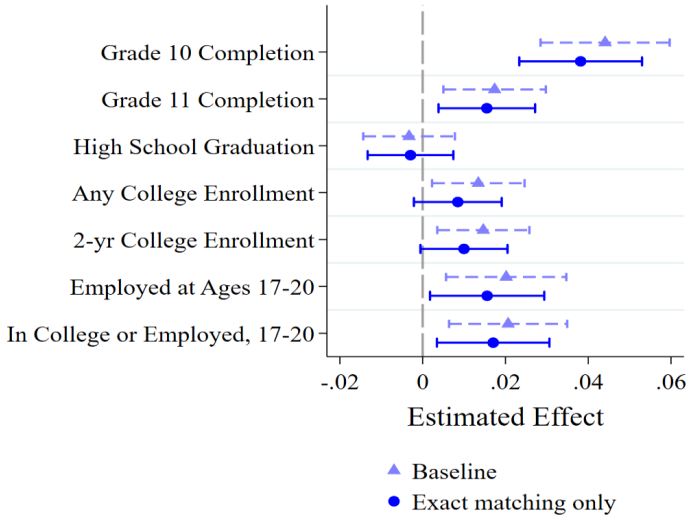
Figure A.25: Robustness of of Short-Run Analysis Results: Exact Matching Only



Notes: The figure shows the short-run analysis results obtained when I do the exact matching omitting the fuzzy matching.

Figure A.26: Robustness of Long-Run Analysis Results: Exact Matching Only

(a) Educational Outcomes and Employment

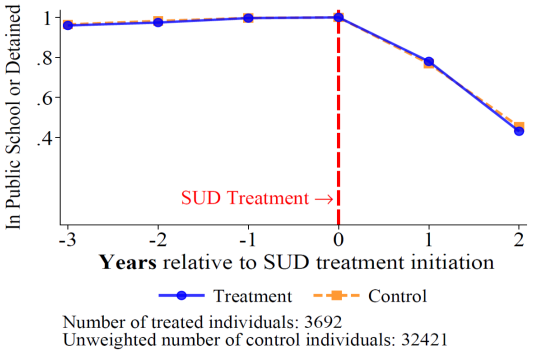


(b) Earnings

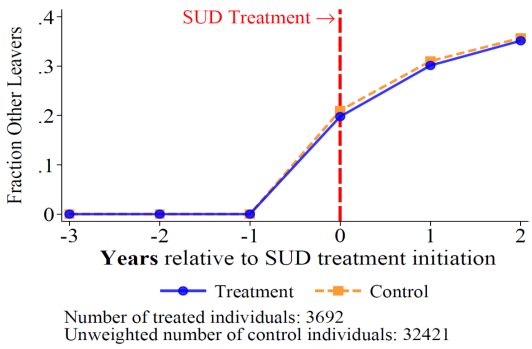
Notes: The figure shows the long-run analysis results obtained when I do the exact matching omitting the fuzzy matching.

Figure A.27: Analysis for Other Exit Reasons: Raw Trends

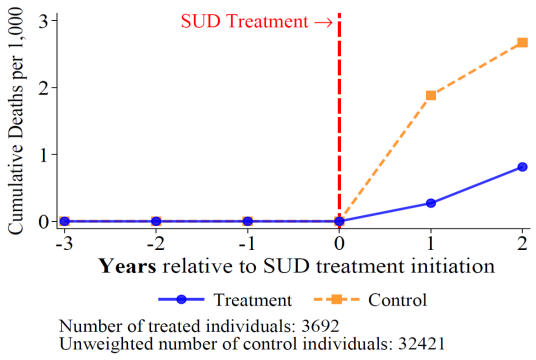
(a) In public school system or detained



(b) Left for Other Exit Reasons: Raw Trends



(c) Cumulative Number of Deaths per 1,000: Raw Trends



Notes: The figure plots raw data trends in the outcomes from three years before to two years after SUD treatment, separately for treatment and matched control individuals.

A.2 Appendix Tables

Table A.1: Average Individual Characteristics Across Treatment, Detainees with Substance-Related Discipline History, and All Detainees

	SUD Treatment School	Substance Disc. History	All Detainees	Diff	*p-val*	Diff	*p-val*
	(1)	(2)	(3)	(1) - (2)		(1) - (3)	
A. Individual Characteristics (Exact Matching Variables)							
Female	0.204	0.167	0.231	0.037	[<0.001]	-0.028	[<0.001]
Non-Hispanic White	0.285	0.192	0.226	0.093	[<0.001]	0.059	[<0.001]
Hispanic	0.544	0.612	0.486	-0.068	[<0.001]	0.057	[<0.001]
Non-Hispanic Black	0.161	0.187	0.278	-0.026	[<0.001]	-0.117	[<0.001]
Age at detention	14.869	14.860	14.611	0.010	[0.532]	0.258	[<0.001]
Free/reduced-price lunch	0.729	0.773	0.787	-0.043	[<0.001]	-0.057	[<0.001]
Special education	0.245	0.269	0.310	-0.023	[<0.001]	-0.064	[<0.001]
Urbanicity of county							
Large central metro	0.559	0.551	0.518	0.008	[0.248]	0.041	[<0.001]
Large fringe metro	0.172	0.177	0.194	-0.005	[0.396]	-0.021	[<0.001]
Medium metro	0.189	0.194	0.174	-0.006	[0.328]	0.014	[0.007]
Small metro	0.047	0.052	0.072	-0.005	[0.099]	-0.025	[<0.001]
Micropolitan	0.018	0.012	0.018	0.007	[<0.001]	0.000	[0.995]
Noncore	0.014	0.014	0.024	0.001	[0.691]	-0.009	[<0.001]
B. Average Absence Rate and Detention History at Baseline (Fuzzy Matching Variables)							
Average absence rate, 1 yr before	0.232	0.217	0.189	0.014	[<0.001]	0.043	[<0.001]
Share of periods detained, 1 yr before	0.108	0.097	0.079	0.010	[<0.001]	0.028	[<0.001]
Share of periods detained, 2 yr before	0.041	0.043	0.041	-0.002	[0.248]	0.000	[0.893]
C. Academic Performance at Baseline (Non-Matching Variables)							
Grade at the time of detention	9.067	9.038	8.846	0.029	[0.050]	0.221	[0.001]
Average past course pass rate, above median	0.610	0.611	0.711	-0.002	[0.811]	-0.102	[<0.001]
Average past reading z-score, above median	0.562	0.555	0.609	0.007	[0.363]	-0.047	[<0.001]
Average past math z-score, above median	0.604	0.596	0.643	0.009	[0.235]	-0.039	[<0.001]
Individuals (total)	5,182	41,775	227,505				
Individuals (unique)	5,182	30,412	155,861				

Notes: The table presents average individual characteristics across (i) juvenile detainees who enter a SUD treatment center school after detention, (ii) juvenile detainees who never attended a SUD treatment center during my sample period (1996–2020) but who were disciplined for substance-related problems, and (iii) all juvenile detainees. All individuals were detained in a juvenile detention center at some point between ages 12–16 over the academic years 1999–2000 to 2017–2018.

Table A.2: Average Individual Characteristics Across Baseline Treatment Sample, Treatment Individuals with No Qualified Matches, Treatment Individuals with No Exact Matches

	Baseline Treatment Sample	No Qualified Matches	No Exact Matches
	(1)	(2)	(3)
A. Individual Characteristics (Exact Matching Variables)			
Female	0.146	0.281	0.448
Non-Hispanic White	0.230	0.347	0.523
Hispanic	0.610	0.512	0.242
Non-Hispanic Black	0.157	0.130	0.190
Age at detention (gap ≤ 1)	14.841	15.042	14.947
Free/reduced-price lunch	0.752	0.648	0.562
Special education	0.207	0.305	0.381
Urbanicity of county			
Large central metro	0.619	0.477	0.309
Large fringe metro	0.163	0.196	0.210
Medium metro	0.183	0.239	0.197
Small metro	0.029	0.063	0.127
Micropolitan	0.004	0.007	0.090
Noncore	0.003	0.018	0.066
B. Average Absence Rate and Detention History at Baseline (Fuzzy Matching Variables)			
Average absence rate, 1 yr before	0.235	0.298	0.066
Share of periods detained, 1 yr before	0.098	0.255	0.103
Share of periods detained, 2 yr before	0.033	0.149	0.043
C. Academic Performance at Baseline (Non-Matching Variables)			
Grade at detention	9.023	9.049	9.280
Average past course pass rate, above median	0.609	0.509	0.648
Average past reading z-score, above median	0.553	0.544	0.608
Average past math z-score, above median	0.597	0.579	0.644
Number of individuals	4,034	285	863

The table presents average individual characteristics across (i) the final treatment sample that is used in my baseline analyses, (ii) individuals who attended a SUD treatment center school but do not have qualified matches (i.e., exact matches with non-outlier distance values), and (iii) individuals who attended a SUD treatment center school but do not have exact matches (i.e., those who are dropped during the exact matching procedure).

Table A.3: Long-Run Effects of SUD Treatment on Earnings at Ages 17–20

	Earnings, Ages 17–20
	(1)
Treatment Individual	-195.88
	(95.66)
	[0.041]
Control group outcome mean	3529.50
Effect size relative to control group mean	-5.55%
Treatment individuals	3,160
Control individuals (weighted)	3,186.4
Control individuals (total)	28,161
Control individuals (unique)	10,610
Observations	31,321
R-squared	0.361

Notes: This table presents coefficients, standard errors (in parentheses), and $p-$values [in brackets] from estimation of equation (1.3). Standard errors are clustered at the individual level.

Table A.4: Summary Table, Other Exit Reasons

Percentage (%)	All (1)	Treat (2)	Control (3)	Diff (2) - (3)	*p-val*	Diff relative to control mean
Not Observed in "other exit reasons" data	64.591	64.870	64.306	0.583	[0.081]	0.91%
Observed in "other exit reasons" data	35.409	35.13	35.694			
1. Left Texas or died	**5.245**	**5.255**	**5.236**	**0.040**	**[0.842]**	**0.76%**
1-A. Enroll in school outside Texas	2.841	2.736	2.949	-0.178	[0.230]	-6.04%
1-B. Returned to home country	2.233	2.438	2.022	0.399	[0.003]	19.73%
1-C. Death	0.172	0.081	0.264	-0.181	[<0.001]	-68.56%
2. Alternative programs	**2.041**	**2.411**	**1.662**	**0.741**	**[<0.001]**	**44.58%**
2-A. Alternative programs toward GED/diploma	2.017	2.384	1.640	0.736	[<0.001]	44.88%
2-B. High School Equivalency certificate outside Texas	0.025	0.027	0.022	0.006	[0.693]	27.27%
3. Moved to other educational setting	**11.380**	**11.241**	**11.523**	**-0.317**	**[0.260]**	**-2.75%**
3-A. Home schooling	8.904	8.613	9.202	-0.607	[0.016]	-6.60%
3-B. Enroll in Texas private school	2.476	2.627	2.321	0.290	[0.038]	12.49%
4. Other reasons	**16.742**	**16.224**	**17.273**	**-1.047**	**[<0.001]**	**-6.06%**
4-A. Expelled for offense	0.251	0.217	0.287	-0.095	[0.033]	-33.10%
4-B. Removed—Child Protective Services	0.491	0.488	0.494	-0.004	[0.943]	-0.81%
Old reason codes (used between 1999–2007)						
4-C. Enroll in other Texas public school (not verified)	9.257	8.722	9.805	-1.044	[0.005]	-10.65%
4-D. Incarcerated in a facility outside the district	3.500	3.277	3.728	-0.443	[<0.001]	-11.88%
4-E. Other	3.243	3.521	2.958	0.540	[<0.001]	18.26%
Individuals (weighted)	7,291.3	3,692.0	3,599.3			
Individuals (total)	36,113	3,692	32,421			
Individuals (unique)	15,628	3,692	11,936			

Notes: The table presents the distribution of "other exit reasons"—reasons for leaving the Texas public school system other than dropout and graduation—across (i) the full sample, (ii) treatment individuals, and (iii) matched control individuals.

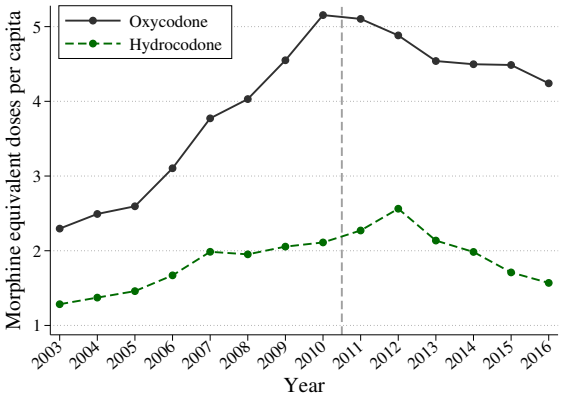
Appendix B

Appendix to Chapter 2

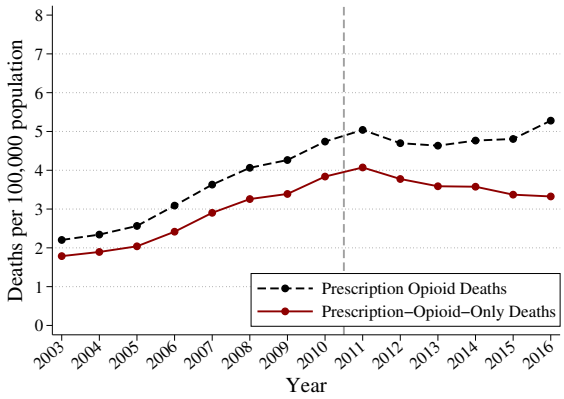
B.1 Supplementary Figures and Tables

Figure B.1: National Trends in the Legal Supply of Opioids and Prescription Opioid Death Rates

Panel A. The per capita legal supply of opioids

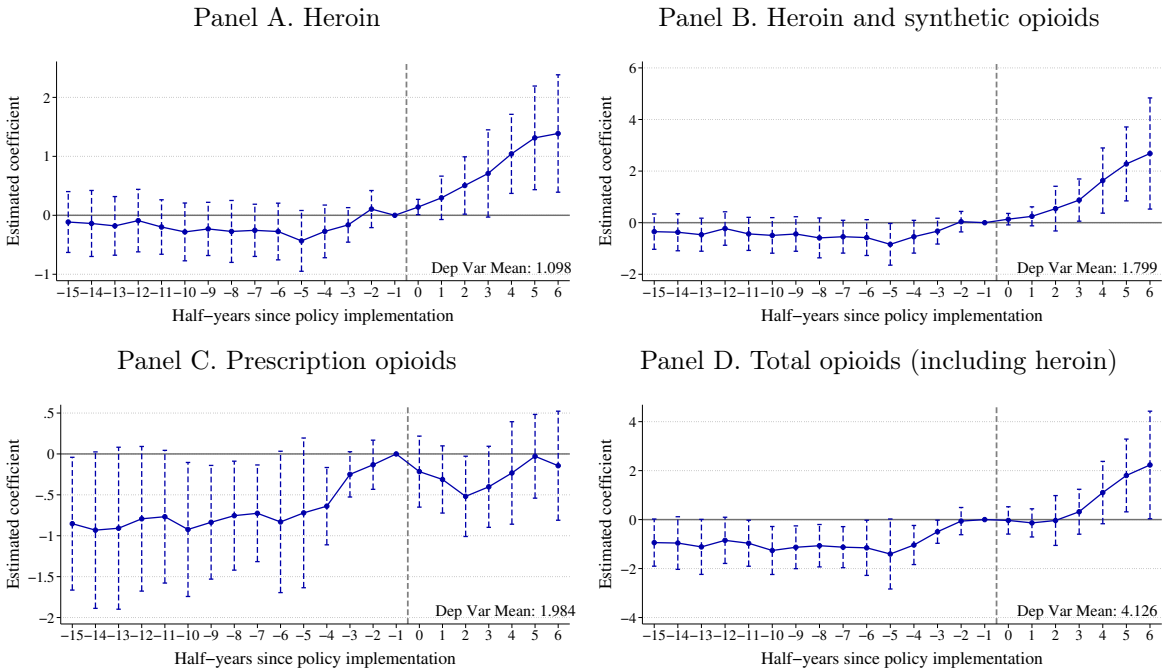


Panel B. Prescription Opioid Deaths per 100,000



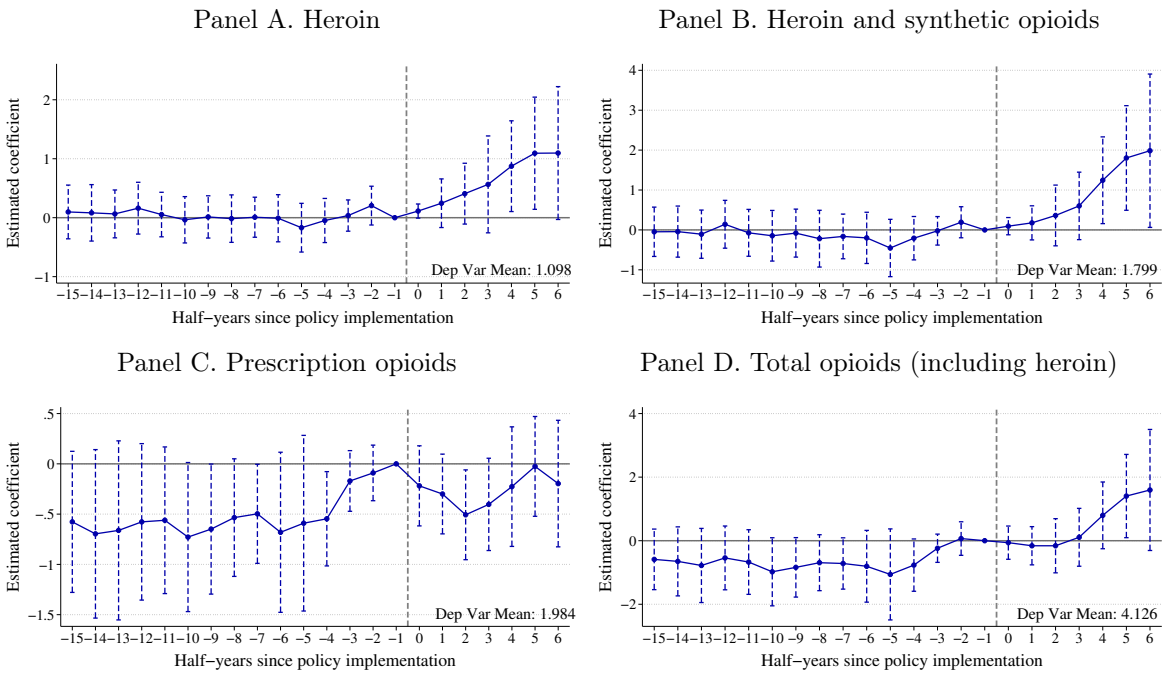
Notes: The figure plots the national trends in the per capita legal supply of opioids (Panel A) and Ruhm-corrected numbers of deaths from prescription opioids per 100,000 population (Panel B). The legal supply of oxycodone and hydrocodone in morphine equivalent doses obtained from the DEA's Automation of Reports and Consolidated Orders System (ARCOS). Ruhm-corrected numbers of deaths per 100,000 population are calculated using data from the National Vital Statistics System (NVSS). Drug overdose deaths are coded using ICD-10 underlying cause of death codes: X40–X44, X60–X64, X85, and Y10–Y14. Prescription opioid mortality rates, which use ICD-10 drug code T40.2, are identical to those in Figure 2.1. Prescription-opioid-only deaths indicate the deaths involved T40.2 but not T40.1, T40.3 or T40.4 at the time of death.

Figure B.2: Baseline Results without the Controls for the Reformulation



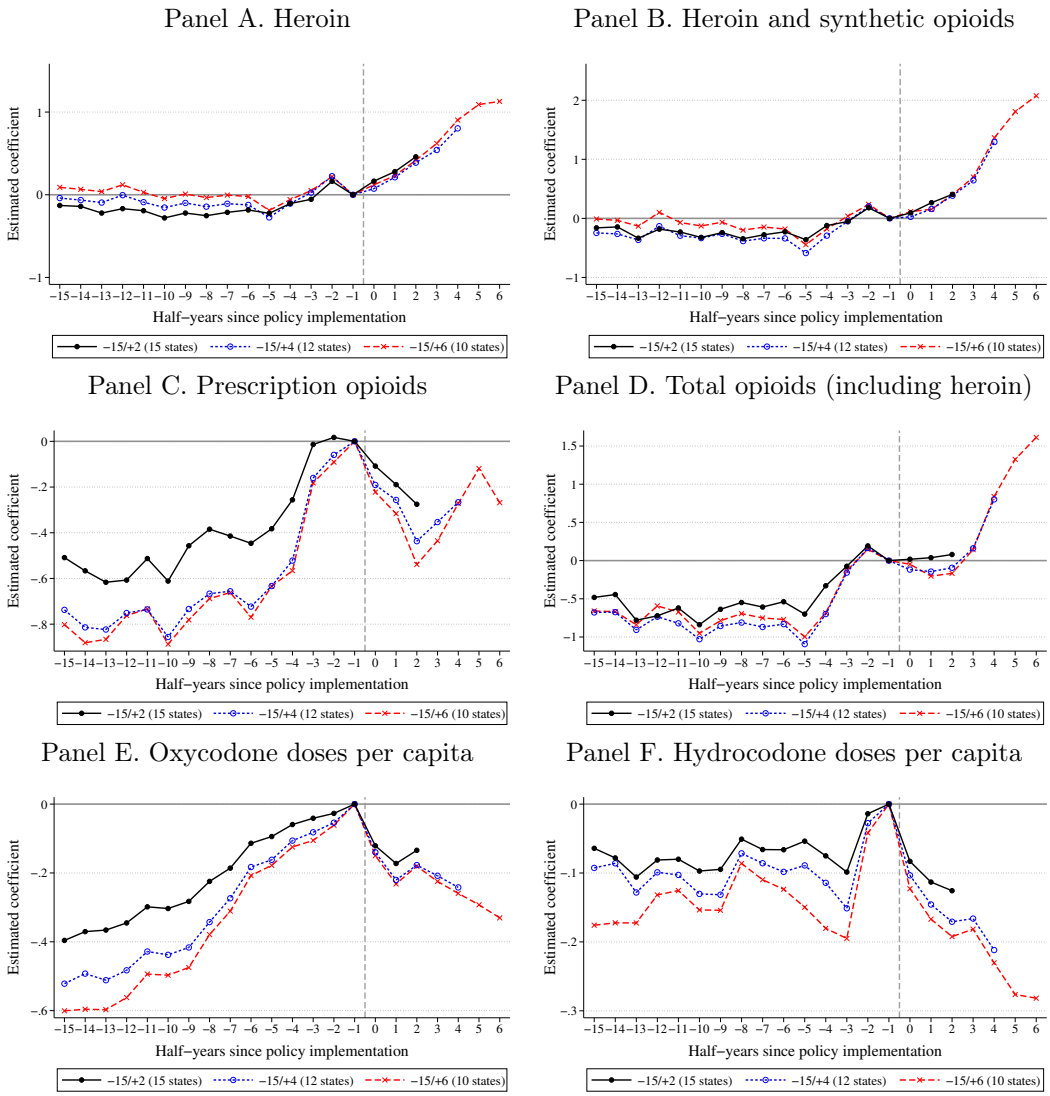
Notes: The figure displays the coefficients on the indicators for pre- and post-periods from the baseline specification (equation 3.1) without the controls for the reformulation. The dependent variable is heroin deaths per 100,000 (drug code T40.1) in Panel A, the combined deaths from heroin and synthetic opioids per 100,000 (drug codes T40.1, T40.4) in Panel B, prescription opioid deaths per 100,000 (drug code T40.2) in Panel C, and total deaths from any opioid, including heroin (drug codes T40.1–T40.4) in Panel D.

