THREE ESSAYS IN HEALTH ECONOMICS

AN ABSTRACT

SUBMITTED ON THE FIRST DAY OF MAY 2023

TO THE DEPARTMENT OF ECONOMICS

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS

OF THE SCHOOL OF LIBERAL ARTS

OF TULANE UNIVERSITY

FOR THE DEGREE

OF

DOCTOR OF PHILOSOPHY

BY

(Jin Xing)

APPROVED: Mary Olson

(Mary Olson), Ph.D.
Director

(Stefano Barbieri), Ph.D.

(Kevin Callison), Ph.D.

John H. G. Edwards

(John Edwards), Ph.D.

Abstract

This dissertation contains three chapters on health economics. In the first chapter, I examine how care provision responds to Florida Medicaid's 2017 payment reform, transitioning from a fee-for-service (FFS) to a prospective payment system (PPS) for outpatient services. I find evidence that physicians reduce the use of procedures that are expected ex ante to be more likely to receive no payment under the new system. Additionally, the effects are concentrated on patients without co-morbidities and are observed only in facilities with above-median propensities to treat Medicaid FFS patients. These findings show that physicians respond to the financial incentives in the Medicaid payment reform, particularly when their revenues depend more on Medicaid. In the second chapter, I assess how the Affordable Care Act (ACA)'s Medicaid expansion affected drug overdose mortality rates. I estimate that the expansion increased drug overdose mortality rates by 0.881 per 100,000 people at the county and quarter levels, with over half of this effect attributed to opioids. However, additional analyses show that expanding insurance itself was not directly responsible for the mortality increase but protected against it, as the effect was lower in expansion counties with greater increases in insurance rates. Instead, I find evidence that the expansion fueled the prevalence of illicitly manufactured drugs, mainly explaining the effects. Moreover, contrary to the expectation, there is no evidence that the expansion increases opioid prescribing rates, suggesting that some expansion states restricted prescription opioids. In the final chapter, I investigate the gateway hypothesis, which contends that marijuana use increases people's risk of progressing to use illicit hard drugs (e.g., cocaine, heroin). I find strong evidence of gateway effects among youth in the U.S., that is, marijuana use hastens hard drug initiation. Furthermore, the effects are more pronounced among those who first used marijuana before the age of 18 and those who used marijuana more frequently. These results inform the current debate over the potential of marijuana use during adolescence to further hard drug involvement and highlight the importance of postponing the onset and reducing the frequency of marijuana use.

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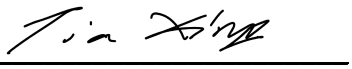
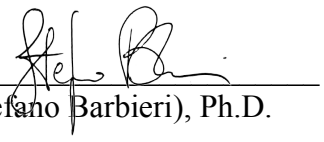


(Jin Xing)

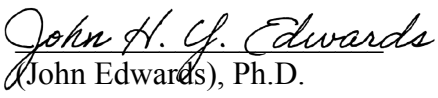
APPROVED: 

(Mary Olson), Ph.D.
Director



(Stefano Barbieri), Ph.D.

(Kevin Callison), Ph.D.



(John Edwards), Ph.D.

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Contents

List of Tables

List of Figures

Chapter 1

Do Financial Incentives Affect Medicaid Care Provision? Evidence from Florida Medicaid's Payment Reform

1.1 Introduction

Slowing health care cost growth has long been a primary objective of U.S. public policy. To this end, there has been a growing interest in transitioning from traditional fee-for-service to alternative payment models (e.g., prospective, capitated, episode-based bundled, pay-for-performance payments). A fee-for-service (FFS) payment system (e.g., actual cost-based reimbursement, fee schedule) reimburses physicians for each additional service provided, rewarding them for performing more unnecessary procedures. Critics blame fee-for-service payments for overprovision, inefficiency, and poor coordination of care, escalating health expenditures without improving health outcomes (e.g., Hackbarth, Reischauer, and Mutti 2008; Arrow et al. 2009; Ginsburg 2011; Ikegami 2015). Unlike fee-for-service, a prospective payment system typically bundles services and pays physicians predetermined amounts, regardless of actual costs for those services performed. As a result, the more care physicians provide under a prospective payment, the lower the profit margins they receive, thereby sharing

financial risk between payers and health care providers (e.g., hospitals, physicians). This may encourage physicians to reduce unnecessary services and lower costs while maintaining or improving the quality of care (Altman 2012). However, prospective payments can result in valuable treatments not being provided, which could worsen health outcomes (Ellis and McGuire 1986). Therefore, how physicians respond to such a transition in the payment system is an important empirical question for determining whether it could be a promising path to promote cost efficiency.