Figure B.3: Baseline Results with the NSDUH Measure of OxyContin misuse



Notes: The figure displays the coefficients on the indicators for pre- and post-periods from the baseline specification (equation 3.1). In all panels, the NSDUH measure, instead of the ARCOS measure, is interacted with time fixed effects to account for exposure to the reformulation. The dependent variable is heroin deaths per 100,000 (drug code T40.1) in Panel A, the combined deaths from heroin and synthetic opioids per 100,000 (drug codes T40.1, T40.4) in Panel B, prescription opioid deaths per 100,000 (drug code T40.2) in Panel C, and total deaths from any opioid, including heroin (drug codes T40.1–T40.4) in Panel D.

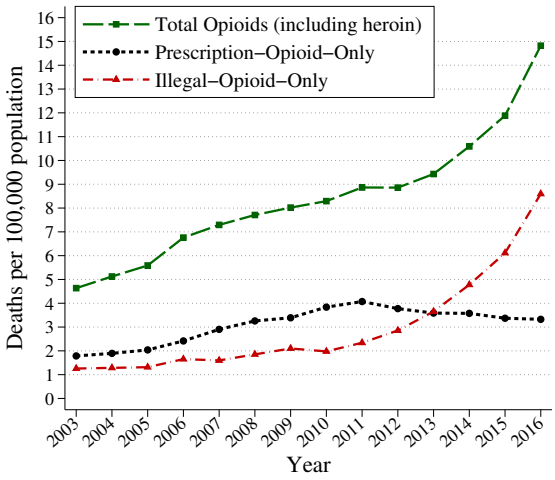
Figure B.4: Baseline Results with Different Event Time Windows



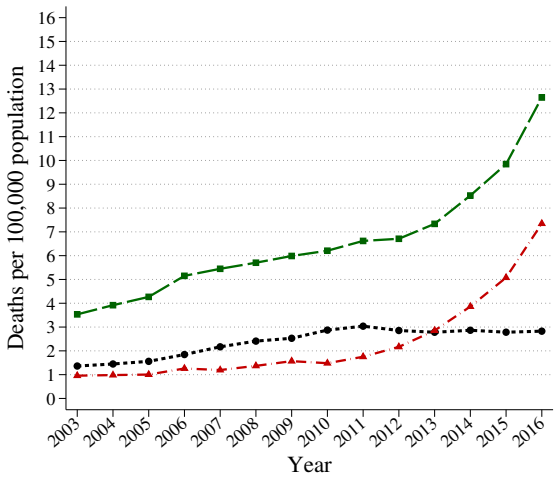
Notes: The figure displays the coefficients on the indicators for pre- and post-periods from the baseline specification (equation 3.1) on three samples with different event time windows (separate regressions). Each sample includes my baseline control group and treated states that were consistently observed during one of the following event time windows: -15/+6, -15/+4, or -15/+2. The dashed red line presents the baseline estimates, obtained using the sample that includes the 10 treated states that are consistently observed during the broadest event time window (-15/+6). The short-dashed blue line corresponds to the 12 treated states that are observed during the -15/+4 event time window. The black solid line corresponds to the 15 treated states that are observed during the narrowest event time window (-15/+2). The dependent variable is heroin deaths per 100,000 (drug code T40.1) in Panel A, illegal opioid death rate (T40.1, T40.4) in Panel B, prescription opioid death rate (T40.2) in panel C, total opioid-related death rate (T40.1–T40.4) in Panel D, oxycodone (morphine equivalent) doses per capita in Panel E, and hydrocodone (morphine equivalent) doses per capita in Panel F. Observations are weighted by state population. The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. Florida is excluded from the control sample in all panels (see Appendix Section B.3). The controls are identical to those in Figure 2.3.

Figure B.5: National Trends in the Exclusive Measures of Opioid Death Rates

Panel A. Ruhm-corrected drug overdose deaths

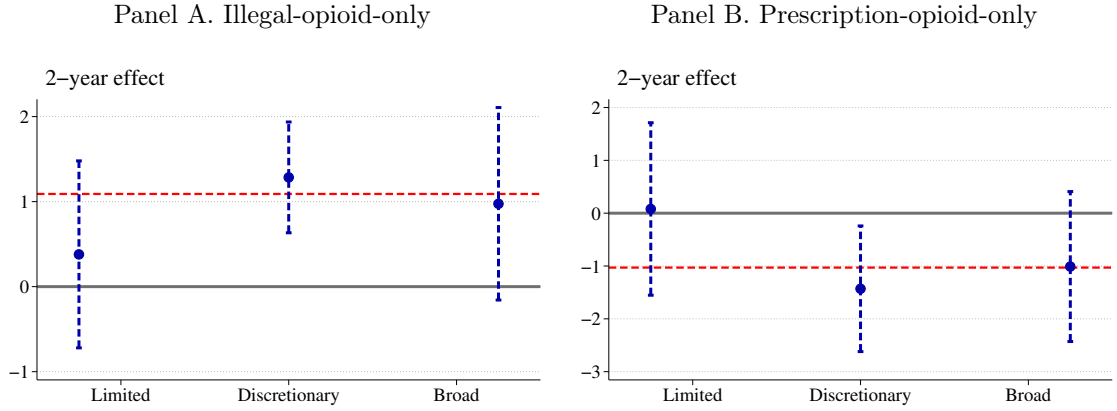


Panel B. Reported drug overdose deaths



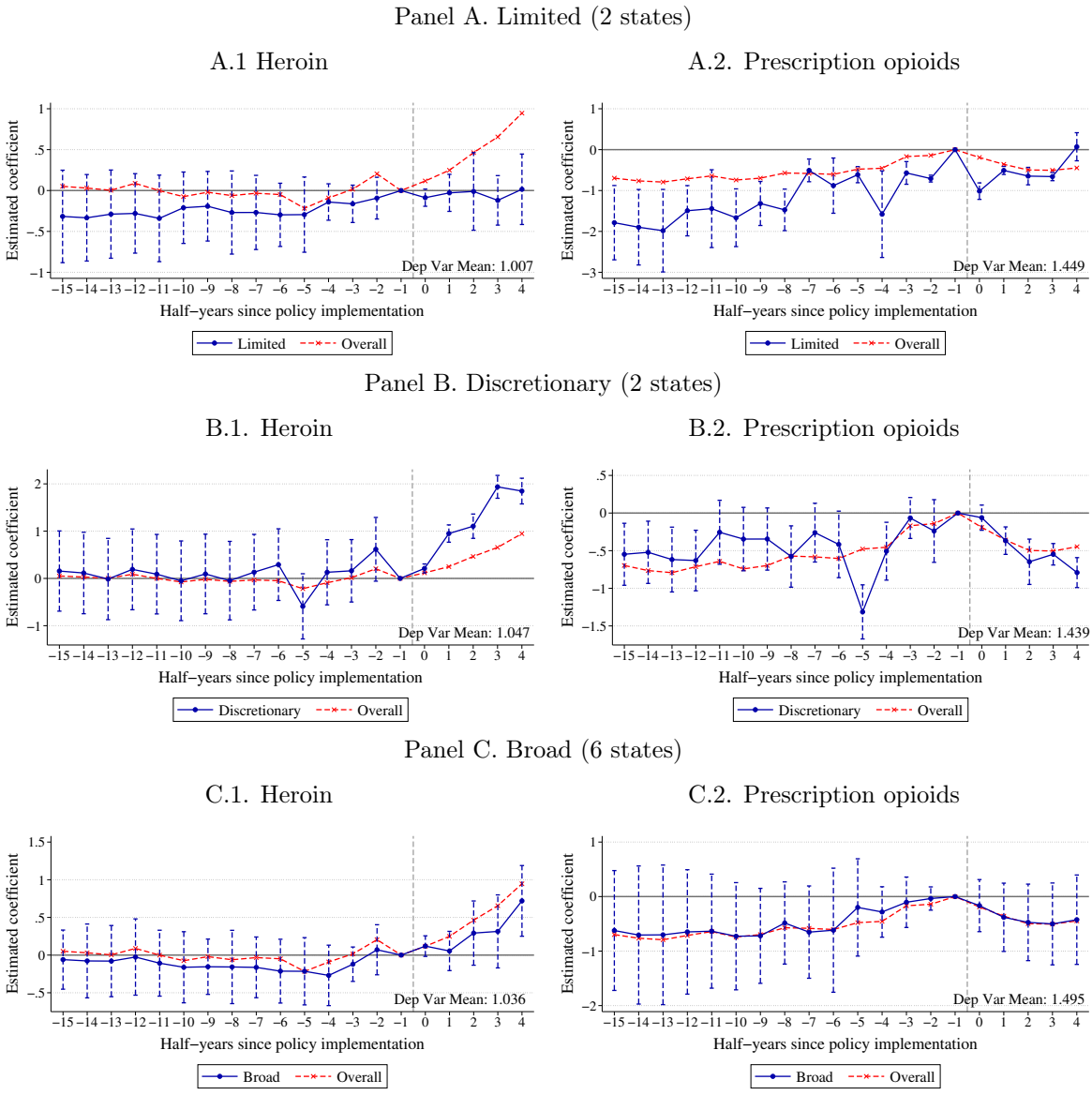
Notes: The figure plots the national trends in corrected and reported numbers of deaths per 100,000 population calculated using mortality data from the National Vital Statistics System (NVSS). Drug overdose deaths are coded using ICD-10 underlying cause of death codes: X40–X44, X60–X64, X85, and Y10–Y14. To identify drug involvement, the following four drug identification codes are used: heroin (T40.1), natural and semisynthetic opioids such as oxycodone and hydrocodone (T40.2), methadone (T40.3), and synthetic opioids excluding methadone, such as fentanyl (T40.4). I calculate total deaths from any opioid, including heroin, by combining T40.1–T40.4. Prescription-opioid-only deaths indicate the deaths involved T40.2 but not T40.1, T40.3, or T40.4 at the time of death. Illegal-opioid-only deaths indicate the deaths involved T40.1 or T40.4 but not T40.2 or T40.3 at the time of death. Reported mortality rates are based on mentions of the specified drugs on the death certificates. Corrected mortality rates are estimated by using the method suggested by Ruhm (2018), which uses information from death certificates that specified at least one drug category to impute drug involvement for cases in which none was specified.

Figure B.6: Heterogeneous Treatment Effects on Exclusive Mortality Outcomes



Notes: The figure shows the heterogeneous treatment effects across my three types of must-access laws: (i) limited laws that apply to certain ingredients or require access under limited circumstances (2 states), (ii) discretionary laws that rely on provider suspicion of abuse (2 states), and (iii) broad laws without such restrictions (6 states). Each panel presents the estimates I obtain when I interact the indicators for all the post-periods (from 0 to +6) from the baseline specification (equation 3.1) with three indicators for limited, discretionary and broad laws. The (horizontal) dashed red line presents the overall estimate, for reference. In Panel A, the dependent variable is illegal-opioid-only deaths per 100,000, which involved T40.1 or T40.4 but not T40.2 or T40.3 at the time of death. In Panel B, the dependent variable is prescription-opioid-only deaths, which involved T40.2 but not T40.1, T40.3, or T40.4. Ruhm-corrected numbers of deaths are used in all panels. In all panels, Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. For all the outcomes, I report the trend break estimates summarizing the two-year effect ($\Delta 5 = (\beta_4 * \mathbf{1}(\text{Law Type}) - \beta_{-1}) - (\beta_{-1} - \beta_{-6})$). I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. Observations are weighted by state population. In all panels, the analysis sample and controls are identical to those in Figure 2.3. Fixed effects for state and half-year are always included.

Figure B.7: Separate Event Studies by Law Type—Opioid Deaths

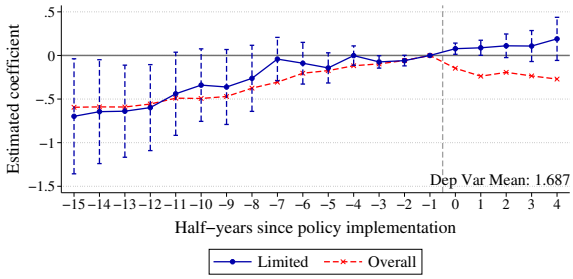


Notes: The figure presents the heterogeneous treatment effects across my three types of must-access laws: (i) limited laws that apply to certain ingredients or require access under limited circumstances (2 states), (ii) discretionary laws that rely on provider suspicion of abuse (2 states), and (iii) broad laws without such restrictions (6 states). The figure displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) obtained when I limit the treated group to each of the three law type. The treated sample is balanced in relative periods from -15 to +4, and the distant relative periods outside the -15/+4 event time window are trimmed. The dashed red line indicates the overall effects of must-access PDMPs among the ten treated states. In all panels, the control sample is the baseline control sample. In the left column (Panels A.1, B.1, and C.1), the dependent variable is heroin deaths per 100,000 (drug code T40.1). In the right column (Panels A.2, B.2, and C.2), the dependent variable is prescription opioid deaths per 100,000 (drug code T40.2). In all panels, Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. Observations are weighted by state population. The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. The controls are identical to those in Figure 2.3.

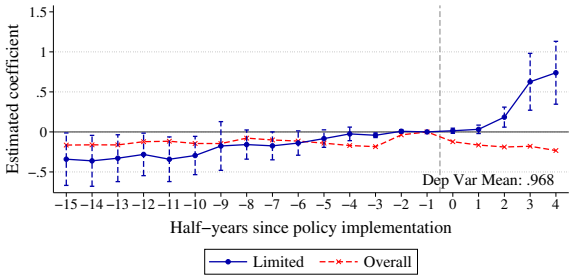
Figure B.8: Separate Event Studies by Law Type—Opioid Supply

Panel A. Limited (2 states)

A.1 Oxycodone doses per capita

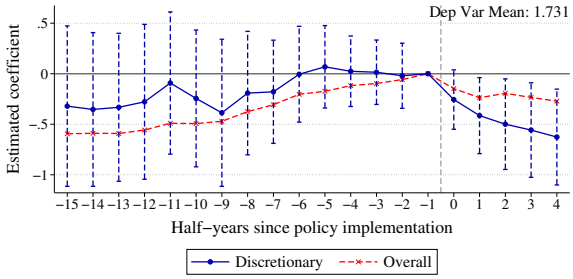


A.2. Hydrocodone doses per capita

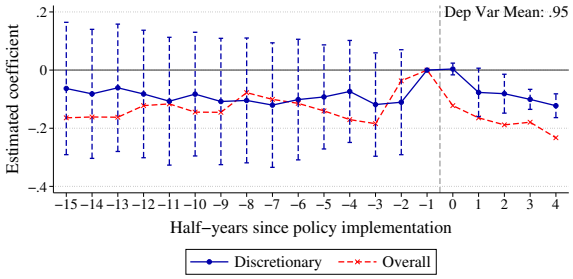


Panel B. Discretionary (2 states)

B.1. Oxycodone doses per capita

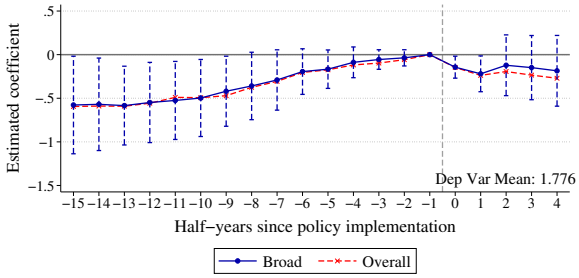


B.2. Hydrocodone doses per capita

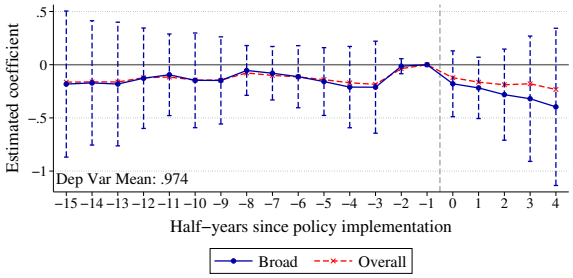


Panel C. Broad (2 states)

C.1. Oxycodone doses per capita

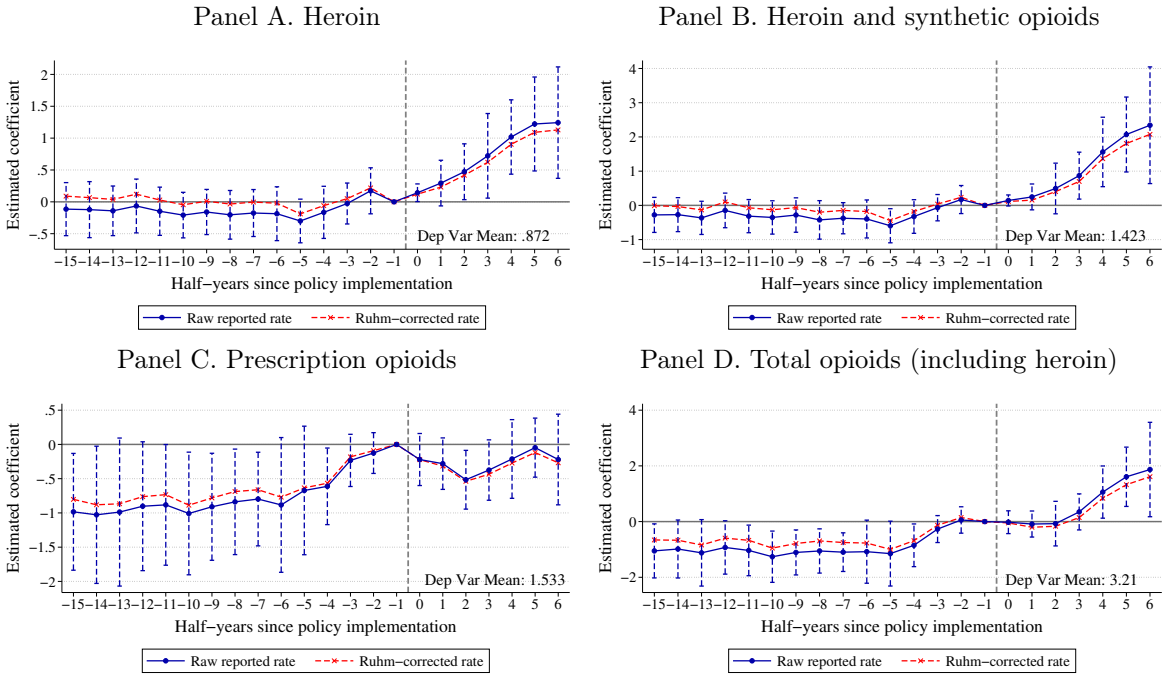


C.2. Hydrocodone doses per capita



Notes: The figure presents the heterogeneous treatment effects across my three types of must-access laws: (i) limited laws that apply to certain ingredients or require access under limited circumstances (2 states), (ii) discretionary laws that rely on provider suspicion of abuse (2 states), and (iii) broad laws without such restrictions (6 states). The figure displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) obtained when I limit the treated group to each of the three law type. The treated sample is balanced in relative periods from -15 to +4, and the distant relative periods outside the -15/+4 event time window are trimmed. The dashed red line indicates the overall effects of must-access PDMPs among the ten treated states. In all panels, the control sample is the baseline control sample. In the left column (Panels A.1, B.1, and C.1), the dependent variable is oxycodone (morphine equivalent) doses per capita. In the right column (Panels A.2, B.2, and C.2), the dependent variable is and hydrocodone (morphine equivalent) doses per capita. Observations are weighted by state population. The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. The controls are identical to those in Figure 2.3.

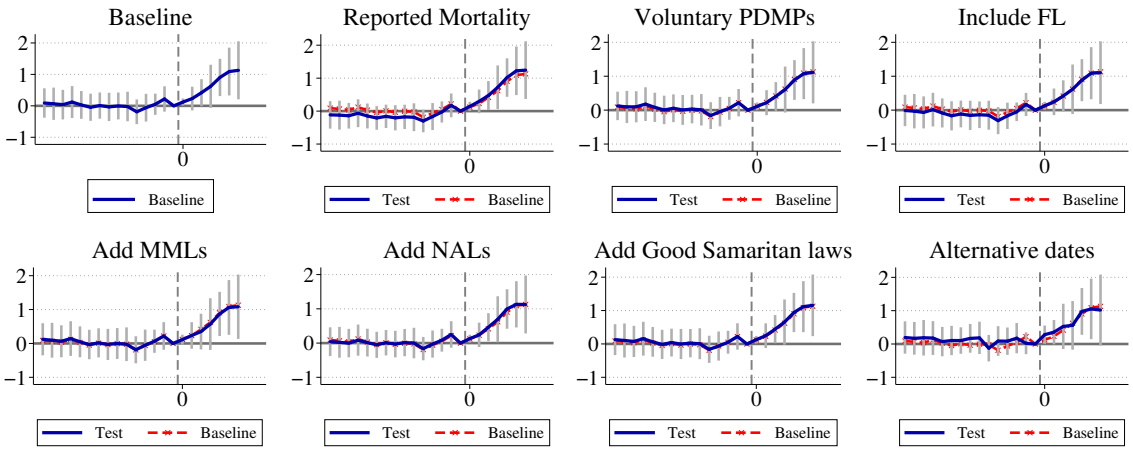
Figure B.9: Effects of Must-Access PDMPs on Raw Reported Opioid Death Rates



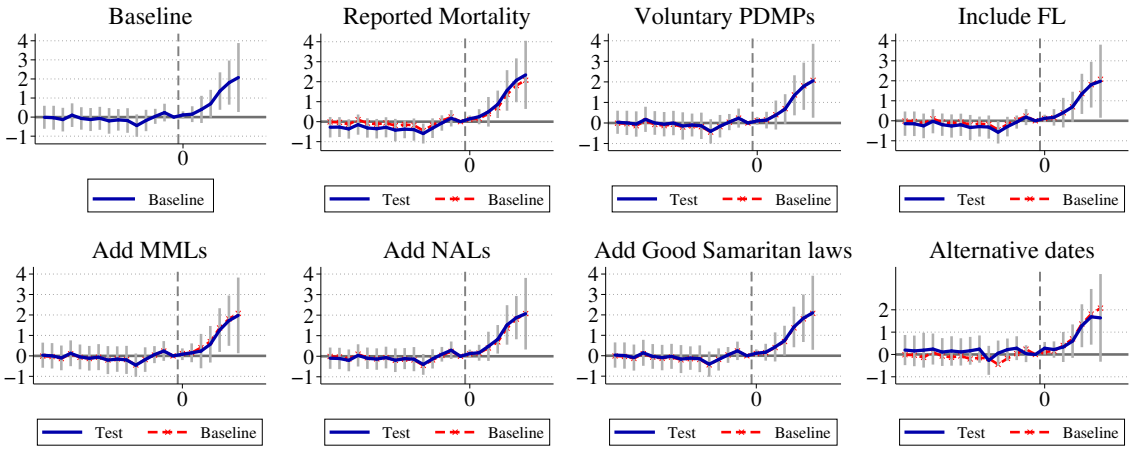
Notes: The figure displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) that I obtain when I use the raw reported death rates instead of the Ruhm-corrected death rates. The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. In Panel A, the dependent variable is heroin deaths per 100,000 (drug code T40.1). In Panel B, the dependent variable is the combined deaths from heroin and synthetic opioids per 100,000 (drug codes T40.1, T40.4). In Panel C, the dependent variable is prescription opioid deaths per 100,000 (drug code T40.2). In Panel D, the dependent variable is total deaths from any opioid, including heroin (drug codes T40.1–T40.4). The raw reported numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used in all panels. Observations are weighted by state population. The sample and controls are identical to those in Figure 2.3.

Figure B.10: Robustness of the Baseline Mortality Estimates to Alternative Explanations

Panel A. Heroin



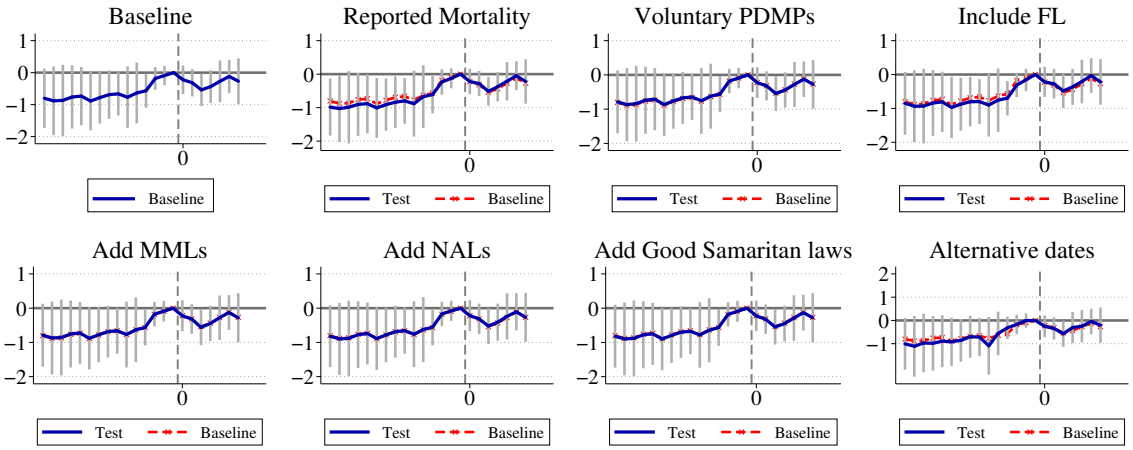
Panel B. Heroin and synthetic opioids



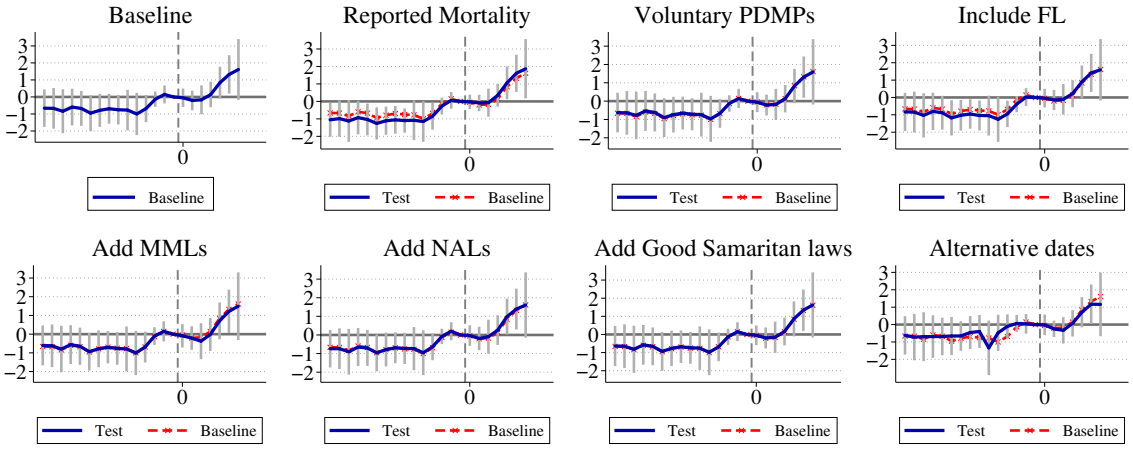
(continued)

Figure B.10: Robustness of the Mortality Estimates to Alternative Explanations (continued)

Panel C. Prescription opioids



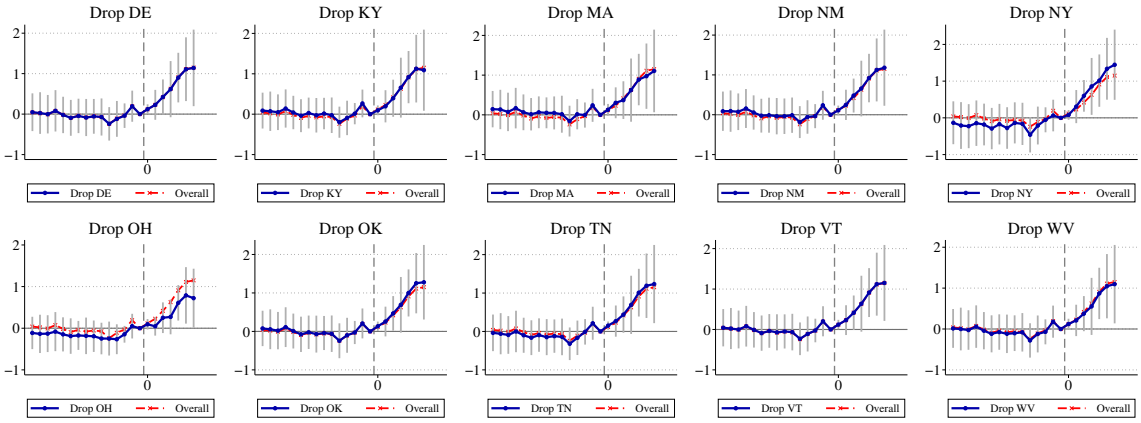
Panel D. Total opioids (including heroin)



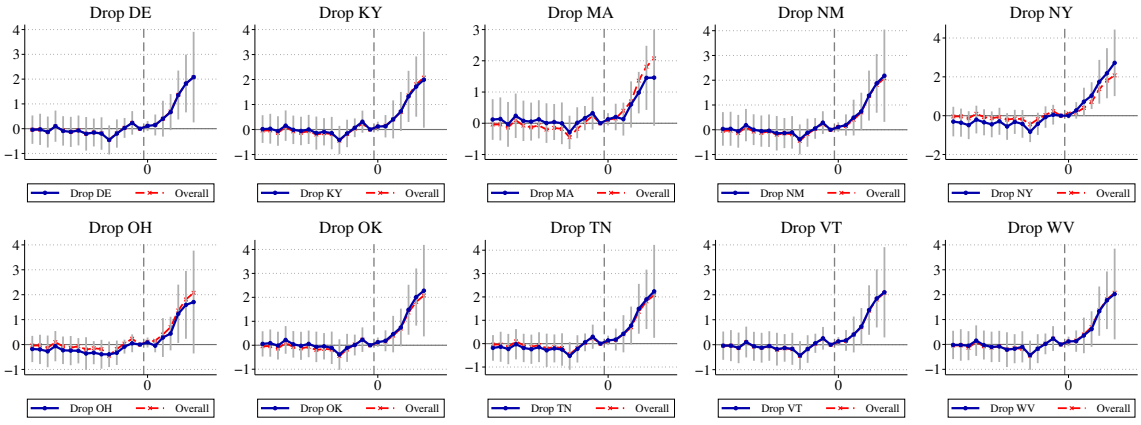
Notes: The figure shows the robustness of the baseline estimates to several sensitivity tests. The dependent variable is heroin deaths per 100,000 (drug code T40.1) in Panel A, the combined deaths from heroin and synthetic opioids per 100,000 (drug codes T40.1, T40.4) in Panel B, prescription opioid deaths per 100,000 (drug code T40.2) in Panel C, and total deaths from any opioid, including heroin (drug codes T40.1–T40.4) in Panel D. The estimates in Panel A are identical to those presented in Table 2.6. The estimates in Panels B, C, and D are identical to those presented in Appendix Table B.4.

Figure B.11: Robustness of the Mortality Estimates to Dropping One Treated State

Panel A. Heroin



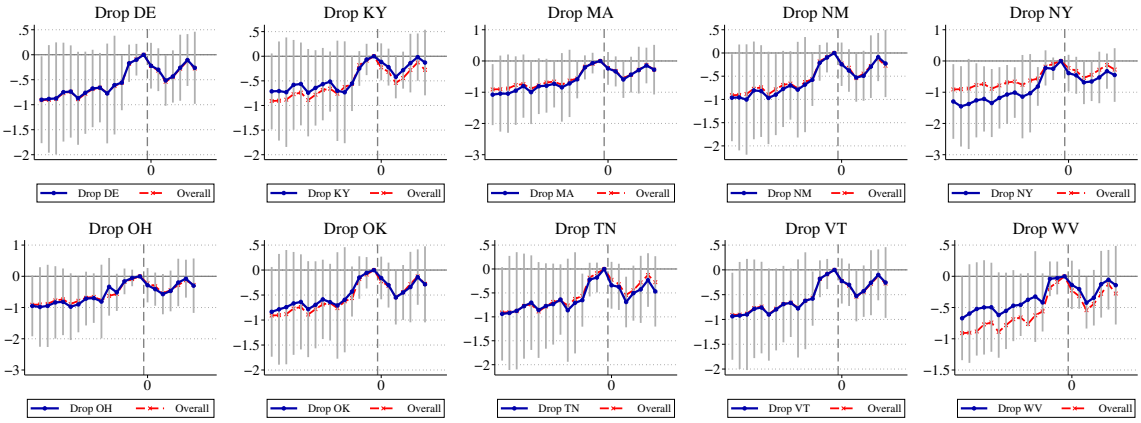
Panel B. Heroin and synthetic opioids



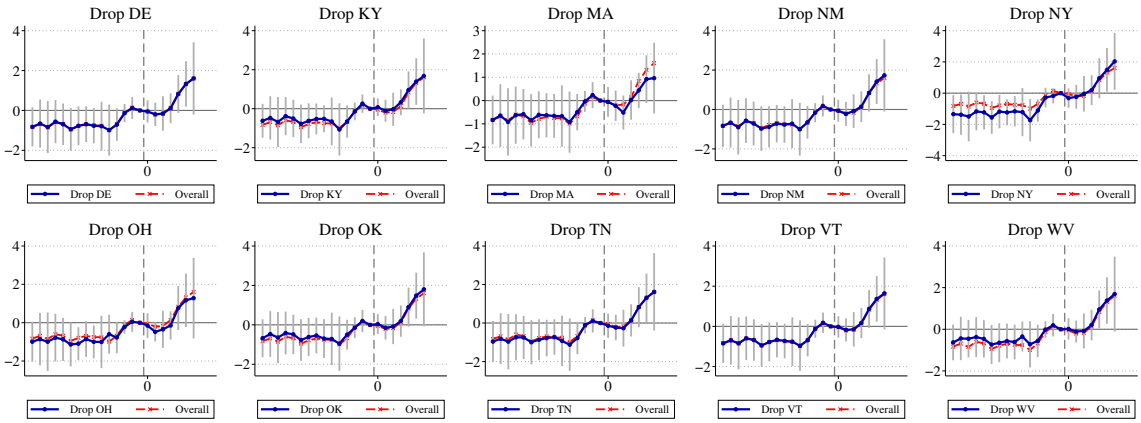
(continued)

Figure B.11: Robustness of the Mortality Estimates to Dropping One Treated State (continued)

Panel C. Prescription opioids

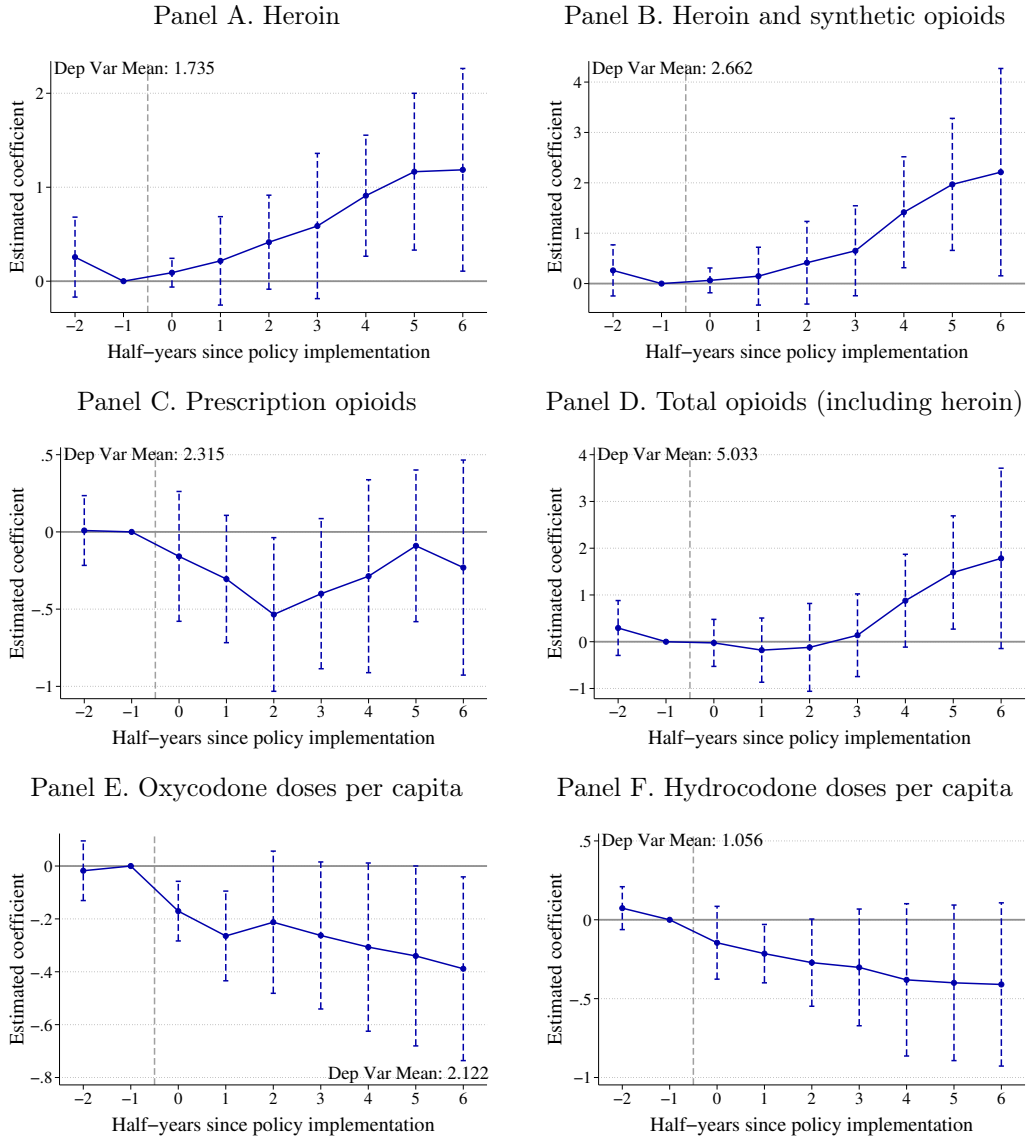


Panel D. Total opioids(including heroin)



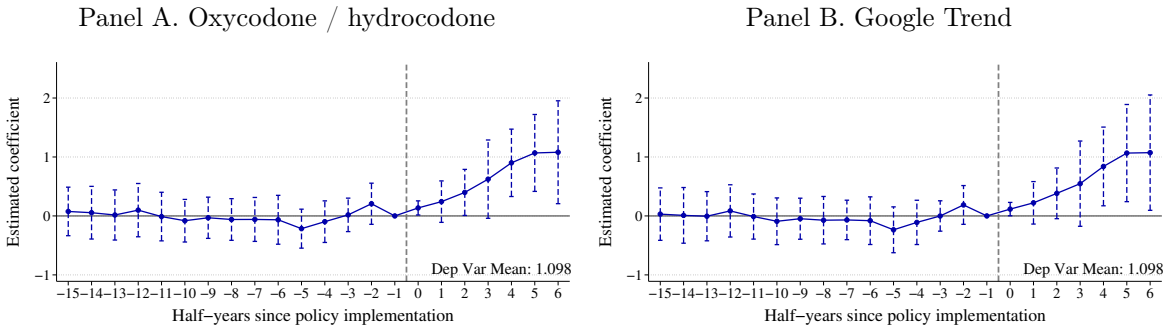
Notes: The figure shows the sensitivity of the mortality estimates to dropping one of the ten treated states. Each panel displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) obtained when I drop one of the ten treated states. The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. The vertical dashed gray line indicates the implementation timing of a must-access PDMP. The dashed red line presents the baseline estimates indicating the overall effects among all the ten treated states. The dependent variable is heroin deaths per 100,000 (drug code T40.1) in Panel A, illegal opioid death rate (T40.1, T40.4) in Panel B, prescription opioid death rate (T40.2) in panel C, and total opioid-related death rate (T40.1–T40.4) in Panel D. Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used in all panels. The control states are the 34 that did not implement must-access policies until 2016h. Florida is excluded from the control sample (see Appendix Section B.3). Observations are weighted by state population. In all panels, the controls are identical to those in Figure 2.3.

Figure B.12: Robustness of the Baseline Estimates to Dropping the Pre-Reformulation Period



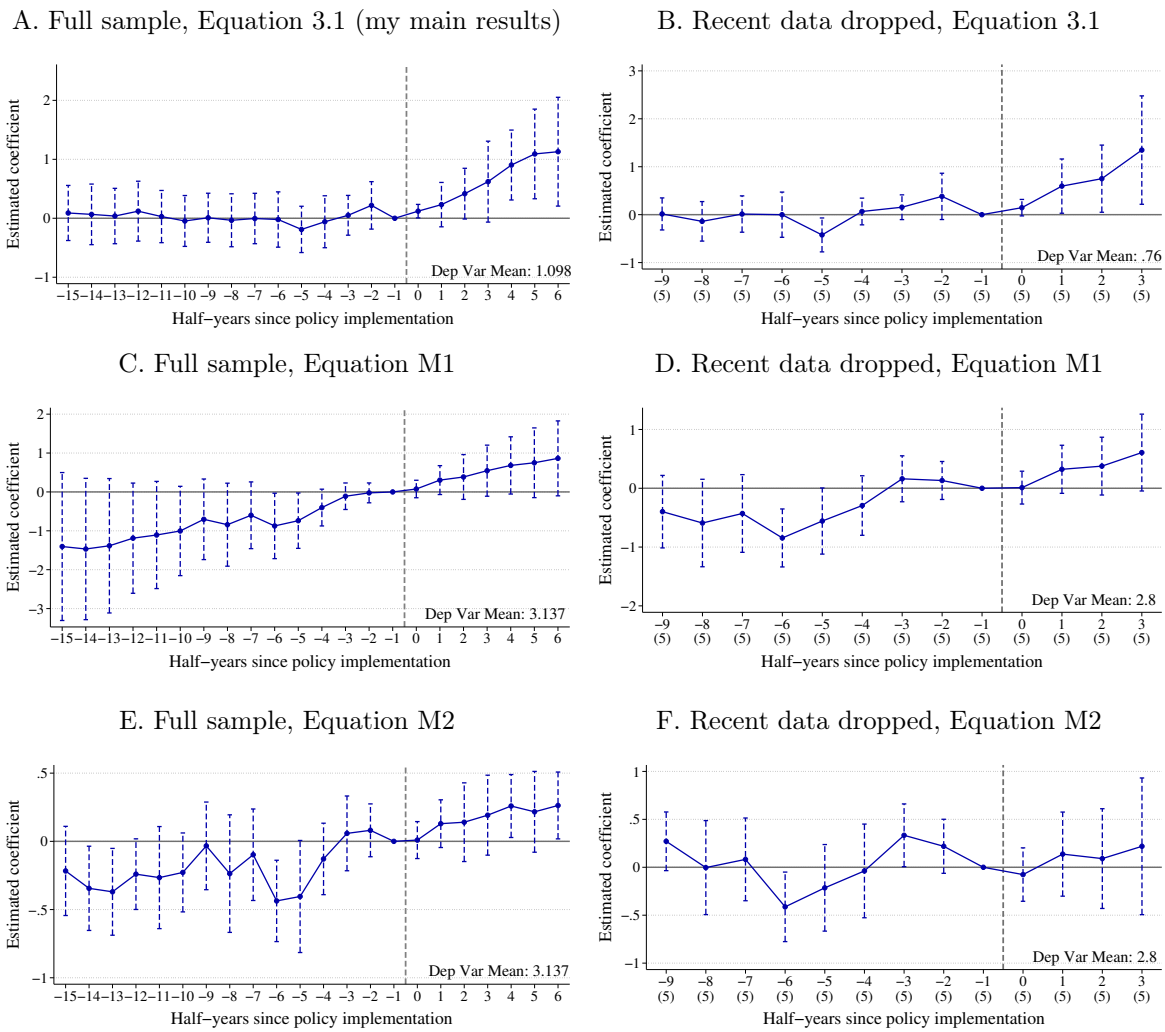
Notes: The figure displays the coefficients on the indicators for pre- and post-periods from the baseline specification (equation 3.1) obtained when I drop the pre-reformulation period (the reformulation was introduced in 2010h2). The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. The dependent variable is heroin deaths per 100,000 (drug code T40.1) in Panel A, illegal opioid death rate (T40.1, T40.4) in Panel B, prescription opioid death rate (T40.2) in panel C, total opioid-related death rate (T40.1–T40.4) in Panel D, oxycodone (morphine equivalent) doses per capita in Panel E, and hydrocodone (morphine equivalent) doses per capita in Panel F. In Panels A–D, Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. In all panels, the treatment states are the nine that implemented must-access PDMPs from 2011h2 to 2013h2, and the treated sample is balanced in relative periods from -2 to +6. The distant relative periods outside the -2/+6 event time window are trimmed. The control states are the 34 that did not implement must-access policies until 2016h2, and the control sample is balanced from 2010h2 to 2016h2. Florida is dropped (see Appendix Section B.3). Observations are weighted by state population. The controls are identical to those in Figure 2.3.

Figure B.13: Alternative Measures of Pre-Reformulation OxyContin Use



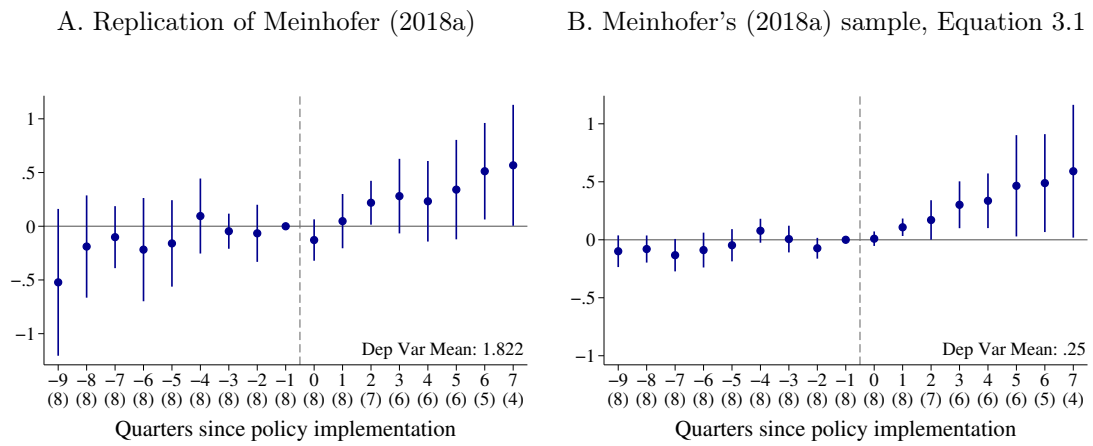
Notes: The figure displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) obtained when I use two alternative measures of pre-reformulation OxyContin use (separate regressions): Panel A uses oxycodone/hydrocodone in morphine equivalent doses per capita, and Panel B uses the Google Trend measure obtained from Beheshti (2019). The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. The dependent variable is heroin deaths per 100,000 (drug code T40.1). Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. Observations are weighted by state population. The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. The controls are identical to those in Figure 2.3.