In this study, I exploit a Florida Medicaid's payment reform to investigate this issue in the context of Medicaid. Effective July 1, 2017, Florida Medicaid replaced its reimbursement methods for outpatient services provided in hospitals and ambulatory surgical centers (ASCs). This study focused on ASCs because, unlike hospitals, ASCs are mostly owned by physicians (Badlani 2019). Thus, physicians with ASCs may have a stronger financial motive to respond to payment reforms.[1] Preceding the reform, Florida Medicaid reimbursed each payable ASC service for a medical claim based on a fee schedule. With the reform, Florida Medicaid adopted an outpatient prospective payment system (OPPS) based on the Enhanced Ambulatory Patient Groups (EAPGs).[2] The new payment system discourages providers from providing additional low-intensity procedures for the same medical episode to curb unnecessary low-intensity procedures. As low-intensity procedures are more adversely affected than high-intensity procedures, the reform induces procedure-specific payment shocks that are plausibly exogenous to other determinants of care provisions.

This study contributes to two strands of the health economics literature: the literature on how financial incentives affect procedure choice as well as the literature

[1] Another reason why this study focused on ASCs is due to data availability. Prior to the reform, Florida Medicaid reimbursed hospitals a provider-specific, cost-based reimbursement rate for each payable outpatient service. Payments were then retrospectively adjusted and settled based on cost reports years after the services were provided. Pre-reform payment rates, a key variable in my empirical model, are unavailable for hospitals but available for ASCs.

[2] As of 2017, states (including DC) that have adopted an EAPG-based OPPS for their Medicaid patients include Colorado, Florida, Illinois, Massachusetts, New York, Ohio, Virginia, Washington, and Wisconsin, as well as Washington DC.

on how health care providers respond to a different type of payment system. While there is a large body of research on Medicare and private payers, little is known about these issues for Medicaid. As Medicaid patients typically constitute a minor fraction of a physician's patients, financial incentives in Medicaid may have less impact than those incentives in Medicare or private payers, whose patients usually comprise the majority of physicians' patients. While previous studies on Medicaid (e.g., Gruber, Kim, and Mayzlin 1999; Grant 2009; Alexander 2017) focused on delivery procedures, this study analyzes across-the-board outpatient procedures for Medicaid patients. Moreover, to the best of my knowledge, this study is the first to estimate the effects of payment reform for Medicaid outpatient services.

Exploiting the variation in the procedure-specific payment shocks, I estimated the effects of the reform on care provision. I found evidence that physicians are responsive to financial incentives in this Medicaid setting. Particularly, they reduce the use of procedures that are expected ex ante to be more likely to receive no payment under the new payment system. The results also showed that physician responsiveness was concentrated in the patients without co-morbidities. Since patients with co-morbidities are sicker than those without co-morbidities, this result is consistent with the notion that physicians respond more to financial incentives when treating healthier patients. In addition, the effects were observed only in ASCs with above-median propensities to treat Medicaid FFS patients, implying that health care providers are responsive to financial incentives when their revenues are more dependent on Medicaid.

As increasingly more states are adopting the Affordable Care Act's Medicaid expansion, it is vital to finance and deliver the expanded health care services cost-efficiently. My findings suggest that physicians' financial incentives may play an important role in determining care provision in Medicaid. In particular, bundling services can potentially reduce the provision of clinically wasteful procedures. As

such, payment policies may hold the promise of promoting cost efficiency and maintaining Medicaid expenditures at a sustainable level.

The remainder of this paper is organized as follows. Section 1.2 reviews the relevant literature. Section 1.3 introduces Florida Medicaid's payment reform and derives the procedure-level policy exposure variables. Sections 1.4 and 1.5 describe the data and the identification strategy, respectively. The results are presented in Section 1.6. Section 1.7 discusses the limitations of the study. Finally, Section 1.8 concludes the paper with policy implications.