Figure B.14: My Analysis Sample with Prior Literature Specification



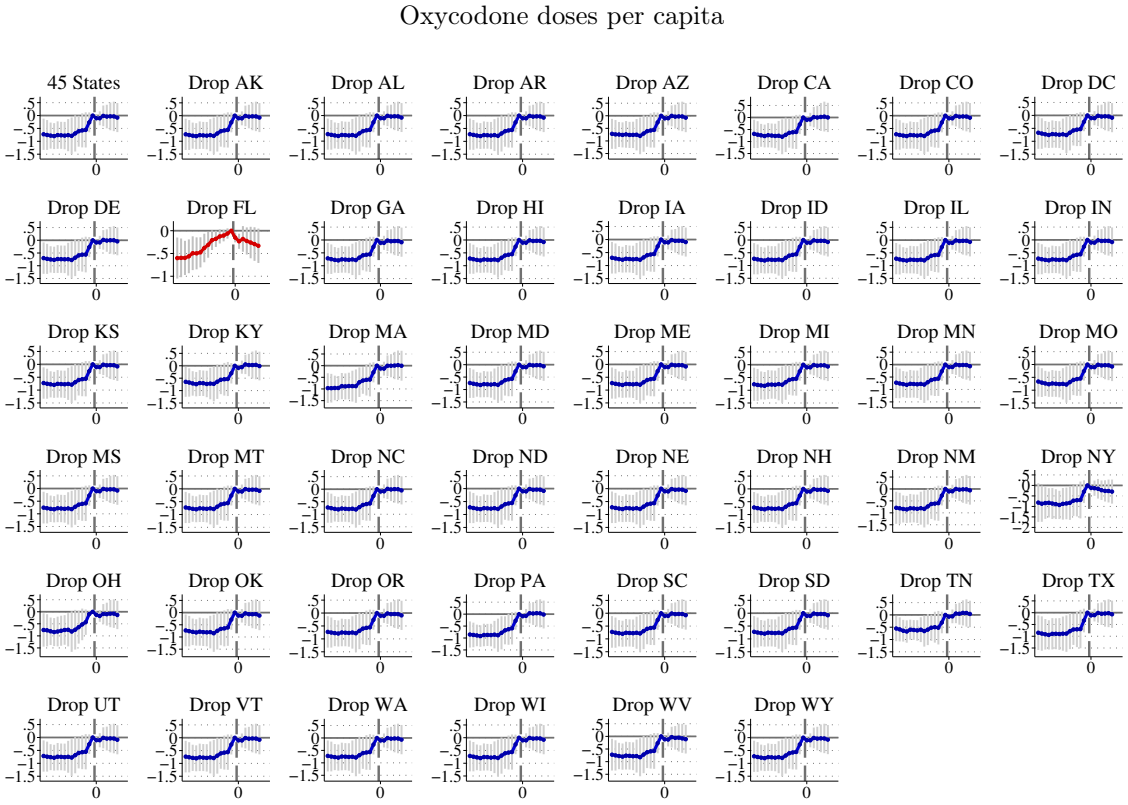
Notes: Each column corresponds to different sample periods. In the left column (Panels A, C, and E), I use my full sample (2003h1–2016h2), and in the right column (Panels B, D, and F), I drop the 2014h1–2016h2 period and reconstruct the sample so that the treated states are balanced in relative periods from -9 to +3. The sample from the right column includes the five treated states. The number of treated states observed in each time period is presented in the parentheses below that period. In all panels, the treated sample is balanced in relative periods, and the distant relative periods outside the given event time window are trimmed. Each row uses one of the three specifications: the top row (Panels A and B) uses my baseline specification (equation 3.1), the middle (Panels C and D) uses Meinhofer's preferred event study specification (equation M1), and the bottom (panels E and F) uses Meinhofer's alternative specification (equation M2). The regressions estimating my specification (equation 3.1) are weighted by population, while the regressions estimating Meinhofer's (equations M1 and M2) are unweighted.

Figure B.15: Meinhofer's (2018a) Analysis Sample with My Specification



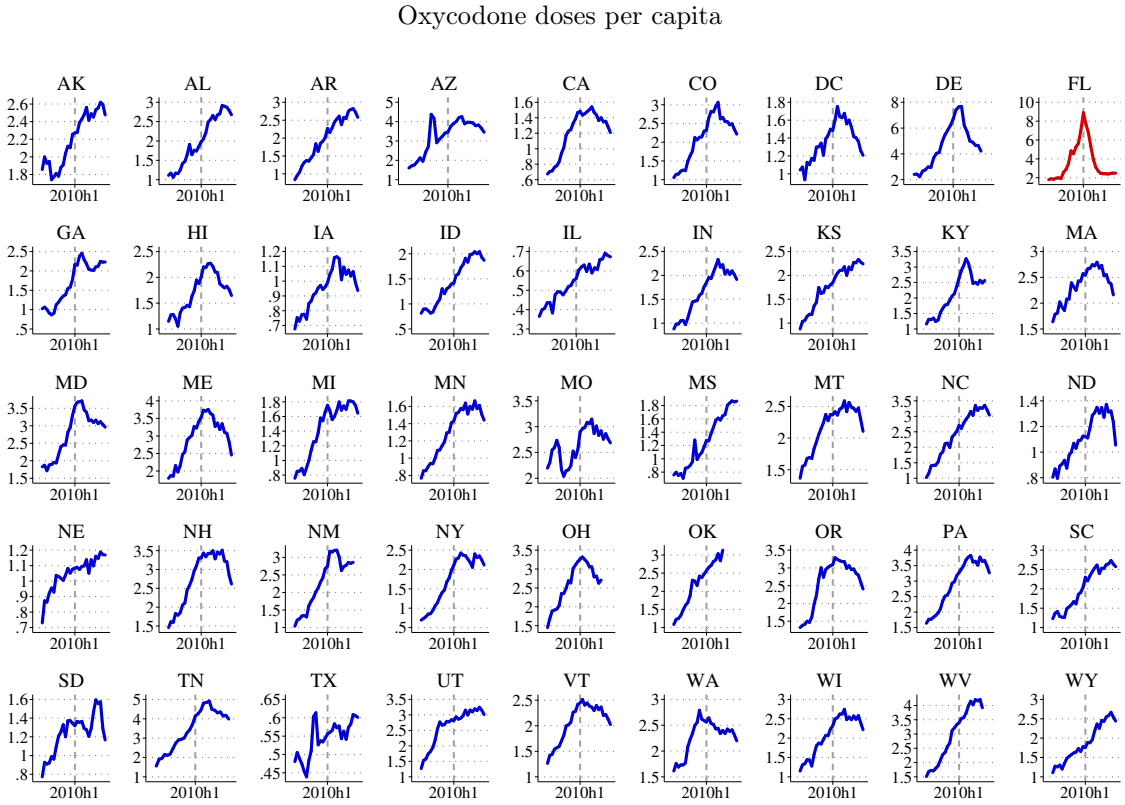
Notes: This is Appendix Figure B.15 in the revised paper. The figure shows how Meinhofer's (2018a) heroin results are affected if I use my specification instead. Panel A displays the replication of Meinhofer's (2018a) event study results for heroin mortality, and Panel B presents the estimates that I obtain when I use my baseline specification (equation 3.1) instead, while everything else, including the analysis sample from Panel A, remains unchanged. The regression estimating my specification (equation 3.1) is weighted by population, while the regression estimating Meinhofer's (equations M1) is unweighted. The sample from Panel A is unbalanced in relative (quarter) periods from -9 to +7. The number of treated states observed in each event time period is presented in the parentheses below that period. The distant relative periods that are outside the -9/+7 event time window are dropped. The control sample is balanced from 2000q1 to 2013q4.

Figure B.16: Sensitivity of Oxycodone Results to Dropping Florida



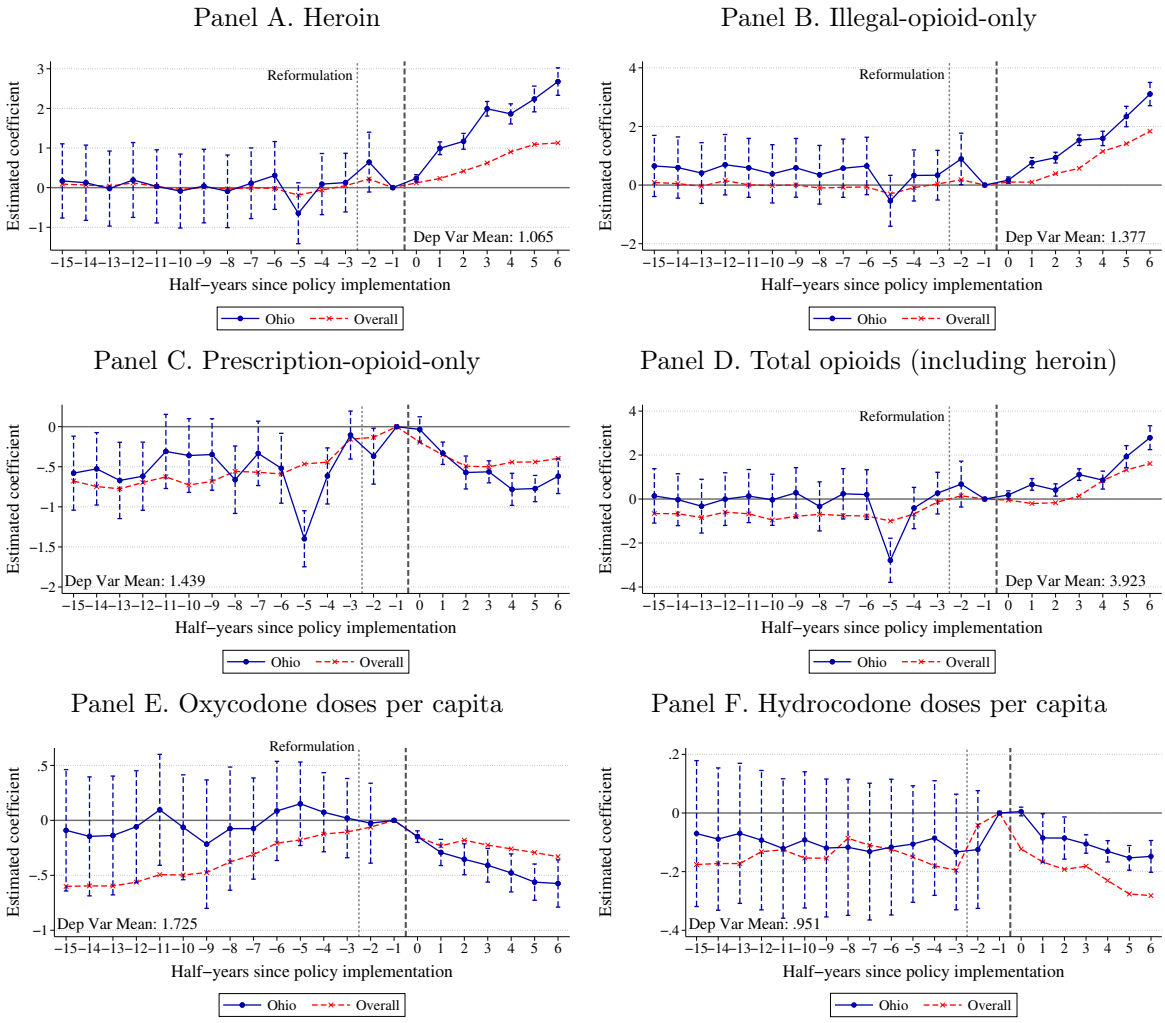
Notes: The top left panel displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) obtained when I include Florida in my analysis sample. The subsequent panels show the sensitivity of the top left panel's estimates to removing one of the 45 states. In the sample from the top left panel, the treated states are the 10 that implemented must-access PDMPs from 2010h2 to 2013h2, and the control states are the 35 that did not implement must-access policies until 2016h2. The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. The dependent variable is oxycodone (morphine equivalent) doses per capita.

Figure B.17: Trends in Per Capita Legal Supply of Oxycodone by State



Notes: Each panel displays the trends in a state's legal supply of oxycodone (morphine equivalent) doses per capita in the half-year period. The dashed gray line indicates 2010h1.

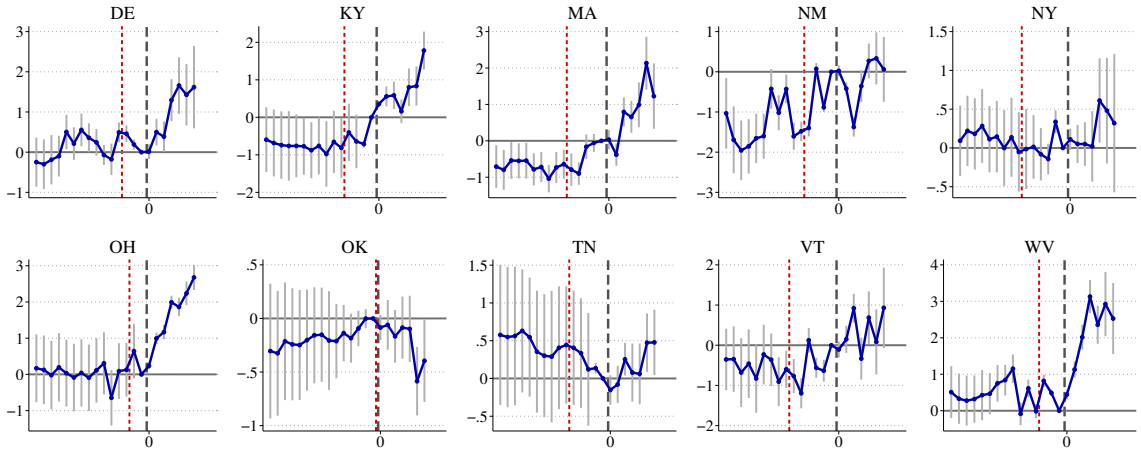
Figure B.18: Effects of the Must-Access PDMP within Ohio



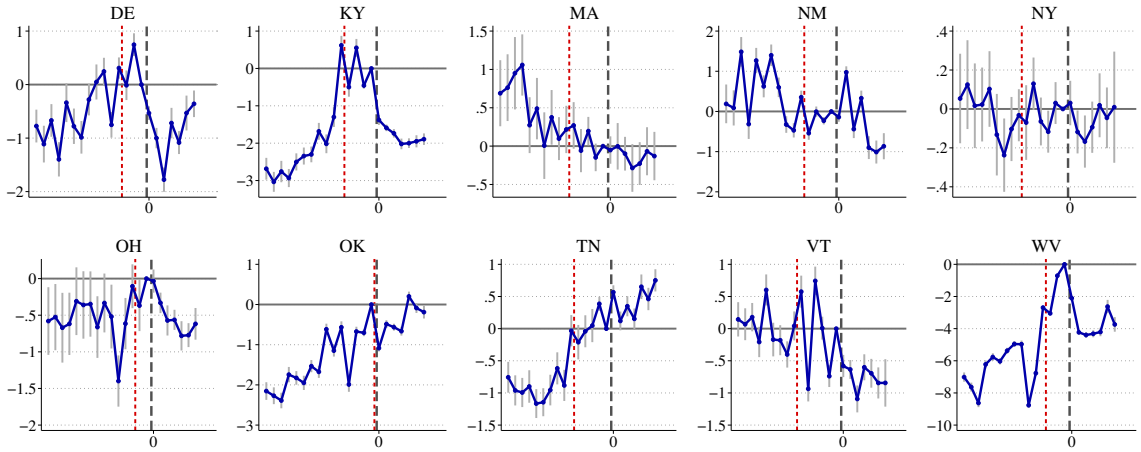
Notes: The figure presents the effect of the must-access PDMP within Ohio. The figure displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) obtained when I drop all the treated states except for Ohio. The last pre-period is omitted. The dashed red line indicates the overall effects of must-access PDMPs among the ten treated states. The (vertical) dashed gray line indicates the implementation timing of a must-access PDMP (between event times -1 and 0). The (vertical) short-dashed gray line indicates 2010h2, when the OxyContin reformulation was introduced (between event times -3 and -2). In all panels, the control sample is the baseline control sample. The dependent variable is heroin deaths per 100,000 (drug code T40.1) in Panel A, illegal-opioid-only deaths per 100,000, which involved T40.1 or T40.4 but not T40.2 or T40.3 at the time of death in Panel B, prescription-opioid-only deaths, which involved T40.2 but not T40.1, T40.3, or T40.4 in panel C, total opioid-related death rate (T40.1–T40.4) in Panel D, oxycodone (morphine equivalent) doses per capita in Panel E, and hydrocodone (morphine equivalent) doses per capita in Panel F. Observations are weighted by state population. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. The controls are identical to those in Figure 2.3. Fixed effects for state and half-year are always included.

Figure B.19: Effects of the Must-Access PDMP within a Single State

Panel A. Heroin



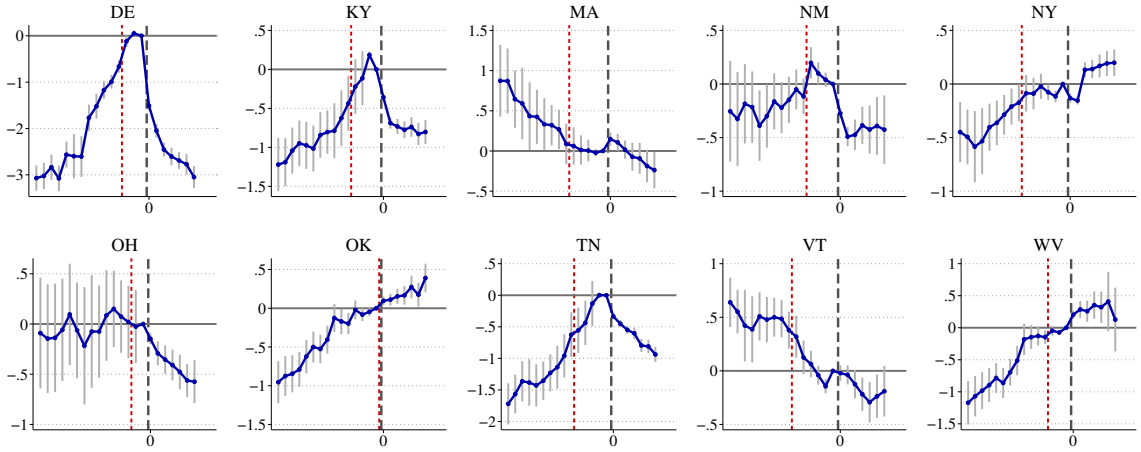
Panel B. Prescription-opioid-only



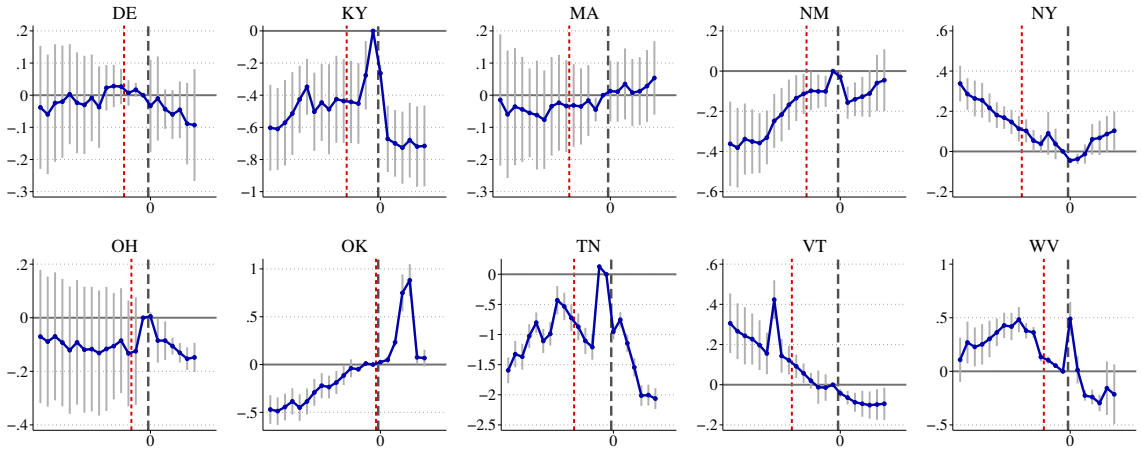
(continued)

Figure B.19: Effects of the Must-Access PDMP within a Single State (continued)

Panel C. Oxycodone doses per capita

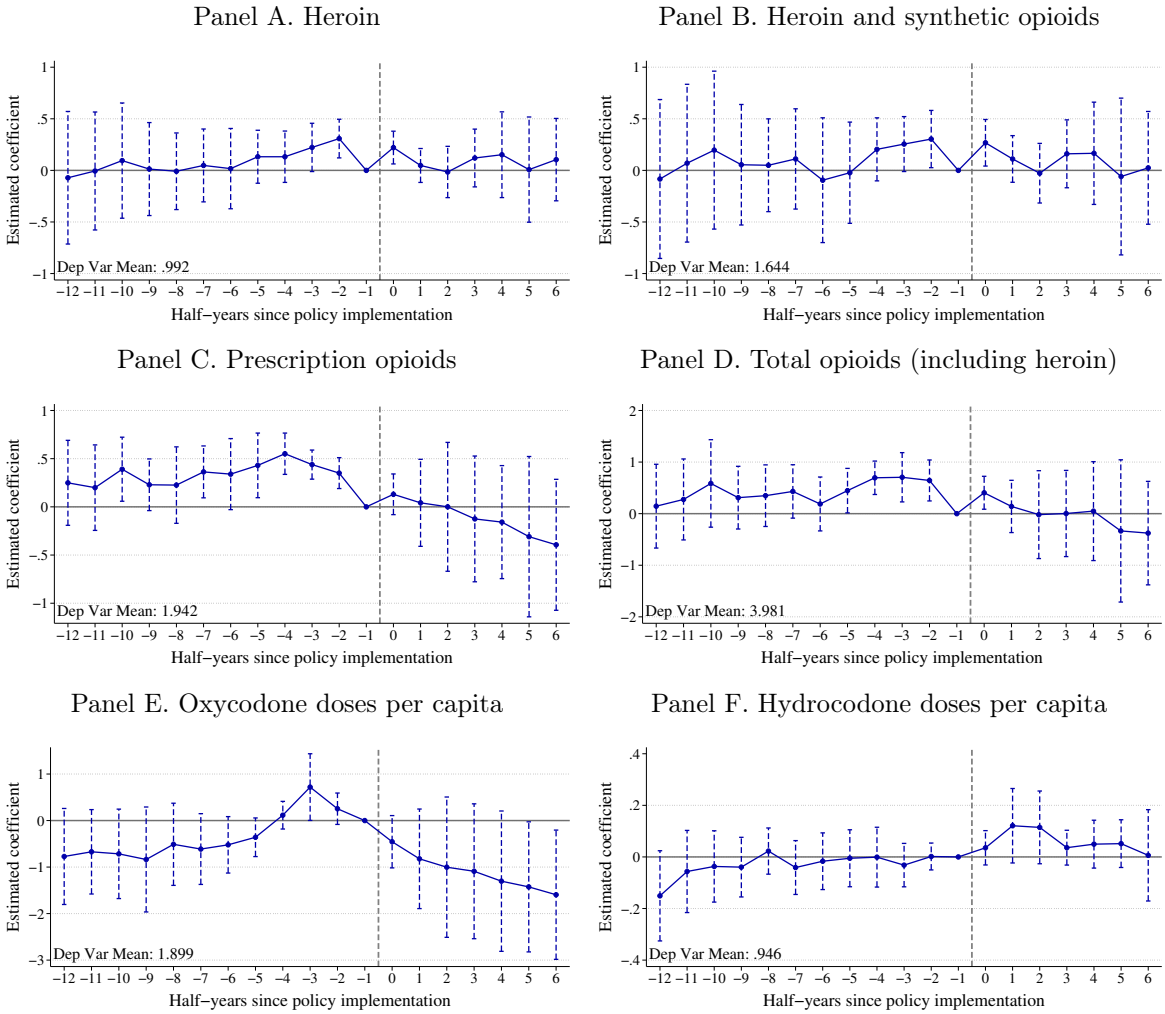


Panel D. Hydrocodone doses per capita



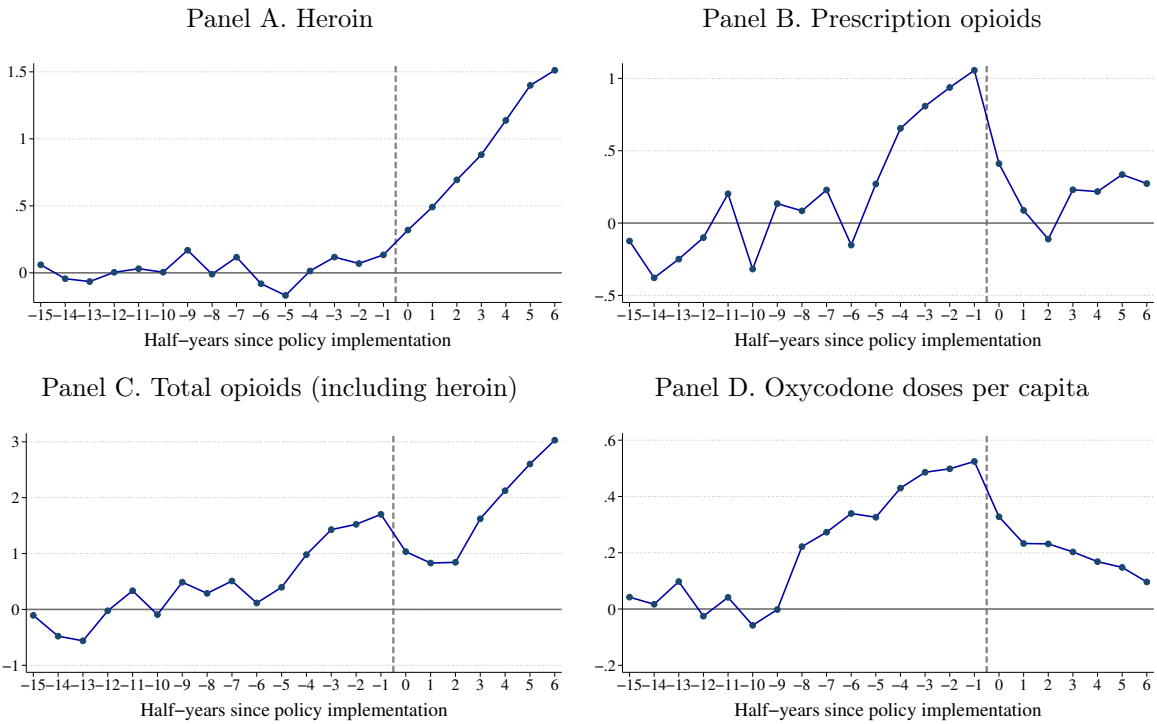
Notes: The figure shows the effect of must-access PDMP within a single treated state. The figure displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences model (equation 3.1) obtained when I drop all the treated states except for one. The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. The (vertical) dashed gray line indicates the implementation timing of a must-access PDMP (between event times -1 and 0). The (vertical) short-dashed red line indicates 2010h2, when the OxyContin reformulation was introduced. The dependent variable is heroin deaths per 100,000 (drug code T40.1) in Panel A, prescription-opioid-only deaths, which involved T40.2 but not T40.1, T40.3, or T40.4 in panel B, oxycodone (morphine equivalent) doses per capita in Panel C, and hydrocodone (morphine equivalent) doses per capita in Panel D. Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used in Panels A–B. Observations are weighted by state population. The control states are the 34 that did not implement must-access policies until 2016h2. Florida is dropped (see Appendix Section B.3). Observations are weighted by state population. The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. The controls are identical to those in Figure 2.3.

Figure B.20: Effects of Pill Mill Laws Among States without Must-Access PDMPs



Notes: The figure shows an independent effect of pain clinic laws among the 35 states that did not implement a must-access PDMP until 2016h2. Among these 35 states, Florida, Mississippi, and Texas implemented pain clinic regulations between 2009h1 and 2011h2. In the regressions, I control for a full set of indicators for pre- and post-periods around the enactment of pill mill laws, the full set of state- and time-varying covariates that I use in the baseline analysis, the ARCOS measure of OxyContin misuse interacted with the time fixed effects, an indicator for whether the state had a voluntary-access PDMP, and the fixed effects for state and half-year. The figure displays the coefficients on the indicators for pre- and post-periods. The last pre-period is omitted. For each period, I present the point estimate and its 95 percent confidence interval that is calculated using standard errors clustered at the state level. The treated sample is balanced in relative periods from -12 to +6. The distant relative periods outside the -12/+6 event time window are trimmed. The control states are balanced from 2003h1 to 2016h2. The dependent variable is heroin deaths per 100,000 (drug code T40.1) in Panel A, illegal opioid death rate (T40.1, T40.4) in Panel B, prescription opioid death rate (T40.2) in panel C, total opioid-related death rate (T40.1–T40.4) in Panel D, oxycodone (morphine equivalent) doses per capita in Panel E, and hydrocodone (morphine equivalent) doses per capita in Panel F. In Panels A–D, Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. Observations are weighted by state population. The controls are identical to those in Figure 2.3.

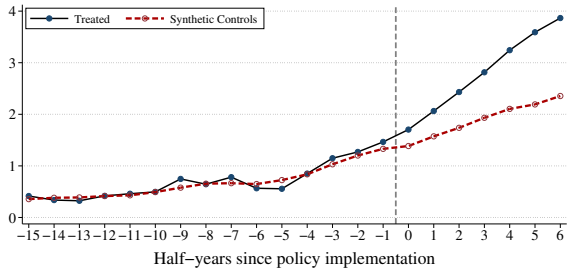
Figure B.21: Outcome Gap Between the Treated and Synthetic Control Groups



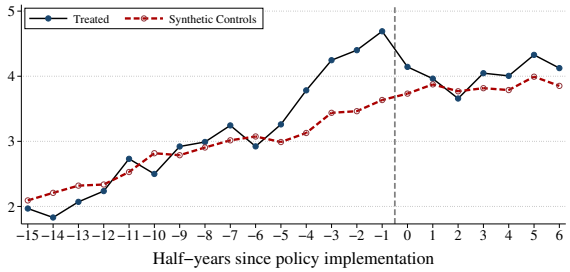
Notes: The figure displays how the (unweighted) average of the outcome gaps between the treated and synthetic control groups changes over time (see Section B.6.3). Appendix Figure B.21 plots how the (unweighted) average of these gaps changes over time. The outcome is heroin deaths per 100,000 (drug code T40.1) in Panel A, prescription opioid death rate (T40.2) in panel B, total opioid-related death rate (T40.1–T40.4) in Panel C, oxycodone (morphine equivalent) doses per capita In Panels A–C, Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. In all panels, the treated states are the 10 that implemented must-access PDMPs from 2010h2 to 2013h2, and the control states are the 10 synthetic controls (see Appendix Table B.5). Both the treated and synthetic control samples are balanced in relative periods from -15 to +6.

Figure B.22: Synthetic Control Analysis—Differential Trends

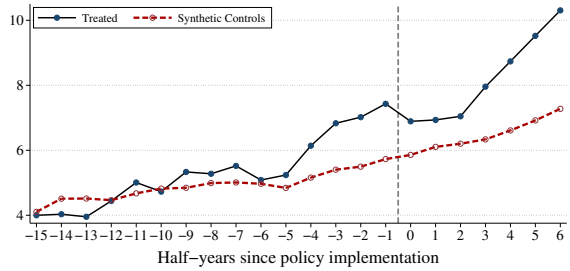
Panel A. Heroin



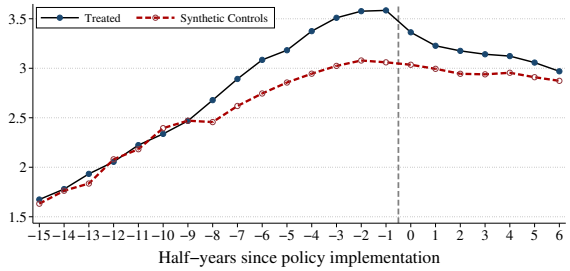
Panel B. Prescription opioids



Panel C. Total opioids (including heroin)



Panel D. Oxycodone doses per capita



Notes: The figure displays the trends in the outcomes separately for the treated and synthetic control groups. The solid black line presents how the (unweighted) average outcomes change over time in the treated states, and the dashed red blue line displays the trends for the control group. The outcome is heroin deaths per 100,000 (drug code T40.1) in Panel A, prescription opioid death rate (T40.2) in panel B, total opioid-related death rate (T40.1–T40.4) in Panel C, oxycodone (morphine equivalent) doses per capita In Panels A–C, Ruhm-corrected numbers of deaths calculated using data from the National Vital Statistics System (NVSS) are used. In all panels, the treated states are the 10 that implemented must-access PDMPs from 2010h2 to 2013h2, and the control states are the 10 synthetic controls (see Appendix Table B.5). Both the treated and synthetic control samples are balanced in relative periods from -15 to +6.

Table B.1: Effects of Must-Access PDMPs on Opioid Overdose Deaths—Summary Effect

	Overdose Deaths per 100,000					
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A. Heroin deaths and illegal opioid deaths per 100,000						
	Heroin (T40.1)			Heroin and Synthetic Opioids (T40.1, T40.4)		
Average effect	1.17**	1.00***	0.60***	1.97***	1.76***	0.99**
	(0.44)	(0.32)	(0.21)	(0.70)	(0.59)	(0.41)
R^2	0.771	0.814	0.862	0.716	0.754	0.827
Mean of dependent variable	1.151			1.884		
Panel B. Prescription opioid deaths and total opioid-related deaths per 100,000						
	Prescription Opioids (T40.2)			Total Opioids (including heroin) (T40.1–T40.4)		
Average effect	0.58***	0.44**	0.28	2.13***	1.84***	1.04**
	(0.21)	(0.17)	(0.20)	(0.69)	(0.59)	(0.42)
R^2	0.811	0.842	0.852	0.756	0.797	0.849
Mean of dependent variable	1.971			4.186		
Panel C. Illegal-opioid-only deaths and prescription-opioid-only deaths per 100,000						
	Illegal-Opioid-Only (T40.1, T40.4 but not T40.2 or T40.3)			Prescription-Opioid-Only (T40.2 but not T40.1, T40.3, or T40.4)		
Average effect	1.61**	1.44***	0.80**	0.27*	0.17	0.13
	(0.61)	(0.52)	(0.36)	(0.15)	(0.12)	(0.14)
R^2	0.710	0.749	0.820	0.815	0.843	0.846
Mean of dependent variable	1.5119			1.5123		
Ruhm (2018) correction	X	X	X	X	X	X
State fixed effects	X	X	X	X	X	X
Half-year fixed effects	X	X	X	X	X	X
Time-varying covariates		X	X		X	X
Pill mill laws		X	X		X	X
OxyContin reformulation			X			X
Number of treatment states	16	16	16	16	16	16
Number of control states	34	34	34	34	34	34
Observations	1,400	1,400	1,400	1,400	1,400	1,400

Notes: The table reports the estimated coefficients obtained when I replace a full set of indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) with a single indicator for the entire post-period. In each column, I include different sets of controls. I use the full sample of the balanced panel of state-half-year from 2003h1 to 2016h2, and Florida is dropped (see Appendix Section B.3). The treatment states are the 16 that implemented must-access PDMPs until 2016h2. The control states are the 34 that did not implement must-access policies until 2016h2, excluding Florida. The dependent variable is heroin deaths per 100,000 (drug code T40.1) in columns 1–3 of Panel A, combined deaths from heroin and synthetic opioids per 100,000 (T40.1, T40.4) in columns 4–6 of Panel A, prescription opioid deaths per 100,000 (T40.2) in columns 1–3 of Panel B, total deaths from any opioid, including heroin, per 100,000 (T40.1–T40.4) in columns 4–6 of Panel B, illegal-opioid-only deaths per 100,000 (which involved T40.1 or T40.4 but not T40.2 or T40.3 at the time of death) in columns 1–3 of Panel C, and prescription-opioid-only deaths per 100,000 (T40.2 but not T40.1, T40.3, or T40.4) in columns 4–6 of Panel C. Ruhm-corrected mortality rates are used in all regressions. Observations are weighted by state population. Controls are identical to those in columns 1, 2, and 4 of Table 2.3. Fixed effects for states and half-years are always included. Standard errors clustered at the state level are in parentheses. ***, **, * denotes statistical significance at 1%, 5%, and 10% levels respectively.

Table B.2: Robustness of Heroin Estimates—Voluntary-Access PDMPs

	Heroin Deaths per 100,000 (T40.1)				
	(1)	(2)	(3)	(4)	(5)
	Baseline	Voluntary-access PDMPs			
		Enactment date			User access
		Horwitz	PDAPS	NAMSDL	Horwitz
1-year effect (β_2)	0.42*	0.40*	0.41*	0.42*	0.40*
	(0.21)	(0.21)	(0.21)	(0.21)	(0.22)
2-year effect (β_4)	0.90***	0.89***	0.90***	0.90***	0.88***
	(0.29)	(0.29)	(0.29)	(0.29)	(0.30)
3-year effect (β_6)	1.13**	1.11**	1.12**	1.13**	1.10**
	(0.46)	(0.45)	(0.46)	(0.46)	(0.46)
Voluntary-access PDMPs		-0.19*	-0.18*	-0.07	-0.17*
		(0.11)	(0.10)	(0.09)	(0.09)
Ruhm (2018) Correction	X	X	X	X	X
State fixed effects	X	X	X	X	X
Half-year fixed effects	X	X	X	X	X
Time-varying covariates	X	X	X	X	X
OxyContin reformulation	X	X	X	X	X
Voluntary-access PDMPs		X	X	X	X
Number of treatment states	10	10	10	10	10
Number of control states	34	34	34	34	34
Observations	1,172	1,172	1,172	1,172	1,172
Mean of dependent variable	1.098	1.098	1.098	1.098	1.098
R^2	0.845	0.846	0.846	0.845	0.847

Notes: The table shows the 1-year effect (β_2), 2-year effect (β_4), and 3-year effect (β_6) from the baseline specification (equation 3.1). Although each regression includes a full set of indicators for the pre- and post-periods, I report the three coefficients above for brevity. The last pre-period is omitted. In all columns, the dependent variable is Ruhm-corrected numbers of heroin deaths per 100,000 (drug code T40.1), which are calculated using data from the National Vital Statistics System (NVSS). In column 1, I repeat my baseline estimates from column 4 of Table 2.3 Panel A. In all columns, I control for fixed effects for states and half-years, the ARCOS measure of pre-reformulation OxyContin misuse interacted with the time fixed effects, and the time-varying covariates, which are identical to those in column 4 of Table 2.3. In columns 2–5, I additionally control for voluntary-access PDMPs. Each column uses start dates of voluntary-PDMPs from a separate source: columns 2–4 use the enactment dates suggested by Horwitz et al. (2018), the PDAPS, and the NAMSDL, respectively; column 5 uses the dates PDMP data became accessible to any authorized user, suggested by Horwitz et al. (2018). Observations are weighted by state population. The treatment states are the 10 that implemented must-access PDMPs from 2010h2 to 2013h2, and the control states are the 34 that did not implement must-access policies until 2016h2. Florida is (see Appendix Section B.3). In all columns, the sample and controls are identical to those in column 4 of Table 2.3. Standard errors clustered at the state level are in parentheses. ***, **, * denotes statistical significance at 1%, 5%, and 10% levels respectively.

Table B.3: Robustness of Heroin Estimates to Removing a Single State

	Heroin Deaths per 100,000 (T40.1)										
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
	Baseline	Drop DE	Drop KY	Drop MA	Drop NM	Drop NY	Drop OH	Drop OK	Drop TN	Drop VT	Drop WV
1-year effect (β_2)	0.42*	0.42*	0.40*	0.37	0.48**	0.60***	0.25	0.47**	0.44*	0.41*	0.36*
	(0.21)	(0.22)	(0.23)	(0.24)	(0.22)	(0.22)	(0.18)	(0.23)	(0.24)	(0.21)	(0.21)
2-year effect (β_4)	0.90***	0.90***	0.91***	0.88***	0.91***	1.01***	0.61***	0.99***	1.00***	0.91***	0.85***
	(0.29)	(0.30)	(0.32)	(0.32)	(0.30)	(0.35)	(0.21)	(0.30)	(0.30)	(0.30)	(0.30)
3-year effect (β_6)	1.13**	1.12**	1.07**	1.08**	1.16**	1.43***	0.71**	1.26**	1.21**	1.13**	1.08**
	(0.46)	(0.46)	(0.49)	(0.51)	(0.47)	(0.47)	(0.35)	(0.48)	(0.50)	(0.46)	(0.47)
Ruhm (2018) correction	X	X	X	X	X	X	X	X	X	X	X
State fixed effects	X	X	X	X	X	X	X	X	X	X	X
Half-year fixed effects	X	X	X	X	X	X	X	X	X	X	X
Time-varying covariates	X	X	X	X	X	X	X	X	X	X	X
OxyContin reformulation	X	X	X	X	X	X	X	X	X	X	X
Number of treatment states	10	9	9	9	9	9	9	9	9	9	9
Number of control states	34	34	34	34	34	34	34	34	34	34	34
Observations	1,172	1,150	1,150	1,150	1,150	1,150	1,150	1,150	1,150	1,150	1,150
Dep var mean	1.098	1.097	1.091	1.085	1.092	1.080	1.060	1.109	1.106	1.098	1.096
R^2	0.845	0.845	0.843	0.840	0.845	0.841	0.831	0.845	0.850	0.845	0.845

Notes: The table shows the sensitivity of the baseline estimates for heroin mortality to removing a single treatment state. The table reports the 1-year effect (β_2), 2-year effect (β_4), and 3-year effect (β_6) from the baseline specification (equation 3.1). Although each regression includes a full set of indicators for the pre- and post-periods, I report the three coefficients above for brevity. The last pre-period is omitted. In all columns, the dependent variable is Ruhm-corrected numbers of heroin deaths per 100,000 (drug code T40.1), which are calculated using data from the National Vital Statistics System (NVSS). Observations are weighted by state population. In column 1, the treatment states are the ten that implemented must-access PDMPs from 2010h2 to 2013h2. In each of columns 2–11, I remove one of these ten states from the analysis sample. In all columns, the treated sample is balanced in relative periods from -15 to +6, and the distant relative periods outside the -15/+6 window are trimmed. In all columns, the control states are the 34 that did not implement must-access policies until 2016h2, and the baseline control sample is balanced from 2003h1 to 2016h2. Florida is dropped (see Appendix Section B.3). In all columns, the controls are identical to those in column 4 of Table 2.3. Fixed effects for state and half-year are always included. Standard errors clustered at the state level are in parentheses. ***, **, * denotes statistical significance at 1%, 5%, and 10% levels respectively.

Table B.4: Robustness of Other Estimates

	Overdose Deaths per 100,000							
	(1) Baseline	(2) Reported mortality	(3) Voluntary PDMPs	(4) Include FL	(5) Add MMLs	(6) Add NALs	(7) Good Sam laws	(8) Alternative dates
Panel A. Heroin and synthetic opioid deaths per 100,000 (T40.1, T40.4)								
1-year effect (β_2)	0.40	0.49	0.39	0.41	0.23	0.48	0.43	0.33
	(0.35)	(0.37)	(0.35)	(0.37)	(0.42)	(0.35)	(0.35)	(0.33)
2-year effect (β_4)	1.36***	1.56***	1.35***	1.35***	1.26**	1.52***	1.41***	1.29**
	(0.49)	(0.50)	(0.48)	(0.50)	(0.53)	(0.48)	(0.49)	(0.47)
3-year effect (β_6)	2.08**	2.34***	2.06**	1.98**	1.98**	2.07**	2.11**	1.64*
	(0.90)	(0.85)	(0.89)	(0.91)	(0.92)	(0.86)	(0.90)	(0.97)
Mean of dependent variable	1.799	1.423	1.799	1.7994	1.799	1.799	1.799	1.792
R^2	0.807	0.797	0.808	0.807	0.814	0.815	0.808	0.813
Panel B. Prescription opioid deaths per 100,000 (T40.2)								
1-year effect (β_2)	-0.54**	-0.52**	-0.54**	-0.48*	-0.56**	-0.52*	-0.55**	-0.57**
	(0.25)	(0.21)	(0.25)	(0.25)	(0.26)	(0.26)	(0.26)	(0.28)
2-year effect (β_4)	-0.27	-0.21	-0.28	-0.21	-0.28	-0.24	-0.29	-0.25
	(0.32)	(0.28)	(0.33)	(0.32)	(0.32)	(0.34)	(0.33)	(0.34)
3-year effect (β_6)	-0.27	-0.22	-0.27	-0.22	-0.28	-0.27	-0.28	-0.21
	(0.35)	(0.33)	(0.35)	(0.33)	(0.35)	(0.36)	(0.36)	(0.37)
Mean of dependent variable	1.984	1.533	1.984	2.039	1.984	1.984	1.984	2.016
R^2	0.861	0.843	0.862	0.860	0.862	0.862	0.862	0.867
Panel C. Total opioid-related deaths per 100,000 (T40.1–T40.4)								
1-year effect (β_2)	-0.17	-0.07	-0.18	-0.11	-0.37	-0.09	-0.15	-0.33
	(0.42)	(0.40)	(0.42)	(0.44)	(0.48)	(0.45)	(0.43)	(0.38)
2-year effect (β_4)	0.84*	1.06**	0.82*	0.90*	0.71	0.99**	0.86*	0.71
	(0.46)	(0.46)	(0.46)	(0.47)	(0.49)	(0.48)	(0.48)	(0.43)
3-year effect (β_6)	1.61*	1.87**	1.60*	1.59*	1.49	1.61*	1.63*	1.16
	(0.89)	(0.84)	(0.89)	(0.88)	(0.90)	(0.87)	(0.89)	(0.90)
Mean of dependent variable	4.126	3.210	4.126	4.205	4.126	4.126	4.126	4.140
R^2	0.842	0.837	0.842	0.841	0.848	0.845	0.842	0.851
Ruhm (2018) correction	X		X	X	X	X	X	X
Number of treatment states	10	10	10	10	10	10	10	8
Number of control states	34	34	34	35	34	34	34	31
Observations	1,172	1,172	1,172	1,200	1,172	1,172	1,172	1,044

Notes: Notes: The table tests the robustness of my baseline heroin mortality estimates to alternative explanations. The table shows the 1-year effect (β_2), 2-year effect (β_4), and 3-year effect (β_6), obtained from the baseline specification (equation 3.1). Although each regression includes a full set of indicators for pre- and post-periods, I report the three coefficients above for brevity. The last pre-period is omitted. Observations are weighted by state population. In all columns in panel A, the dependent variable is combined deaths from heroin and synthetic opioids per 100,000 (drug codes T40.1, T40.4). In all columns in panel B, the dependent variable is prescription opioid deaths per 100,000 (drug codes T40.2–T40.3). In all columns in panel C, the dependent variable is total deaths from any opioid, including heroin (drug codes T40.1–T40.4). In column 1 of Panel A, I repeat my baseline estimates from column 4 of Table 2.3 Panel B. In column 1 of Panels B and C, I repeat my preferred estimates from column 4 of Table 2.5. In column 2, I use the raw reported numbers of deaths, and the other columns use the Ruhm-corrected numbers of deaths. Both the corrected and reported numbers of deaths are calculated using data from the National Vital Statistics System (NVSS). In column 3, I control for an indicator for whether a state had a voluntary-access PDMP. In column 4, I include Florida in the analysis sample. Florida is dropped from the control group in the other columns (see Appendix Section B.3). In columns 5–7, I include several other co-occurring opioid-related policies one by one: in column 5, I include a time-varying indicator for whether the state had medical marijuana laws (MMLs) as well as an indicator for whether the state had legal and operational dispensaries; in column 6, I add an indicator for whether the state had naloxone access laws (NALs), and in column 7, I add an indicator for whether the state had Good Samaritan overdose prevention laws. Columns 5–7 suggest that my estimates are robust to including several other co-occurring state opioid-related policies. In column 8, I use alternative start dates of must-access PDMPs listed in the third column of Table 2.1, and in this estimation, the treated states are the 8 that implemented must-access PDMPs from 2010h2 to 2013h2, and the control states are the 31 that did not implement must-access policies until 2016h2, excluding Florida. In all columns, the distant event periods outside the -15/+6 window are trimmed. In all columns, I control for fixed effects for state and half-year, an indicator for whether a state had a pill mill law, the ARCOS measure of OxyContin misuse interacted with the half-year fixed effects, and the time-varying covariates that are identical to those in column 4 of Table 2.3. Standard errors clustered at the state level are in parentheses. ***, **, * denotes statistical significance at 1%, 5%, and 10% levels, respectively.