1.2 Related Literature

This study relates to two strands of literature. First, it relates to the literature on how financial incentives affect the health care supply.[3] Theoretically, this relationship is undetermined a priori. A neoclassical model of physicians as profit-maximizing firms under market demand constraints predicts that the level of service will decrease following a price cut. However, this view fails to consider that physicians may not be constrained by market demand and may induce patients' demand for their asymmetric information advantages over patients regarding their medical conditions and treatments. Thus, physicians may increase the volume of services to recoup the income loss due to a price cut or even sustain a "target income."[4] To incorporate the two polar cases of profit maximization and target income, McGuire and Pauly (1991) proposed a model in which physicians maximize their generalized utility. The utility depends positively on net income and leisure and negatively on demand inducement due to, for example, ethics, threats of malpractice suits, and patient expectations. With this utility function, a lower price would exert downward pressure on physicians' income (the "income effect") and, simultaneously, induces physicians to switch to more ex-

[3]See McGuire (2000) and Chandra, Cutler, and Song (2011) for reviews on this literature.

[4]See Johnson and Rehavi (2016) for evidence on physician-induced demand due to the information gap between physicians and patients.

pensive alternatives (the "substitution effect"). How healthcare supply responds to a lower price depends on the relative sizes of income and substitution effects. When the substitution effect dominates, demand inducement is less profitable. The physician may substitute away from services directly affected, thereby decreasing the level of these services performed, resembling a profit-maximizing firm.[5] Conversely, when the income effect dominates, demand inducement becomes more desirable. Physicians may induce demand by increasing the level of services performed. In an extreme case, when the income effect is all that matters, physicians seek a "target income," entirely undoing the price cut.[6] Empirically, evidence on how physicians respond to financial incentives is mixed. Some studies find positive effects on care provision, i.e., physicians prescribe more procedures when the payment increases or when the payment of an alternative procedure decreases (Gruber, Kim, and Mayzlin 1999; Hadley et al. 2009; Grant 2009; Clemens and Gottlieb 2014; Alexander 2017; Foo, Lee, and Fong 2017). However, consistent with demand inducement, other studies find negative financial incentive effects (Rice 1983; Gruber and Owings 1996; Yip 1998; He and Mellor 2012; Jacobson et al. 2013). Most of these studies focused on the provision of specific procedures, such as C-sections, coronary artery bypass grafting, diagnostic tests, and chemotherapy. Regardless of the mixed evidence, the notion that physicians would increase the volume of care due to price cuts is commonly assumed in policymaking. For example, the Health Care Financing Administration (HCFA) assumes that half of any Medicare payment reduction will be offset by a volume increase (Physician Payment Review Commission 1991).

Second, this study relates to the literature on how providers respond to a different

[5] A profit maximizer only considers the marginal profit, disregarding other factors such as income. Accordingly, their income effect is always zero (Folland, Goodman, and Stano 2016).

[6] Another mechanism for the level of services to increase with a price cut is via a backward-banding supply curve. That is, at a sufficiently high income, the supply of labor bends backwardly to be downward sloping. Along the segment of the backward-banding supply curve, the physician becomes so rich that they spend more time in leisure to enjoy the high income (Folland, Goodman, and Stano 2016). See Hadley et al. (2009) for suggestive support for the existence of a backward-banding supply curve for Medicare services.

payment scheme such as prospective (Cutler 1993; Ellis and McGuire 1996; Dafny 2005), capitated (Dickstein 2011; Ho and Pakes 2014), episode-based bundled (Carroll et al. 2018), and pay-for-performance payments (Darden, McCarthy, and Barrette 2019; Alexander 2020). These studies provide evidence that providers may respond to payment reforms by changing the intensity of services, becoming more likely to admit profitable patients, reallocating patients across facilities, shifting cost burdens to patients untargeted by the payment scheme, and altering coding practices to their favor.