Table B.5: Synthetic Control States

Treated State	Synthetic Control State					
Panel A. Heroin deaths per 100,000 (T40.1)						
Delaware	30.1% UT,	20.5% AZ,	17.7% ID,	10.9% DC,	7% KS,	13.9% Other
Kentucky	54% WI,	23.5% AK,	16.6% AZ,	4.9% DC,	1% MN,	
Massachusetts	48.6% AK,	19.6% DC,	13.6% NH,	8.9% MD,	4% ME,	5.2% Other
New Mexico	47.4% MO,	28.3% PA,	24.3% UT			
New York	17.7% WA,	16.3% NH,	14.1% NC,	13.7% WY,	13.5% MD,	24.7% Other
Ohio	65.9% MO,	23.8% UT,	6.1% MD,	4.1% DC		
Oklahoma	68.7% ND,	13.3% NH,	7.1% NE,	4.7% NC,	3.7% AZ,	2.5% Other
Tennessee	34.4% MS,	34.2% ND,	12.1% AZ,	7.5% AL,	5.5% KS,	6.3% Other
Vermont	54.1% MN,	31% AK,	8% IL,	6.9% WY		
West Virginia	24.5% KS,	20.6% MO,	19.8% MT,	18.6% HI,	11.3% OR,	5.2% UT
Panel B. Prescription opioid deaths per 100,000 (T40.2)						
Delaware	48.5% WY,	38.1% IN,	11.7% AK,	1.6% UT		
Kentucky	74.8% UT,	25.2% WY				
Massachusetts	79.2% TX,	14.7% ND,	4.6% DC,	1.5% CO		
New Mexico	97.6% UT,	2.4% AK				
New York	25.5% IA,	22.6% IL,	21.1% MD,	17.5% HI,	8.7% DC,	4.5% Other
Ohio	34.3% AZ,	29.1% NH,	16.5% WY,	15.1% HI,	4.9% AR,	0.1% SD
Oklahoma	55.7% UT,	28.6% AK,	15% WY,	0.7% NH		
Tennessee	35.9% WY,	31.9% UT,	27.1% AZ,	5.1% PA		
Vermont	35% ID,	23.2% UT,	19.1% ND,	12.3% DC,	6.1% TX,	4.4% WY
West Virginia	100% UT					
Panel C. Total opioid-related deaths per 100,000 (T40.1–T40.4)						
Delaware	48.8% MO,	22.1% WY,	12.2% CO,	10.1% AK,	5.3% AL,	1.4% NH
Kentucky	75.9% UT,	24.1% MO				
Massachusetts	61.1% IL,	19.1% CO,	8.1% HI,	6.4% UT,	2.3% KS,	3% Other
New Mexico	100% UT					
New York	29.7% IA,	22.6% KS,	16.9% DC,	13.6% MD,	12.9% HI,	4.5% Other
Ohio	89.6% AZ,	4.9% MO,	2.5% UT,	1.8% WY,	1.1% MD	
Oklahoma	57.8% UT,	26.4% AK,	15.8% NH,			
Tennessee	47.3% MO,	23.2% UT,	10.7% PA,	7.2% WI,	6.5% ME,	5% Other
Vermont	54.8% IL,	37.5% UT,	5.2% WY,	2.5% DC		
West Virginia	100% UT					
Panel D. Oxycodone doses per capita						
Delaware	100% AZ					
Kentucky	54.1% GA,	27.9% PA,	18% MD			
Massachusetts	23.8% AK,	19.6% UT,	13.3% MO,	12.9% NE,	11.2% WA,	19.2% Other
New Mexico	42.3% MD,	40.8% CO,	17% GA			
New York	66.7% GA,	26% IN,	7.3% NH			
Ohio	38.2% ME,	26.8% NH,	18.6% WA,	8.6% MO,	7.7% PA	
Oklahoma	27.2% UT,	25.6% AR,	21.3% MI,	12.1% OR,	9% AZ,	4.8% NH
Tennessee	50.2% ME,	43.4% AZ,	6.4% PA			
Vermont	29.2% WA,	20.3% MD,	16% NE,	12.4% MI,	10.5% NH,	11.6% Other
West Virginia	37.9% NH,	28.2% MD,	20.9% PA,	13% ME		

Notes: This table shows how synthetic control states included in the sample from Appendix Figures B.21 and B.22 are constructed. Each synthetic control state is calculated as a linear combination of the subset of my 34 control states (Florida is excluded from the control sample; see Appendix Section B.3). Values are independently rounded, and for synthetic states with more than six control states, remaining states are grouped into an "other" category. The outcome is heroin deaths per 100,000 (drug code T40.1) in Panel A, prescription opioid death rate (T40.2) in panel B, total opioid-related death rate (T40.1–T40.4) in Panel C, oxycodone (morphine equivalent) doses per capita

B.2 Comparisons with the Prior Literature

In this section, I compare my study with the prior literature on two dimensions—data period and model specification. Because Meinhofer (2018a) is most closely related to my paper, I focus on comparing my study with hers. For simplicity, Meinhofer (2018a) is often referred to as Meinhofer in this section. In Appendix Figure B.14, I show how my heroin mortality results change if I drop recent data or use the approach suggested by Meinhofer with my analysis sample. Similarly, in Appendix Figure B.15, I show how my replication of Meinhofer's event study results for heroin mortality change if I use my model specification instead of hers, while keeping everything else unchanged. Overall, my exercises suggest two things. First, using a longer period, which allows for including additional post-periods and several more implementations of must-access PDMPs, is key to identifying the overall spillover effects of the policy. Second, the heroin estimates from my model specification provide stronger evidence of the spillover effect of must-access PDMPs compared with those from Meinhofer's event study specifications, regardless of whether I include a longer data period or not. The estimates from my model specification are statistically more significant in the post-period and better address concerns about pre-trends in heroin mortality. These differences generated by the specification choices are more pronounced when I use a longer period.

B.2.1 Data Period

In Appendix Figure B.14, I show the consequences of employing more recent data. Each column of Appendix Figure B.14 corresponds to different sample periods. In the left column (Panels A, C, and E), I use my full sample (2003h1–2016h2), and in the right column (Panels B, D, and F), I drop the 2014h1–2016h2 period and reconstruct the sample so that the treated states are balanced in relative periods. The sample from the right column includes the five treated states that are consistently observed from nine half-years prior to implementation and three half-years after implementation. The number of treated states observed in each event

time period is presented in the parentheses below that period.[1]

The first row (Panels A and B) shows how my baseline estimates for heroin mortality (Panel A) change if I drop the recent data period. The two panels display the coefficients on the indicator for pre- and post-periods from the baseline specification (equation 3.1) on the full sample (Panel A) and on the sample without the recent data (Panel B). As seen in Panel B, even if I drop the period after 2013, I still find suggestive evidence of the spillover effects on heroin mortality. However, compared with the estimates in Panel A, the estimates in Panel B have much larger effect sizes, and all the coefficients in Panel B become close to zero and insignificant if I drop Ohio, one of the five treated states. Overall, Panels A and B suggest that using a longer data period, which allows for including several additional implementations of must-access PDMPs and additional post-periods, is crucial to obtaining robust estimates that reflect the overall spillover effects on heroin mortality.

B.2.2 Model Specification

In this paper, I provide causal evidence that must-access PDMPs have increased heroin mortality, and my estimates are robust to controlling for several other co-occurring state and national opioid-related policies, including the 2010 OxyContin reformulation. Above, I show that using more recent data is crucial to identifying these effects of must-access policies. However, not only a longer data period but also my model specification contributes to my findings. Because the importance of controlling for the reformulation is discussed in Section 3.5, I focus on describing the consequences of other specification choices in this section. Because Meinhofer (2018a) is most closely related to my paper, I focus on comparing my econometric model with hers.

Below, I present my baseline specification (equation 3.1, provided below for convenience as equation A1) and the event study specifications used in Meinhofer (equations M1 and M2). Equation M1 is Meinhofer's (2018a) preferred event study specification. Note

[1]While my full sample (used in the left column) excludes one treated state, Nevada, to include more pre-periods, the sample from the right column does not exclude Nevada because it has five treated states only and the estimates are not robust to excluding one of them. The difference in the number of pre-periods across the two samples is attributable to the fact that Nevada, which implemented the earliest must-access PDMP in the nation, has nine pre-periods in my sample.

that the notations in equations M1 and M2 slightly differ from those in the original equations presented in Meinhofer (2018a) (I use my preferred notations for an easier comparison of specifications), although they are fundamentally the same. To distinguish between year in equations M1 and M2 and half-year in equation 3.1, I change the subscript for my half-year variable from t to h to indicate half-year, only in this section. The model specifications used in the two studies are as below.

Equation (3.1) from my paper:

$$y_{sh} = \alpha_s + \alpha_h + \sum_{k \neq -1} \beta_k \mathbf{1}(Policy_{sk}) + X_{sh}\delta + oxy_s \cdot \omega_h + \varepsilon_{sh} \tag{A1}$$

Equation (4) from Meinhofer (2018a):

$$ln(Y_{stq} + 1) = \alpha_s + \alpha_t + \alpha_q + \sum_{k \neq -1} \beta_k \mathbf{1}(Policy_{sk}) + \gamma ln(P_{stq}) + \theta_s \cdot t + \varepsilon_{stq} \tag{M1}$$

Equation (3) from Meinhofer (2018a):

$$ln(Y_{stq} + 1) = \alpha_s + \alpha_t + \alpha_q + \sum_{k \neq -1} \beta_k \mathbf{1}(Policy_{sk}) + \gamma ln(P_{stq}) + \varepsilon_{stq} \tag{M2}$$

where $ln(Y_{stq} + 1)$ is the log of quarter-level overdose deaths, and Meinhofer (2018a) adds 1 to all outcomes to avoid losing observations with count zero. P_{stq} is state population, and $y_{sh} = Y_{sh}/P_{sh} * 100,000$ is overdose deaths per 100,000. α_s are state fixed effects, and α_t, α_h, and α_q are fixed effects for year, half-year, and quarter (seasonality), respectively. $oxy_s \cdot \omega_t$ in equation 3.1 indicates the measure of pre-reformulation OxyContin use interacted with the half-year fixed effects. $\theta_s \cdot t$ in equation M2 is state-specific (year-level) trends. ε_{stq} is the error term. Note that the regressions in my study are weighted by state population, while those in Meinhofer are not; to be consistent, throughout this section, the regressions estimating my specification (equation 3.1) are weighted by population, while the regressions estimating Meinhofer's (equations M1 and M2) are unweighted. Although not reported in the paper, my baseline heroin estimates are stable across the weighted and unweighted regressions.

In Appendix Figure B.14, each row estimates one of the three specifications: the top row (Panels A and B) estimates my baseline specification (equation 3.1), the middle

(Panels C and D) estimates Meinhofer's preferred event study specification (equation M1), and the bottom (Panels E and F) estimates Meinhofer's alternative event study specification (equation M2). Panel C shows that my results from the full sample (Panel A) are substantially affected if I use Meinhofer's preferred specification, while everything else that includes the full sample remains unchanged. If I use the same specification as in Panel C and drop the period after 2013, I obtain the results shown in Panel D, which are expected to be similar to Meinhofer's heroin results (Meinhofer uses data through 2013). In fact, Panel D has a results pattern similar to my replication of Meinhofer (see Panel A of Figure B.15), although balancedness, data frequency, and the time window length are different across the two figures (the replication of Meinhofer is explained in detail below). All the panels in Figure B.14 use half-year frequency data, and thus Panels C–F employ equations M1 and M2, which are based on quarter frequency, by including half-year fixed effects instead of fixed effects for year and quarter (seasonality).

Compared with Panels A and B, which are based on my specification, Panels C and D show that the estimated policy effects are statistically less significant, and there is evidence of a pre-trend prior to policy implementation. In particular, with the more recent data in Panel C, using equation M1 leads to a clear upward pre-trend in the entire pre-period, providing evidence of a violation of the parallel trends identification assumption. Finally, Panels E and F display the results obtained using equation M2, which drops state-specific time trends from equation M1. Panels E and F suggest that the estimates presented in Panels C and D, which are based on equation M1, are sensitive to dropping state-specific time trends. The sizes of the coefficients are much smaller in Panels E and F than in Panels C and D, and Panel F suggests no effect of the policy on heroin-related deaths.

B.2.3 Replication of Meinhofer (2018a)

As discussed above, Figure B.14 suggests that the estimates from my specification, as compared with those from Meinhofer's preferred specification, provide more compelling heroin results. However, one may have a concern about whether other factors drive these findings, such as legal coding, Ruhm (2018) correction, or data frequency. To address this concern, I perform an exercise similar to that in Appendix Figure B.14 but using the

replication of Meinhofer (2018a). I first replicate Meinhofer's event study results for heroin-related deaths and then test how these estimates change if I use my specification (equation 3.1) instead, while keeping everything else unchanged.

Panel A of Appendix Figure B.15 shows my replication of Meinhofer's event study results for heroin-related deaths, and Panel B presents the estimates that obtained when I use my baseline specification (equation 3.1) instead, while everything else, including the analysis sample from Panel A, remains unchanged. Although I cannot directly compare the estimates from my baseline model with those from Meinhofer's log-transformed model, the estimates from Panels A and B have similar trends in both the pre- and post-periods. However, the estimates in Panel B better address the pre-treatment differences between the treated and control groups, and the coefficients in the post-period are statistically more significant compared with those presented in Panel A. These findings are consistent with those from Figure B.14.[2] In summary, Appendix Figures B.14 and B.15 suggest that regardless of whether more recent data are included or not, my specification (equation 3.1) allows for clearer evidence of the spillover effect on heroin deaths than equation M1 and that the differences generated by the specification choices are more pronounced when I include additional years of data.

B.3 Dropping Florida

In the 2000s, increasing numbers of pill mills caused a dramatic rise in the opioid supply in Florida. As a result, Florida was at the center of the nation's opioid epidemic in the late 2000s and was an extreme outlier both in levels of and trends in opioid supply (Meinhofer 2016). In response, Florida passed several laws in 2010 and 2011 that strictly regulated pain clinics. These aggressive regulations led to a huge drop in the opioid supply in Florida. Appendix Figure B.17 displays the trends in each state's per capita legal supply of oxycodone. This figure shows that Florida is an outlier that experienced both a sharp

[2]Note that the sample used in Panels B and D of Figure B.14 is balanced in relative (half-year) periods from -9 to +3, while the sample used in Figure B.15 is unbalanced in relative (quarter) periods from -9 to +7 (the corresponding half-year event time window is -4.5/+3.5). Although the event time windows and the number of treated states included are different in the two figures, the results patterns in the overlapped relative periods are similar across the figures.

increase and decrease in oxycodone supply within a decade. There was a large spike in Florida's per capita legal supply of oxycodone throughout the 2000s until the pill mill peak in 2010, and then the oxycodone supply began to decrease sharply as a result of aggressive regulations in 2010 and 2011.

I exclude Florida from my control group for all analyses in this study. The key assumption of my difference-in-differences model is that, in the absence of must-access PDMPs, the trends in the outcomes would have been the same across the treated and control groups. However, given that Florida experienced dramatic policy changes around the time of my treatment states' implementation of the must-access PDMPs, including Florida may potentially violate the parallel assumption. In fact, I find that the results for oxycodone doses per capita are sensitive to whether I include Florida in my control group or not, as shown in Appendix Figure B.16. The first panel of the top row of Appendix Figure B.16 presents the oxycodone estimates from my baseline specification (equation 3.1) obtained using a sample that includes my ten treatment states and all of the 35 states that did not implemented a must-access PDMP until 2016h2, including Florida. As illustrated in Appendix Figure B.16, the estimates from this sample are sensitive to removing Florida. In any panels without Florida, I observe a sudden decrease in oxycodone supply following policy implementation, and a negative trend in oxycodone supply is found in the entire post-period. However, once I include Florida in my analysis, all coefficients for the post-periods become close to zero. Although not reported, after dropping Florida, my oxycodone results are robust to removing one of the treated or control states. In contrast to the sensitivity of my oxycodone results to including Florida, my mortality results are robust to whether Florida is included, as shown in Section 2.6 (see columns 1 and 4 of Table 2.6 and those of Appendix Table B.4).

B.4 Must-Access PDMP in Ohio

In this section, I explore the policy effect in Ohio in particular and propose three possible explanations for the strong effect of Ohio's must-access PDMP, which relied on provider suspicion. In Section 2.6, I test the robustness of the baseline heroin mortality estimates to removing one treated state (see Appendix Table B.3, the corresponding regression coefficients

are presented in Panel A of Appendix Figure B.11). As shown in Appendix Table B.3, regardless of which treated state is dropped, the estimates are statistically significant and qualitatively similar to the baseline estimates. However, when I drop Ohio, the magnitudes of the heroin mortality estimates are slightly attenuated, although the coefficients for the two- and three-year effects remain statistically significant (see column 7 of Appendix Table B.3).

In this section, I first explore the effect of must-access policy within Ohio and show that Ohio's policy had stronger effects on my outcomes than the policies in the other treated states. I then propose three possible explanations for the strong impact of Ohio's initial must-access PDMP on the heroin death rate—a sharp increase in PDMP utilization, the existence of a complementary law, and high accessibility of heroin. As mentioned in Section 2.5.6, Ohio implemented its initial must-access PDMP in 2011h2 and then strengthened its must-access law in 2015h2 (at event time +8). However, the strengthened law cannot explain why my heroin estimates are affected when I remove Ohio because the most distant post-period in my analysis is event time +6 (3 years after implementation) and Ohio strengthened its law at event time +8 (4 years after the initial implementation). Therefore, in this section, I focus on discussing why Ohio's initial must-access PDMP, which relied on provider suspicion, had stronger effects on the outcomes than the must-access policies in the other treated states. Throughout this section, I use the phrase *Ohio's initial must-access PDMP* synonymously with *Ohio's must-access PDMP*. Also, note that Ohio has the second-largest population among my ten treated states, and my regressions are weighted by state population. The strong impact of Ohio's (initial) must-access policy and Ohio's large population explain why the coefficients become smaller when I remove Ohio.

B.4.1 Effects of Must-Access PDMP within Ohio

I first investigate how the must-access PDMP impacted the mortality outcomes within Ohio. In Appendix Figure B.18, I present the estimates from the baseline specification (equation 3.1) that I obtain by dropping all the treated states except Ohio.[3] The estimates in blue show the impact of Ohio's must-access PDMP, and the point estimates in red are my

[3]An alternative way to estimate the impact of Ohio's PDMP is to use the full sample and interact an indicator for Ohio with the full set of indicators for pre- and post-periods. However, the results I observe when limiting my treatment group to Ohio are similar to those I obtain when including the interactions.

baseline estimates, indicating the overall effects of the policy among the ten treated states, including Ohio. The (vertical) dashed gray line indicates the implementation timing of a must-access PDMP (between event times -1 and 0). The (vertical) short-dashed gray line indicates 2010h2, when the OxyContin reformulation was introduced (between event times -3 and -2).

Appendix Figure B.18 suggests that Ohio's must-access PDMP had stronger impacts on most outcomes compared with the overall effects among the ten treated states. Following implementation of the must-access PDMP policy, heroin and illegal opioid mortality sharply increased (Panels A and B), and these increases coincided with sudden decreases in prescription opioid mortality (Panel C) and the legal supply of opioids (Panels E and F). Note that a sharp decrease in prescription opioid mortality at event time -5 is due to a temporary drop in Ohio's prescription opioid deaths in 2009h1. Overall, in Appendix Figure B.18, I observe a clear and strong substitution pattern between legal and illegal opioid mortality following Ohio's policy implementation and I find no apparent pre-trends in the outcomes, evidence of that supports the parallel trends assumption. As mentioned above, Ohio has the second-largest population among my ten treated states, and my regressions are weighted by state population.[4] The strong impact of Ohio's must-access policy and Ohio's large population explain why my heroin mortality estimates become smaller when I remove Ohio. Below, I propose three possible explanations that may account for the strong effects of the must-access PDMP in Ohio.

B.4.2 PDMP Utilization

The first plausible explanation for the strong impact of Ohio's must-access PDMP is that Ohio's policy was associated with a dramatic increase in PDMP utilization. In 2011h2, Ohio enacted its initial must-access law, which required prescribers to review a patient's prescription history at the beginning of treatment and annually after that, if they had reason to believe that treatment with controlled substances in Schedules II–V would exceed

[4]This is another reason why removing Ohio has a relatively larger effect on the estimates than dropping one of the other treated states. Although not reported in the revised paper, I find similar policy effects regardless of whether I weight observations by state population, but removing Ohio has a smaller effect on the estimates with the unweighted regressions.

12 continuous weeks (Urahn 2016). Even though this initial must-access law primarily relied on provider suspicion, the utilization of the PDMP increased dramatically, from 911,000 reports requested in 2010, to 1.8 million in 2011, 5.4 million in 2012, 7.3 million in 2013, 10.8 million in 2014, and 16.5 million in 2015 (the 2016 Ohio Automated Rx Reporting System (OARRS) Annual Report[5]).

Other actions may also have increased PDMP participation in Ohio. In 2012h2, the state published guidelines for prescribing opioids in emergency departments, and in 2013h2 guidelines were established for the long-term prescription of opioids, both initiatives may have encouraged the utilization of the PDMP and reduced opioid prescriptions. For example, the Governor's Cabinet Opiate Action Team (2014) encouraged providers to "consider checking Ohio Automated Rx Reporting System (OARRS) for all patients who will receive an opiate," demonstrating that Ohio encouraged providers to use the PDMP in situations beyond those prompted by their suspicion.

In 2015h2, Ohio strengthened the must-access law by adding further requirements for the utilization of the PDMP. Based on the updated laws, prescribers must request a PDMP report on a patient under certain circumstances, even without provider suspicion. Following implementation of the updated mandate in 2015, PDMP utilization increased again, from 1.2 million queries in April to 1.4 million queries in September, reflecting a 17% increase (PDMP Center of Excellence 2016).

Transition from Ohio's initial must-access PDMP in 2011h2 to the updated program in 2015h2 occurred gradually, rather than a single implementation date marking a sudden increase in PDMP utilization (Urahn 2016). As a result of Ohio's consistent efforts, utilization of Ohio's PDMP system increased dramatically, and opioid prescriptions decreased sharply. Following the implementation of Ohio's initial must-access PDMP in 2011, the rate of individuals who see five or more prescribers and five or more pharmacies in a three month period to obtain controlled substances (commonly referred to as doctor shopping) decreased by over half, by the last quarter of 2013 (PDMP Center of Excellence 2014). According to the 2016 OARRS Annual Report (see footnote 61), for the period 2012 to 2016, the total doses of opioids dispensed to Ohio patients decreased by 162 million doses

[5]https://www.ohiopmp.gov/documents/Annual%20Report%20(2016).pdf (last accessed May 2020)

(or 20.4%), while the number of opioid prescriptions issued to Ohio patients decreased by 2.5 million (or 20%); during that same period, the state experienced a 78.2% decrease in the number of individuals who see multiple prescribers to obtain controlled substances illicitly. Ohio is one of the potential models for states looking to mandate PDMP use (PDMP Center of Excellence 2016). The dramatic increase in PDMP utilization, which resulted from implementing the initial must-access PDMP, publishing guidelines on opioid prescriptions, and a sharp decrease in opioid prescriptions as a result of increasing PDMP utilization, can explain why Ohio's mandate had dramatic effects on heroin and other opioid mortality even if it relied on provider suspicion.

B.4.3 Complementary Law—Pill Mill Law

Another initiative that may account for the strong effects of the must-access PDMP in Ohio is the enactment of a complementary law. Around the time of policy implementation of the must-access PDMP, three states (Kentucky, Ohio, and Tennessee) also enacted pill mill laws, which impose strict regulations on pain clinics to prevent them from issuing opioid prescriptions without medical indication. Buchmueller and Carey (2018) view pain clinic laws as complements to must-access laws, as they target a slightly different channel of misuse than PDMP policies. Must-access laws target a large fraction of providers, while pain clinic laws directly regulate the behavior of the small share of providers, who prescribe high volumes of opioids without medical indication. Ohio and Kentucky, which implemented the must-access PDMP a year after implementing the pill mill law, experienced large decreases in opioid prescriptions following the mandate (PDMP Center of Excellence 2016). Appendix Figure B.19 presents the effect of the must-access PDMP within each state by plotting the estimates from the baseline specification (equation 3.1) that I obtain by dropping all the treated states except one. The (vertical) dashed gray line indicates the implementation timing of a must-access PDMP (between event times -1 and 0). The (vertical) short-dashed red line indicates 2010h2, when the OxyContin reformulation was introduced. Appendix Figure B.19 suggests that for most outcomes, must-access PDMPs had larger impacts in Ohio and Kentucky, compared with the policies in other treated states. The pill mill law, considered a complementary law to must-access policies, may have contributed to the strong impact of

Ohio's must-access PDMP.

However, this also raises a concern that pill mill laws may not be complements to must-access laws but a confounder that drives variation in my mortality outcomes. To address this concern, I test whether pain clinic laws have an independent effect when passed in the absence of a must-access law, following the approach used by Buchmueller and Carey (2018). If pill mill laws alone have little or no impact on my outcomes, they are not likely to drive my results. In Appendix Figure B.20, I test for an independent effect of pain clinic laws using data on the 35 states, that did not implement a must-access PDMP until 2016h2. Among these 35 states, Florida, Mississippi, and Texas implemented pain clinic regulations between 2009h1 and 2011h2. I consider the event time window -12/+6, during which these three states are consistently observed. The distant relative periods outside this event time window are trimmed. For this test, I control for a full set of indicators for pre- and post-periods around the enactment of pill mill laws, the full set of state- and time-varying covariates that I use in the baseline analysis, the ARCOS measure of OxyContin use interacted with the time fixed effects, an indicator for whether the state had a voluntary-access PDMP, and the fixed effects for state and half-year. Appendix Figure B.20 suggests that, in the absence of a must-access law, pill mill laws in these three states had no effect on all the outcomes except oxycodone supply. The negative effects on oxycodone supply observed in Panel E are primarily driven by Florida, which experienced a dramatic change in its oxycodone supply (see Appendix Section B.3); I find no effect of pill mill laws on oxycodone supply if I drop Florida. I conclude that this policy is not likely to affect mortality outcomes when providers are not also required to access the PDMP database. My findings are consistent with Brighthaupt et al. (2019), who find pill mill laws had no effect on prescription opioid, heroin, or synthetic opioid overdose deaths in Ohio and Tennessee. It is possible that a pill mill law alone has no substantial effects, but it may work to strengthen the effects of the must-access PDMP if implemented together.

B.4.4 Accessibility of Heroin

Finally, high accessibility of heroin in Ohio is also likely to contribute to the strong association between must-access PDMP and heroin mortality. The nation's major heroin routes, I-70 and

I-75, pass through Ohio, allowing users easy access to heroin and illegal fentanyl. It is not surprising that a supply-side drug policy has a stronger spillover effects in the area where people can easily find affordable substitutes.

B.5 Additional Heterogeneity Analysis

In Appendix Figure B.19, I investigate the effect of the must-access PDMP within each treated state by plotting the estimates from the baseline specification (equation 3.1) that I obtain by dropping all the treated states except one. The (vertical) dashed gray line indicates the implementation timing of a must-access PDMP (between event times -1 and 0). The (vertical) short-dashed red line indicates 2010h2, when the OxyContin reformulation was introduced. Although the estimates are noisy because the treated sample includes one state only, the results presented in Appendix Figure B.19 allows for a better understanding of the differential effects of must-access policies across states.

B.6 Additional Robustness Analysis

B.6.1 Consequences of Excluding Six Treated States

To estimate the policies' medium-run effects, I focus on the treated states that were consistently observed during an event time window that runs from -15 to +6. As a result, among the 16 states that implemented a must-access PDMP during my sample period, the six states that were not observed at some point during the -15/+6 window are excluded.[6] To address the consequences of excluding these six states, I test how my estimates change when I include more treated states in the analysis sample. Note that I construct the sample so that the treated states are balanced in relative periods, so the event time window decreases as the number of treated states included increases. Appendix Figure B.4 presents the coefficients obtained when I estimate the baseline specification (equation 3.1) on three different samples. The dashed red line presents my baseline estimates, obtained using the sample that includes the ten treated states that are consistently observed during the -15/+6

[6] My sample period is from 2003h1 to 2016h2, and in this period, the 16 states implemented must-access PDMPs from 2007h2 to 2016h2.

event time window. The short-dashed blue line corresponds to the 12 treated states that are consistently observed during the -15/+4 event time window, and the black solid line corresponds to the 15 treated states that are consistently observed during the -15/+2 window.[7]

In Appendix Figure B.4, the lines connecting the estimates from each of these three samples closely track one another for all outcomes. Given that the estimated short-term effects are similar across the three samples, I prefer to use the sample that allows me to look at the longer-term effects, which is my analysis sample. By looking at the longer-term effects, I can better understand how heroin mortality and other mortality outcomes change over time and compare how the longer-run impacts differ from the short-run impacts. In Appendix Table B.1, I also report the summary effect of must-access laws among all the 16 treatment states, which I obtain by replacing the full set of indicators for pre- and post-periods with a single indicator for the entire post-period.

B.6.2 Analysis with the Post-Reformulation Data Period

Appendix Figure B.12 presents the estimates from my baseline specification (equation 3.1) obtained when I drop the pre-reformulation time period and reconstruct the sample so that the treated sample is balanced in related periods. I consider an event time window that runs from -2 to +6, during which the nine treated states were consistently observed. The distant periods outside the -2/+6 are trimmed. As presented in Appendix Figure B.12, I see that estimates are very similar to my baseline estimates. In addition, the consequences of controlling for the reformulation are similar to those observed in my baseline analysis: although not reported, if I drop the controls for the reformulation (the interaction of the measure of pre-reformulation OxyContin use and the time fixed effects) from the regressions, the estimated effects on heroin mortality and illegal opioid mortality become larger, which is consistent with my findings from

[7]Although there are the 16 states that implemented must-access PDMPs until 2016h2, the sample with the -15/+2 window has 15 states. This is because Nevada, which implemented the must-access PDMP for the first time in the nation, only has the nine pre-periods that are observed during my sample period. Although not reported, including Nevada in my analysis sample by limiting the number of pre-periods to nine does not change my main results. I prefer to include more pre-periods by dropping Nevada because I can observe the trends in heroin mortality for a longer period while not affecting the estimates significantly.

the baseline analysis (see Figure 2.4). In summary, Appendix Figure B.12 suggests that my estimates are robust to dropping the pre-reformulation period and that accounting for the reformulation is important, not only for addressing pre-trends but also for obtaining more accurate estimates.

B.6.3 Synthetic Control Analysis

As described in Section 3.5, I find that must-access PDMPs have increased the heroin death rate and that this increase coincided with a sudden decrease in prescription opioid mortality. These findings implicitly assume that any pre-treatment differences between the groups can be explained by my econometric model (equation 3.1). However, a concern that some of the unaccounted for pre-period differences between the two groups may be responsible for my results motivates me to conduct a synthetic control analysis as a robustness analysis. I construct a comparable synthetic control state for each treated state based on pre-period data in such a way that the synthetic control state outcome trends are similar to those of the treated state prior to policy implementation. If the baseline results are comparable to those from the synthetic control analysis, my results are not likely to be driven by unaccounted for pre-treatment differences between the groups.

For each of my ten treated states, I construct a synthetic control state from the 34 control states that never implemented a must-access policy,[8] matching on the value of the outcome variable in each of the 15 pre-treatment periods.[9][10] Each synthetic control state is composed of a weighted average of observations from the subset of the 34 control states. A set of synthetic controls are constructed for each of the following outcomes: heroin mortality, prescription opioid mortality, total opioid-related mortality, and oxycodone doses per capita. Table B.5 shows the makeup of the synthetic states for each outcome.

Using observations from the treated and synthetic control groups, I create a sample so that the treated and synthetic control samples are strongly balanced in relative periods,

[8] Florida is excluded in the analysis (see Appendix Section B.3).

[9] A synthetic control analysis has been more widely conducted for a single treated unit or multiple units with the same treatment timing, but recent papers extend this method for the case of multiple units with differential timing of treatment (e.g., Kleven (2019); Acemoglu et al. (2016)).

[10] I use the Stata command `synth` to construct synthetic controls. See https://fmwww.bc.edu/RePEc/bocode/s/synth.html for a description of the `synth` command.

from -15 to +6. Using this sample, I calculate the outcome gap between each treated state and its synthetic control state for each event time. Appendix Figure B.21 plots how the (unweighted) average of these gaps changes over time. In addition, Appendix Figure B.22 depicts the outcome trends for the treated and synthetic control groups separately. The solid black line reflects how the (unweighted) average outcomes change over time in the treated states, and the dashed red line reflects the trends for the control group.

As shown in Appendix Figures B.21 and B.22, my synthetic control analysis suggests a larger policy effect size, but the results pattern is very similar to that observed in my main analysis (see Figure 2.3). Although I still observe upward pre-trends in prescription opioid mortality and oxycodone consumption,[11] the sudden decreases in these outcomes in the first post-period provide suggestive evidence for the substitution.

B.6.4 Alternative Measures of Pre-Reformulation OxyContin Use

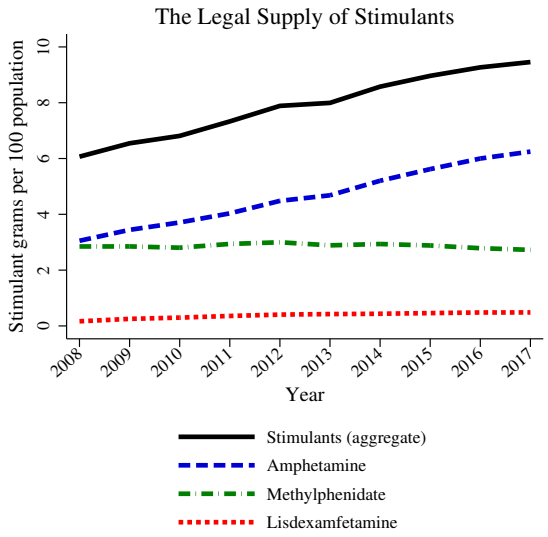
Appendix Figure B.13 displays the coefficients on the indicators for pre- and post-periods from the baseline difference-in-differences specification (equation 3.1) obtained when I use two alternative measures of pre-reformulation OxyContin use (separate regressions): Panel A uses oxycodone / hydrocodone in (morphine equivalent) doses per capita, and Panel B uses the Google Trend measure suggested by Beheshti (2019). My heroin mortality results are robust to using each of these alternative measures.

[11] The reason for this is that a few treated states experienced a sharp increase in prescription opioid mortality and in the legal supply of oxycodone in the pre-period.

Appendix C

Appendix to Chapter 3

Figure C.1: Raw Trends in Stimulant Distribution

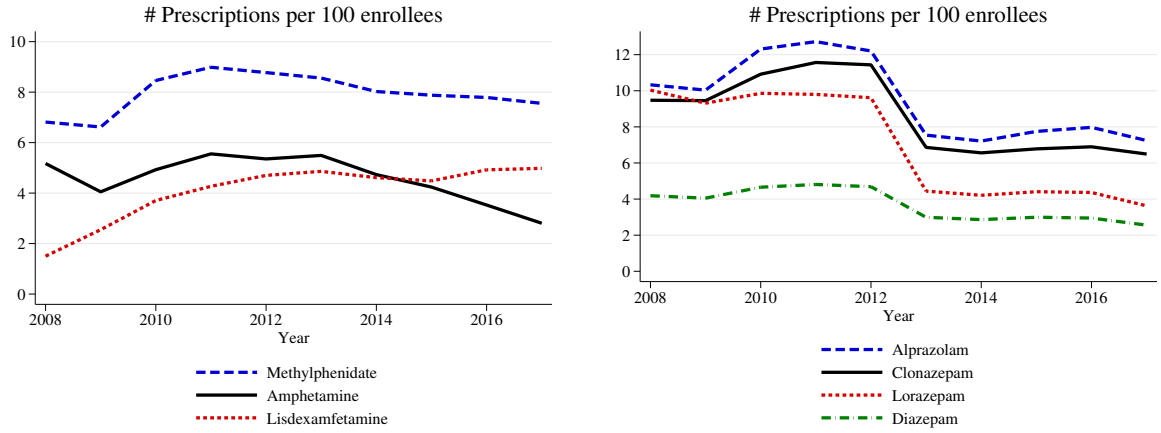


Notes: This figure plots the raw trends in the legal supply of stimulant grams per 100 population separately for all stimulants (black solid line), amphetamine (blue dashed line), methylphenidate (green dash-dot line), and lisdexamfetamine (red short-dashed line). Stimulant grams are adjusted for potency and converted into amphetamine-equivalent grams (see Table C.1).

Figure C.2: Medicaid RX Time Series

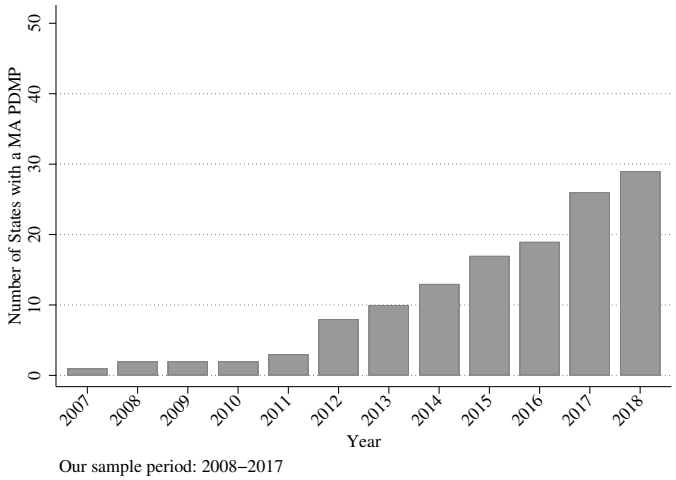
(a) Stimulants

(b) Benzodiazepines



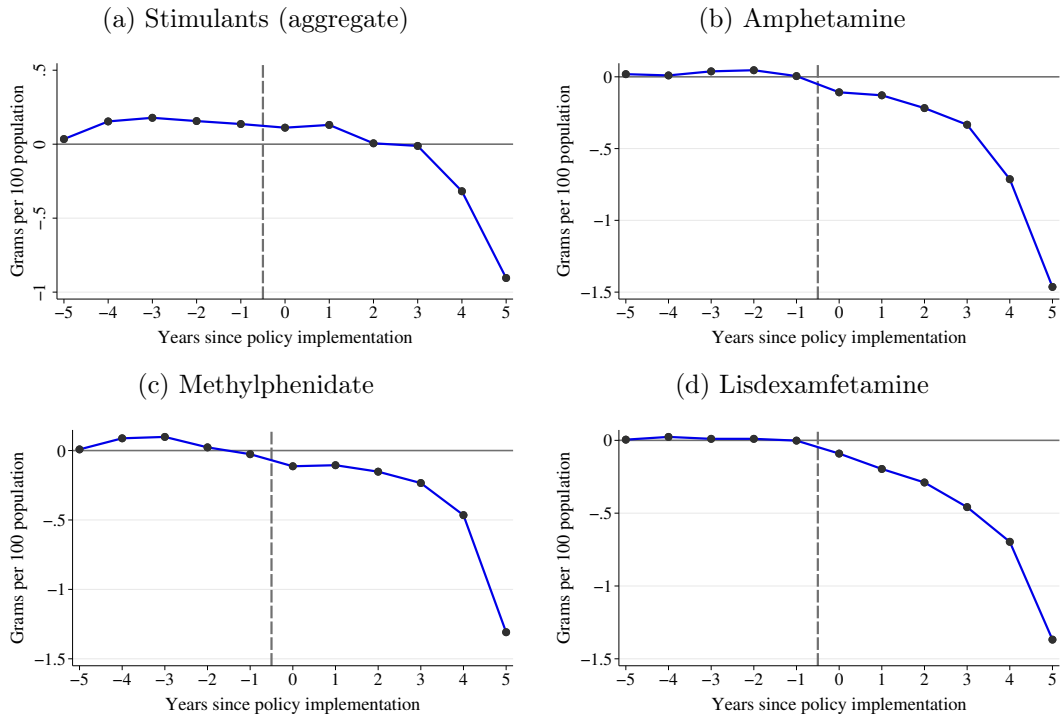
Notes: These figures plot the raw trends in the number of prescriptions per 100 Medicaid enrollees.

Figure C.3: Trends in the Number of States with MA PDMPs



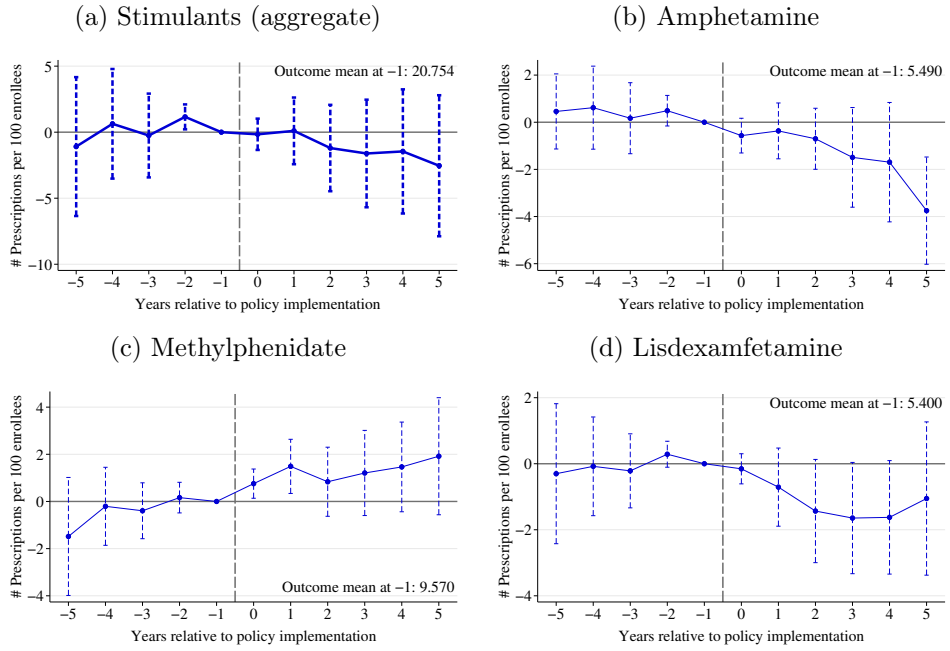
Notes: This figure shows how the number of states with a MA PDMP changes over time. The number of treated states are calculated using the effective dates of MA PDMPs reported in Table 3.1.

Figure C.4: Synthetic Control Analysis, Effects of MA PDMPs on Stimulant Distribution (ARCOS Data)



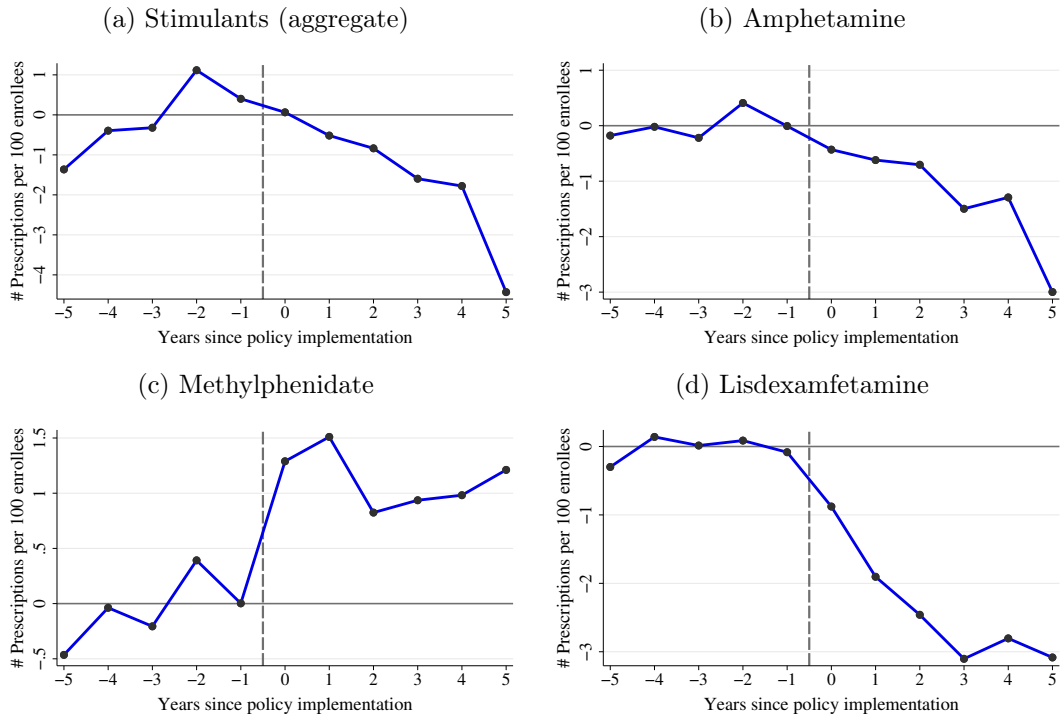
Notes: These figures show how the (weighted) average difference in the outcome between the treated and synthetic control groups changes over time.

Figure C.5: Effects of MA PDMPs on Stimulant Prescribing (Medicaid Data)



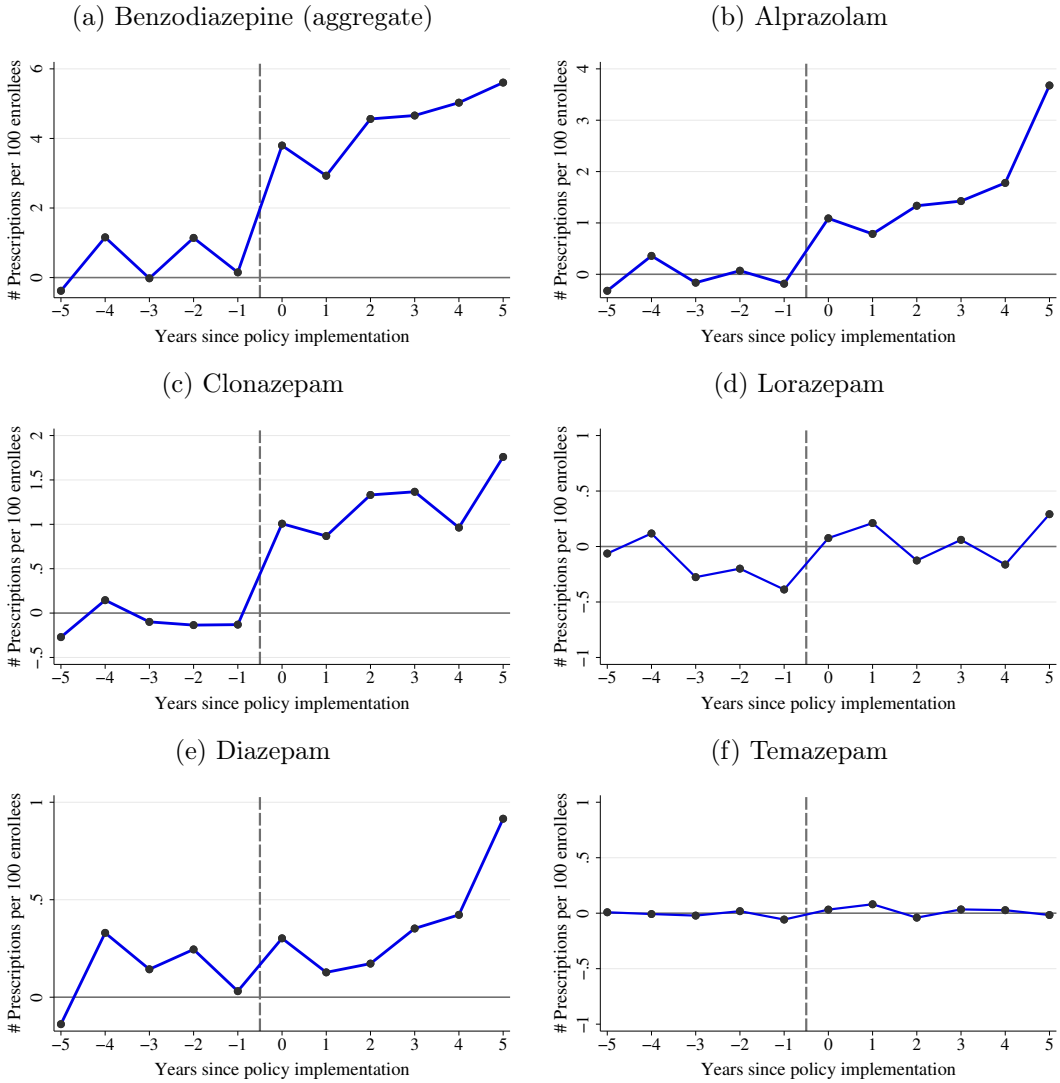
Notes: These figures present the coefficients and 95% confidence intervals on the interactions between the indicator for treated states and the indicators for each of the years before and after policy implementation obtained from estimation of equation (3.1). The year before the policy implementation is the omitted category. The regressions include state and year fixed effects. Standard errors are clustered at the state level.