However, although there is a large body of research on Medicare and private payers in these two strands of literature, research on Medicaid is limited. Gruber, Kim, and Mayzlin (1999), Grant (2009), and Alexander (2017) provide Medicaid studies focusing on C-sections. Moreover, while the effects of payment reforms have been widely examined in inpatient and other settings, little is known about their effectiveness for outpatient services, except for He and Mellor (2012), who examined Medicare's transition to an outpatient prospective payment system in 2000.[7]

1.3 Background

1.3.1 Florida Medicaid's Payment Reform

Florida Medicaid's 2017 reform changed its payment method for outpatient services provided to patients with Medicaid FFS.[8] Under the previous payment system, each outpatient procedure provided by ASCs was categorized into one of 14 groups accord-

[7]Other Medicare settings studied in the literature include inpatient acute care (Cutler 1993), inpatient psychiatric care (Norton et al. 2002), inpatient rehabilitation care (Sood, Buntin, and Escarce 2008), skilled nursing facility care (White 2003; Grabowski, Afendulis, and McGuire 2011), and home care (McCall et al. 2003). See Salkever (2000) and Chalkley and Malcomson (2000) for reviews of relevant literature.

[8]Information in this section is drawn from Florida's Agency for Health Care Administration (AHCA) website. For more details, see https://ahca.myflorida.com/medicaid/finance/finance/institutional/hoppps.shtml.

ing to the procedure's Current Procedural Terminology (CPT) code. Each procedure is reimbursed by a scheduled fee for its group. However, the new payment method—an outpatient prospective payment system (OPPS) based on the Enhanced Ambulatory Patient Groupings (EAPGs)—categorizes outpatient procedures that are clinically similar and require similar resource costs into an EAPG group. Compared with the previous grouping, the EAPG grouping is much more granular. There are 564 different EAPGs under version 3.12 of the 3M Enhanced APG System Definitions Manual (3M 2015), the same version used by Florida Medicaid through my study period. Defined by the EAPG Definitions Manual through a list of CPT codes, "significant procedures" are usually the primary reason for a medical episode and require the majority of resources incurred during the episode. With an assigned EAPG group, each significant procedure performed received a payment amount according to the following formula:

$$\text{Payment} = \underbrace{\text{base rate} \times \text{EAPG weight}}_{\text{full payment}} \times (\text{consolidate/discount factor}). \tag{1.1}$$

In the formula, the "base rate" is a common factor for all procedures.[9] "EAPG weight" measures the cost required to perform the procedure relative to the average procedure cost and is constant across procedures in the same EAPG group but varies across EAPG groups.[10] Florida Medicaid did not update EAPG weights through 2018, the end of the study period. By factoring in the EAPG weight, the EAPG-

[9]During the design phase of the new payment system, base rates are calibrated using historical outpatient claim data so that the reform is conducted in a budget-neutral manner separately for hospitals and ASCs, holding physicians' behavior fixed. The base rate was $276.66 in the state fiscal year (SFY) of 2017, which spans between 2017 Q3 and 2018 Q2. It increased to $279.40 in the SFY of 2018.

[10]An EAPG weight is based on statewide providers' average cost of performing any procedure in the EAPG. This ensures that a service receives the same payment regardless of the provider and incentivizes providers to improve efficiency. EAPG weights are calibrated such that the volume-weighted average of all the weights is 1. For example, the EAPG group, "level 1 skin repair," has a weight of 0.5772, which indicates that the resource cost of any procedure in the group is 0.5772 times that of a weighted average procedure.

based OPPS links payments to procedure intensity.

The product of the first two terms, base rate × EAPG weight, is referred to as the "full payment." However, not all services are reimbursed for the full payment. Instead, the EAPG-based OPPS provide sophisticated ways to bundle services to curb unnecessary ones and promote cost efficiency. Specifically, the significant procedure with the highest weight during an episode is designated as the "primary procedure." During the same episode, an additional significant procedure performed is consolidated and receives zero payment if it is the same as or clinically related to the primary procedure; in this case, the "consolidate factor" in the formula is 0. Conversely, if the additional significant procedure is clinically unrelated to the primary procedure, it receives a discounted payment of 50% of its full payment; in this case, the "discount factor" in the formula is 50%. The rationale behind consolidation and discount is that the resource cost required to perform a procedure alongside a related procedure is less than the cost required to perform the procedure itself. Whether any two procedures are deemed clinically related is established by the EAPG developer 3M based on clinical judgment. Nonetheless, both the rule and discount factors can be altered by the Medicaid agency to adjust the financial incentives.[11]

The EAPG payment for the entire episode is the sum of all payment amounts for all service items[12]. Adapted from the EAPG Definitions Manual, Table 1.1 shows an example of applying the EAPG-based OPPS to fictitious episode services.

1.3.2 Expected Payment Shock

This study examines how care provision responds to the financial incentives created by the reform. By switching from a fee-for-service to a prospective payment system,

[11]Florida Medicaid sets the discount factor and adopts 3M's default rule of determining consolidated and discounted procedures.

[12]In contrast, an inpatient diagnosis-related group (DRG) payment method assigns a DRG to an entire claim and reimburses a flat rate based on the DRG code for all services performed during a medical episode.

the reform created procedure-specific payment shocks. Moreover, the reimbursement for a procedure depends on whether the same procedure or a related procedure with a higher intensity is performed for the same episode. That is, the procedure-specific payment shock is context-based rather than fixed. For example, the pre-reform payment for a procedure with a CPT code of 31525 was \$717. In the post-reform period, its payment becomes \$0 when it is consolidated due to being performed alongside a related procedure with a higher intensity (e.g., the procedure with a CPT code of 31545 in Table 1.1). However, its post-reform payment becomes \$196 when it is the primary procedure during the episode and receives full payment.

I construct expected payment shocks to gauge the reform-induced financial incentives as described below. The purpose of doing so is to generate measures of financial incentives using only pre-reform and pre-determined data so that these measures are plausibly exogenous. For procedure i, let P_i^0 and P_i^1 denote the pre-reform payment and post-reform full payment, respectively. Additionally, p_i^c denotes the likelihood of consolidation (i.e., propensity for consolidation), which depends on how and in what context the procedure is used; p_i^d, the likelihood of discount (i.e., propensity for discount); accordingly, the likelihood of receiving full payment is $1 - p_i^c - p_i^d$. Given that the discount and consolidation factors are 1/2 and 0, respectively, the post-reform payment is thus $(1/2) \times P_i^1$ when i is discounted and 0 when i is consolidated. The expected post-reform payment, $\tilde{P}_i^1$, can thus be expressed as a weighted sum of the payments with probabilities as weights:

$$\tilde{P}_i^1 = \left(1 - p_i^c - p_i^d\right) \times P_i^1 + p_i^d \times \left(\frac{1}{2} \cdot P_i^1\right) + p_i^c \times 0. \tag{1.2}$$

I quantify the reform-induced financial incentives using the "expected payment shock" (in percentage), $\Delta\tilde{P}_i$, which can be approximated as the log difference between

the expected post-reform payment and pre-reform payment,

$$\Delta\tilde{P}_i \approx \ln \tilde{P}_i^1 - \ln P_i^0 \approx \left(\ln P_i^1 - \ln P_i^0\right) - p_i^c - \frac{1}{2} \cdot p_i^d. \tag{1.3}$$

In Equation (1.3), the expected payment shock is approximately decomposed into a linear combination of three terms corresponding to three procedure-level policy exposure variables: (1) the log difference between the post-reform full payment and pre-reform payment, referred to as the "full-payment shock," $\Delta \ln P_i = \ln P_i^1 - \ln P_i^0$, (2) the propensity for consolidation, p_i^c, and (3) the propensity for discount, p_i^d. Later, I calculate and use these policy variables for model estimation. Note that they each relate to the three features of the EAPG-based OPPS: full payment, consolidation, and discount.