Figure C.6: Synthetic Control Analysis, Effects of MA PDMPs on Stimulant Prescribing (Medicaid Data)



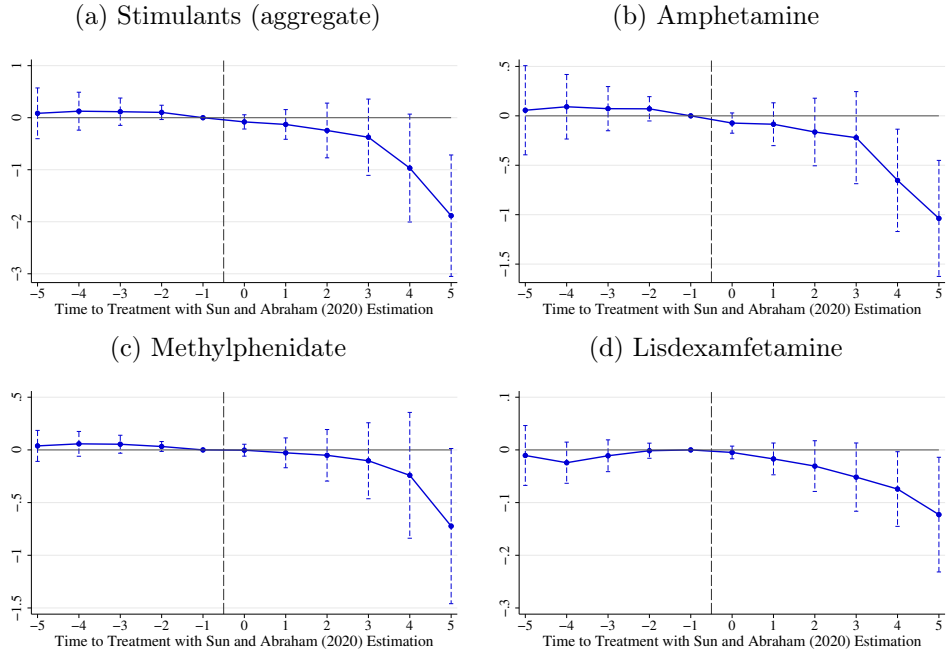
Notes: These figures show how the (weighted) average difference in the outcome between the treated and synthetic control groups changes over time.

Figure C.7: Synthetic Control Analysis, Effects of MA PDMPs on Benzodiazepine Prescribing (Medicaid Data)



Notes: These figures show how the (weighted) average difference in the outcome between the treated and synthetic control groups changes over time.

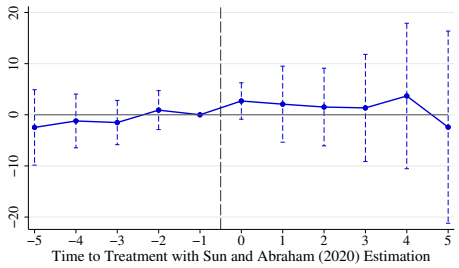
Figure C.8: Effects on Stimulant Distribution (ARCOS Data): Sun and Abraham (2021) Estimates



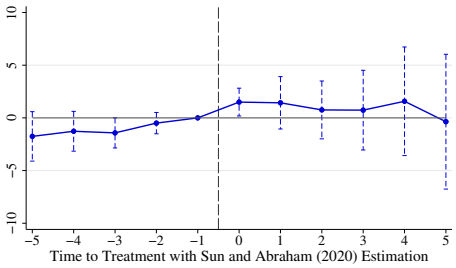
Notes: These figures present the coefficients from Sun and Abraham (2021) estimates for the stimulant distribution outcomes.

Figure C.9: Effects on Benzodiazepine Prescribing: Sun and Abraham (2021) Estimates

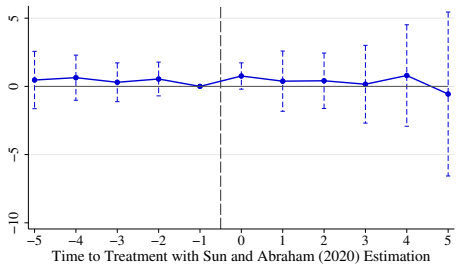
(a) Benzodiazepine (aggregate)



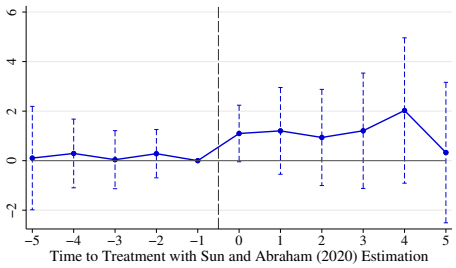
(b) Alprazolam



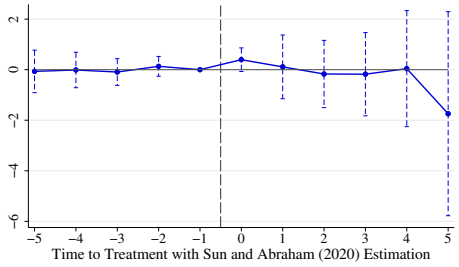
(c) Clonazepam



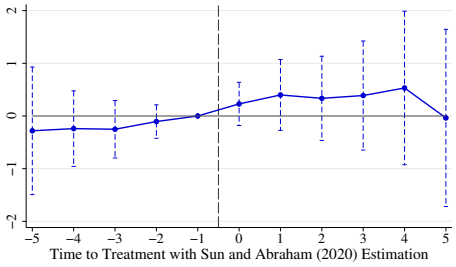
(d) Lorazepam



(e) Diazepam

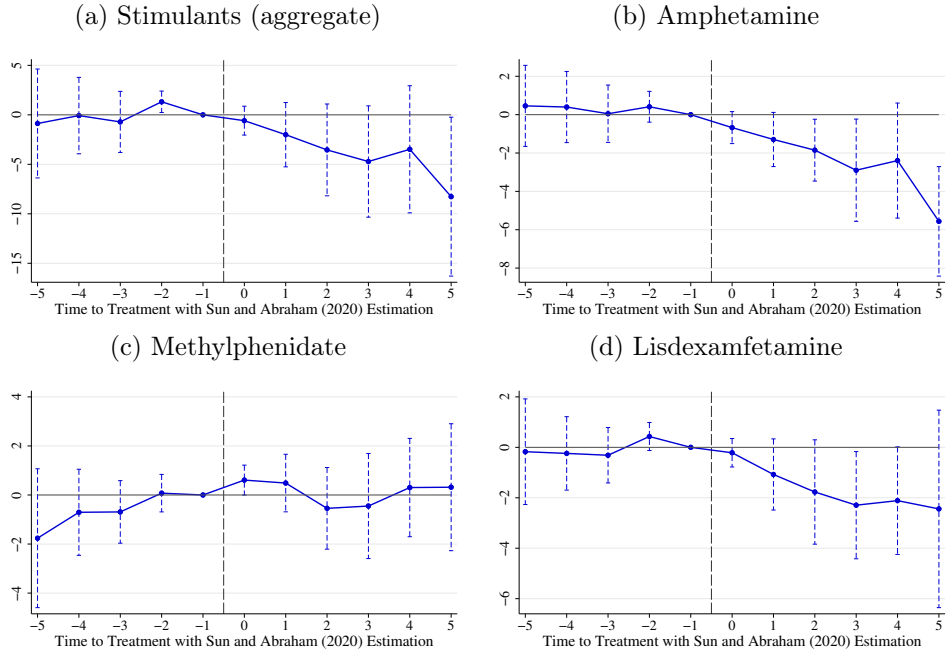


(f) Temazepam



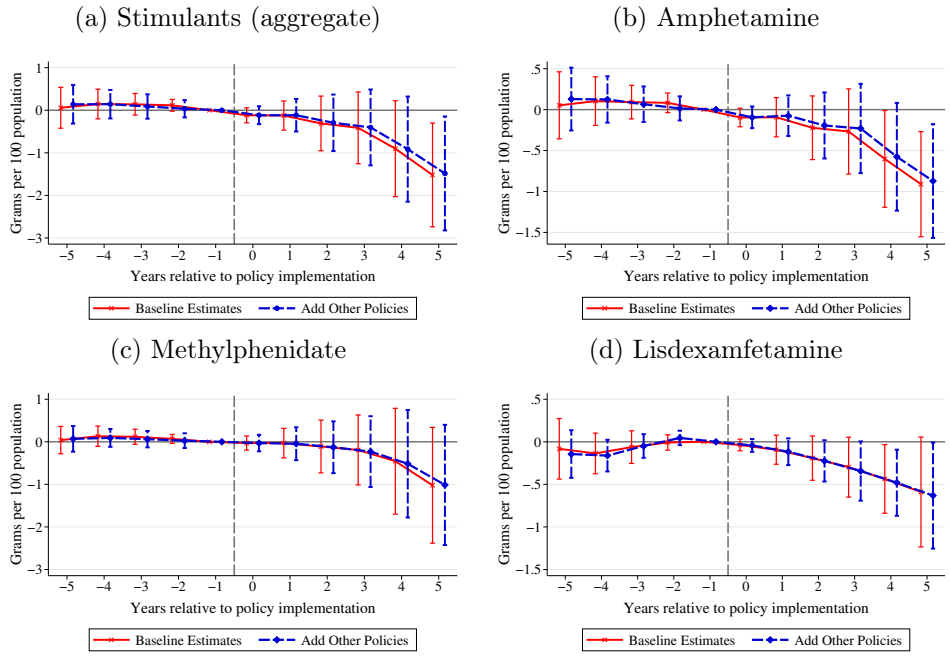
Notes: These figures present the coefficients from Sun and Abraham (2021) estimates for the benzodiazepine prescribing outcomes.

Figure C.10: Effects on Stimulant Prescribing (Medicaid Data): Sun and Abraham (2021) Estimates



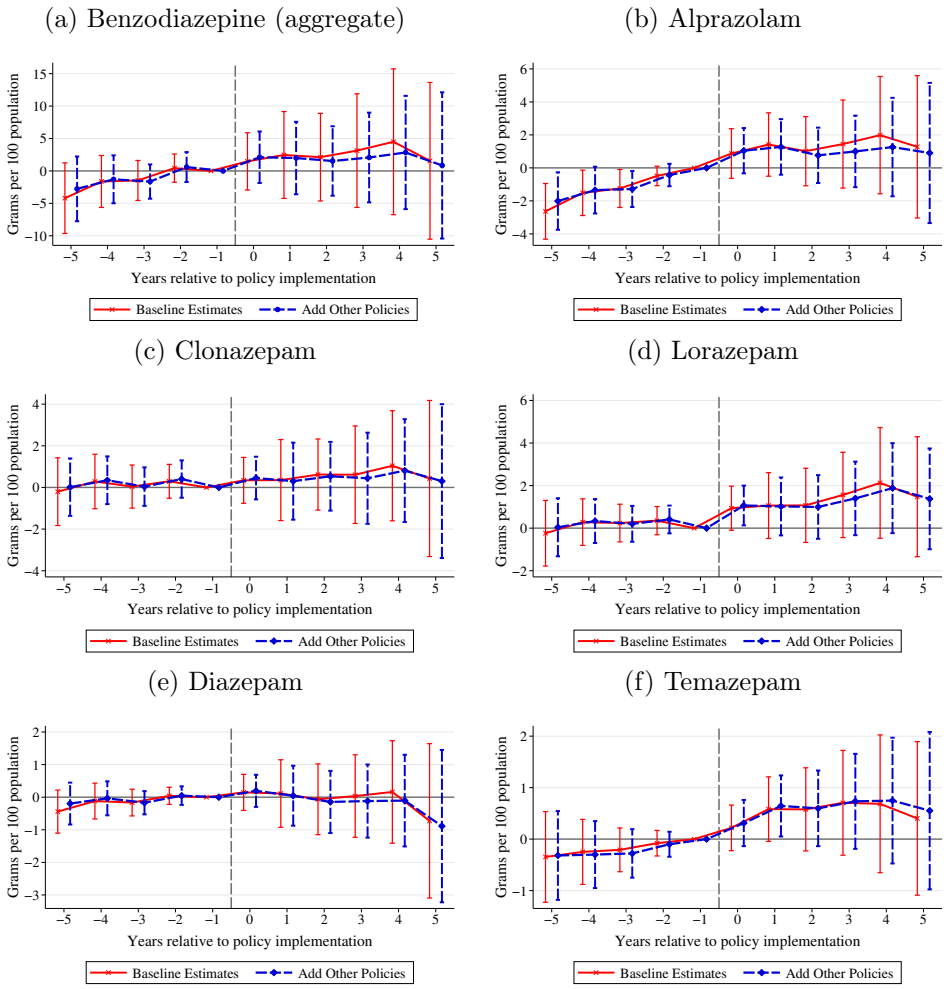
Notes: These figures present the coefficients from Sun and Abraham (2021) estimates for the stimulant prescribing outcomes.

Figure C.11: Robustness of the Stimulant Distribution Results (ARCOS Data)



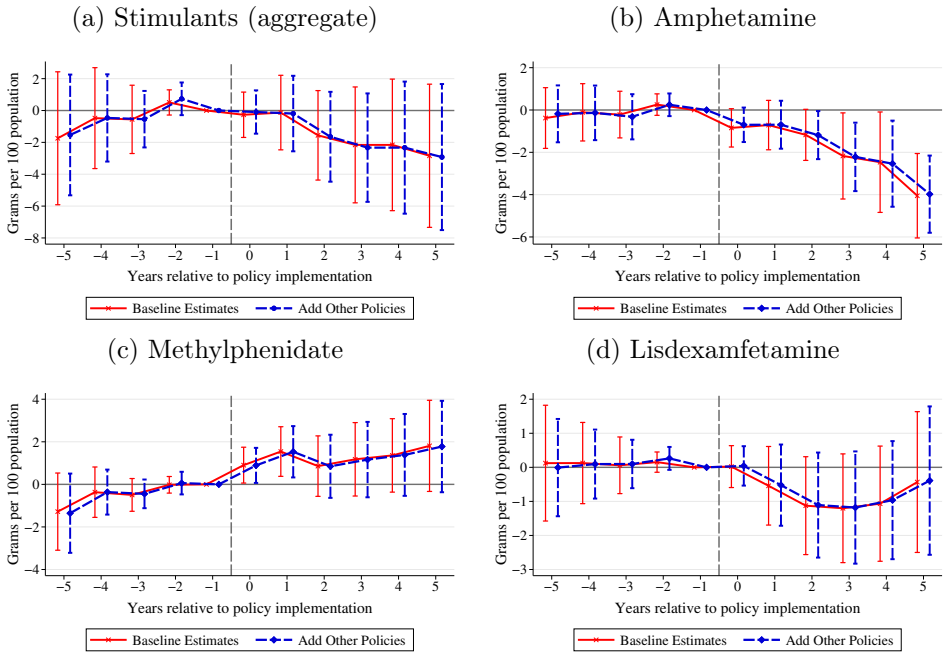
Notes: These figures show the robustness of our results to adding co-occurring opioid-related policies to the baseline model, as described in Section 3.5.1. The dependent variables are amphetamine-equivalent stimulant grams per 100 population. The solid red line indicates the baseline estimates and their 95 percent confidence intervals, and the dashed blue line presents the point estimates and associated 95 percent confidence intervals obtained by adding to the baseline model (i) an indicator for whether the state has a naloxone access law (NAL), (ii) an indicator for having a Good Samaritan overdose prevention law, and (iii) an indicator for a pain management clinic law.

Figure C.12: Robustness of the Benzodiazepine Prescribing Results



Notes: These figures show the robustness of our results to adding co-occurring opioid-related policies to the baseline model, as described in Section 3.5.1. The dependent variables are the number of benzodiazepine prescriptions per 100 Medicaid enrollees. The solid red line indicates the baseline estimates and their 95 percent confidence intervals, and the dashed blue line presents the point estimates and associated 95 percent confidence intervals obtained by adding to the baseline model (i) an indicator for whether the state has a naloxone access law (NAL), (ii) an indicator for having a Good Samaritan overdose prevention law, and (iii) an indicator for a pain management clinic law.

Figure C.13: Robustness of the Stimulant Prescribing Results (Medicaid Data)



Notes: These figures show the robustness of our results to adding co-occurring opioid-related policies to the baseline model, as described in Section 3.5.1. The dependent variables are the number of stimulant prescriptions per 100 Medicaid enrollees. The solid red line indicates the baseline estimates and their 95 percent confidence intervals, and the dashed blue line presents the point estimates and associated 95 percent confidence intervals obtained by adding to the baseline model (i) an indicator for whether the state has a naloxone access law (NAL), (ii) an indicator for having a Good Samaritan overdose prevention law, and (iii) an indicator for a pain management clinic law.

Table C.1: Dose Equivalence for Stimulants

Drug	Milligram
Amphetamine	5
Methylphenidate	10
Lisdexamfetamine	30

Notes: This table lists dose equivalents in milligrams for stimulants, taken from Meinhofer (2018b) and ADHD Medication Calculator (`http://www.adhdmedcalc.com`).

Table C.2: List of Stimulants and Benzodiazepines

Generic Name	Brand Name
Stimulants	
Amphetamine	Adderall
Methylphenidate	Ritalin, Methylin, Metadate, Concerta, Daytrana, Aptensio
Lisdexamfetamine	Vyvanse
Benzodiazepines	
Alprazolam	Xanax, Niravam
Clonazepam	Klonopin
Diazepam	Diastat, Valium
Lorazepam	Ativan
Temazepam	Restoril

Notes: This table lists brand names for each generic type of stimulant and benzodiazepine that are used to construct Medicaid prescribing outcomes.

Table C.3: Synthetic Control Analysis, Effects on Stimulant Distribution

	Aggregate (1)	Amphetamine (2)	Methyl. (3)	Lisdexamf. (4)
Synthetic Control Analysis				
Dependent variable: amphetamine equivalent stimulant grams per 100 population				
Immediate effect	0.112	-0.108	-0.057	-0.015
1-year effect	0.130	-0.129	-0.053	-0.033
2-year effect	0.006	-0.218	-0.076	-0.048
3-year effect	-0.012	-0.334	-0.117	-0.076
4-year effect	-0.318	-0.713	-0.233	-0.116
5-year effect	-0.904	-1.464	-0.654	-0.228
Outcome mean at -1	9.300	5.562	3.274	0.464

Notes: This table shows the immediate effect, 1-year effect, 2-year effect, 3-year effect, 4-year effect, and 5-year effect from our synthetic control analysis (Appendix Figures C.4).

Table C.4: Effects of MA PDMPs on Stimulant Prescribing (Medicaid Data)

	Aggregate (1)	Amphetamine (2)	Methyl. (3)	Lisdexamf. (4)
Dependent variable: Number of prescriptions per 100 enrollees				
Immediate effect	-0.16	-0.57	0.76**	-0.15
	(0.59)	(0.37)	(0.31)	(0.23)
1-year effect	0.10	-0.37	1.49**	-0.71
	(1.25)	(0.59)	(0.57)	(0.59)
2-year effect	-1.20	-0.70	0.84	-1.43*
	(1.62)	(0.64)	(0.73)	(0.78)
3-year effect	-1.61	-1.49	1.21	-1.65*
	(2.02)	(1.05)	(0.90)	(0.84)
4-year effect	-1.46	-1.69	1.46	-1.62*
	(2.33)	(1.26)	(0.94)	(0.85)
5-year effect	-2.54	-3.75***	1.92	-1.05
	(2.65)	(1.13)	(1.23)	(1.15)
State fixed effects	Y	Y	Y	Y
Year fixed effects	Y	Y	Y	Y
Time-varying covariates				
Mean at -1	20.754	5.490	9.570	5.400
Observations	425	441	449	430
R^2	0.846	0.755	0.770	0.831

Notes: This table shows the immediate effect, 1-year effect, 2-year effect, 3-year effect, 4-year effect, and 5-year effect from equation (3.1). Although each regression includes a full set of indicators for pre- and post-periods, we report the coefficients on the post-periods above for brevity. The year before the policy implementation is the omitted category. Observations are weighted by state population. The dependent variables are stimulant prescriptions per 100 Medicaid enrollees. The regressions include state and year fixed effects, as well as time-varying covariates (age and race compositions). The mean of dependent variable is calculated using observations from the treated sample measured in the last pre-policy period. Standard errors clustered at the state level are in parentheses. ***, **, * denotes statistical significance at 1%, 5%, and 10% levels, respectively.

Table C.5: Synthetic Control Analysis, Effects on Stimulant Prescribing (Medicaid Data)

	Aggregate (1)	Amphetamine (2)	Methyl. (3)	Lisdexamf. (4)
Synthetic Control Analysis				
Dependent variable: Number of stimulant prescriptions per 100 enrollees				
Immediate effect	0.064	-0.431	1.289	-0.878
1-year effect	-0.517	-0.620	1.510	-1.904
2-year effect	-0.836	-0.704	0.824	-2.460
3-year effect	-1.596	-1.498	0.936	-3.103
4-year effect	-1.779	-1.293	0.982	-2.804
5-year effect	-4.431	-2.998	1.211	-3.083
Outcome mean at -1	21.460	5.960	9.810	5.560

Notes: This table shows the immediate effect, 1-year effect, 2-year effect, 3-year effect, 4-year effect, and 5-year effect from our synthetic control analysis (Appendix Figure C.6).

Table C.6: Synthetic Control Analysis, Effects on Benzodiazepine Prescribing

	Aggregate (1)	Alprazolam (2)	Clonazepam (3)	Lorazepam (4)	Diazepam (5)	Temazepam (6)
Synthetic Control Analysis						
Dependent variable: Number of benzodiazepine prescriptions per 100 enrollees						
Immediate effect	3.796	1.088	1.007	0.076	0.302	0.032
1-year effect	2.929	0.786	0.868	0.211	0.127	0.080
2-year effect	4.561	1.335	1.331	-0.126	0.172	-0.040
3-year effect	4.659	1.425	1.367	0.060	0.353	0.034
4-year effect	5.028	1.779	0.963	-0.163	0.423	0.027
5-year effect	5.606	3.677	1.759	0.291	0.915	-0.016
Outcome mean at -1	33.912	10.482	9.849	6.122	4.020	1.257

Notes: This table shows the immediate effect, 1-year effect, 2-year effect, 3-year effect, 4-year effect, and 5-year effect from our synthetic control analysis (Appendix Figures C.7).

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Contribution Statement

Chapter 3 of this dissertation is based on the manuscript, "Beyond Opioids: The Effect of Prescription Drug Monitoring Programs on Non-Opioid Drug Prescribing," which is jointly authored with David Beheshti. My contributions included primary conception of the empirical design and performance of primary statistical analysis. David Beheshti contributed to primary conception of the research idea and critical revision of the manuscript. Both co-authors contributed to acquisition and cleaning of data, interpretation of results, and drafting of the manuscript.