1.4 Data

The dataset in my analysis (hereafter referred to as the analysis dataset) was constructed from Florida Ambulatory Discharge Data from 2015 to 2018, obtained from the Florida Agency for Health Care Administration (AHCA). The discharge data contained information about the universe of Florida outpatient discharges. Each observation of the discharge data pertains to a patient's discharge and consists of information such as CPT codes for up to 30 procedures performed, the year-quarter of the discharge, the principal payer (e.g., Medicaid, Medicare, private payers), the attending physician's identification number, and the facility's identification number and type (e.g., hospital, ASC). Pre-reform ASC fee schedules and EAPG weights are from the Florida AHCA website.[13]

This study focused on the significant procedures performed for Medicaid FFS

[13]Historical ASC fee schedules can be accessed through https://ahca.myflorida.com/medicaid/review/Historical_Reim.shtml; EAPG weights, https://ahca.myflorida.com/medicaid/finance/finance/institutional/hoppps.shtml.

patients treated with ASCs. As the pricing logic for non-significant procedures differs from that for significant procedures, I provide an analysis of ancillary procedures in the Appendix.

Recall that the full payment, base rate × EAPG weight, is the payment that the procedure receives when it is not consolidated or discounted; for procedure i, the full-payment shock ($\Delta \ln P_i$) is the log difference between the full payment in the first year of the post-reform period and the pre-reform payment. The distribution of full-payment shocks is shown in Figure 1.1. Roughly speaking, the full-payment shock is bell-shaped, centered around zero, and fairly symmetric. For most procedures, the full payment was similar to the pre-reform payment. Procedures with a full-payment shock that falls in the left (or right) tail of the distribution are adversely (or favorably) affected when they become primary procedures.

Moreover, for procedure i, I measured its propensity for consolidation (p_i^c) as the total number of procedures that would be consolidated under the reform divided by the total number of procedures performed in the pre-reform period (2015 Q1–2017 Q2).[14] For example, the procedure with a CPT code of 43239, "Esophagogastroduodenoscopy, flexible, transoral; with biopsy, single or multiple," was performed 7,537 times in the pre-reform period. Of the 7,537, 572 would be consolidated to another related significant procedure under the new system; thus, its propensity for consolidation was calculated as 572/7,537 = 0.076. The propensity for discount (p_i^d) was computed in a similar manner. Figure 1.2 shows the distributions of the propensities for consolidation and discount. Both propensities varied between zero and one. Most procedures have zero or a low propensity for consolidation, indicating that they would never or are not likely to be consolidated under the EAPGs, holding physicians' behavior fixed. Thus, most procedures are not expected ax ante to be significantly influenced by the consolidation under the new system. However, the consolidation

[14] YYYY QX stands for quarter X of year YYYY.

could greatly affect procedures with a high propensity for consolidation. For the propensity for discount, the spike at zero indicates that a vast majority of procedures are not expected ax ante to be discounted under the new system.

As this study exploits the procedure-specific payment shocks induced by the reform, the unit of observation of the analysis dataset is at the procedure and year-quarter level. Table 1.2 reports the summary statistics for the analysis dataset. A procedure is selected if it is present in the pre-reform discharge data, and its pre-reform payment rate is available for calculating procedure-level policy exposure variables. Using this criterion, 965 unique procedures categorized into 101 EAPGs were selected.[15] With 16 year-quarters, the total number of observations was 15,440. On average, 1.45 procedures per 1,000 discharges were performed per procedure and year-quarter combination. The average propensities of consolidation and discount computed using pre-reform data were 0.19 and 0.11. The mean payment for a procedure was $880.90 in the pre-reform period and $962.80 if it received the full payment in the post-reform period. The average number of discharges per quarter is 4,009.19. EAPG weights ranged between 0.43 and 47.02, with a mean of 3.47.

For each procedure performed during the study period between 2015 Q1 and 2018 Q4, I assigned a payment type indicating whether the procedure received full payment, was consolidated, or discounted under the EAPG-based OPPS according to the EAPG Definitions Manual. As pre-reform procedures were not paid via EAPGs, I interpret this assignment as the payment type that would be assigned under the new system. Figure 1.3 shows the average number of significant procedures per discharge during the study period: total and by payment type. The total is equal to the sum of the numbers by the payment type. Immediately after the reform, the average number of significant procedures per discharge decreases discretely, mainly driven

[15]There were 987 unique significant procedures in 112 EAPGs performed for Medicaid recipients in the pre-reform period. Thus, the analysis dataset represents the vast majority of all possible significant procedures.

by consolidated procedures. Meanwhile, the reform does not appear to affect the average numbers for full-payment and discounted procedures, as the level of each series is similar before and after the reform. This suggests that the reform may associate with a decrease in consolidated procedures (but not with the other two types of procedures). Whether this relationship is causal will be taken up in the following sections.

1.5 Method

1.5.1 Baseline Specification

In this section, I empirically examine how care provision responds to reform-induced financial incentives. Here, for procedure i, care provision is measured by the procedure rate, defined as the number of procedures performed per 1,000 discharges. In a previous section, I capture the incentive in three policy exposure variables for procedure i, namely, the full payment shock ($\Delta \ln P_i$), the propensity for consolidation (p_i^c), and the propensity for discount (p_i^d). Here, I relate procedure rate of i at year-quarter t (2015 Q1–2018 Q4) to these policy measures in a baseline fixed effects model, flexibly allowing each variable to affect the procedure rate differently, as follows:

$$\text{Procedure rate}_{it} = \left(\alpha \cdot \Delta \ln P_i + \beta \cdot p_i^c + \gamma \cdot p_i^d\right) \times \text{reform}_t + I_i + T_t + \epsilon_{it}. \quad (1.4)$$

where reform_{it} is an indicator for the timing of the reform, equaling 1 if $t \geq$ 2017 Q3 and 0 otherwise; I_i, procedure fixed effects; T_t, year-quarter fixed effects; ϵ_{it}, the error term.

In Equation (1.4), α, β, and γ are the coefficients of interest. These fixed-effects estimates compare the pre-and post-reform differentials in the procedure rate between procedures more affected by the reform and other procedures. Specifically, α measures

the influence of a full-payment shock on the procedure rate. β measures the pre- and post-reform change in the procedure rate for "always-consolidated" procedures ($p_i^c = 1$) relative to that for "never consolidated" procedures ($p_i^c = 0$). Similarly, γ measures the response of the procedure rate to the reform for an "always-discounted" procedure ($p_i^d = 1$) relative to that for a "never-discounted" procedure ($p_i^d = 0$).

What can be inferred from the signs of α, β, and γ? First, the sign of α is ambiguous in principle, depending on the relative magnitudes of the income and substitution effects. When the income effect exceeds the substitution effect, incentive effects on the procedure rate are negative ($\alpha < 0$). Conversely, when the substitution effect exceeds the income effect, incentive effects on the procedure rate are positive ($\alpha > 0$). $\alpha = 0$ when the full payment shock has neither income nor substitution effects or when income and substitution effects offset each other. Second, because a consolidated procedure incurs costs while receiving no payment, procedures with a higher p_i^c are expected ex ante to be more adversely affected by the reform. Therefore, if the procedure rate of procedures with a higher p_i^c decreases more after the reform (i.e., $\beta < 0$), this would indicate that physicians respond to financial incentives when prescribing procedures. Finally, because discounting is equivalent to a price decrease, the argument for α applies to γ, and thus the sign of γ is ambiguous. That is, the relative sizes of the income and substitution effects determine whether the procedure rate of a procedure with a higher p_i^d decreases or increases more after the reform.

Year-quarter fixed effects, T_t, capture the effects of state-wide trends (e.g., demographics of Medicaid FFS patients) on the procedure rate. Alternatively, I controlled for EAPG/year-quarter fixed effects, $\text{EAPG}_i \times T_t$, which account for variables at the EAPG-quarter level (e.g., demographics of Medicaid FFS patients with similar conditions).

For a given procedure, as the number of observations increases, the computed propensity for consolidation approximates the actual propensity. Therefore, for pro-

cedures that are occasionally performed, the computed propensity for consolidation may be more noisy in measuring the actual propensity. To address this measurement error issue, I constructed a categorical measure of the propensity for consolidation (p_i^c). Specifically, I grouped the procedures into the following three groups. "No propensity for consolidation" (Group 0) consists of procedures that are expected ex ante unlikely to be consolidated ($p_i^c = 0$). "Low propensity for consolidation" (Group 1) consists of procedures moderately likely to be consolidated. "High propensity for consolidation" (Group 2) consists of procedures most likely to be consolidated. Here, a high (or low) propensity for consolidation is defined as being above (or below) the median of p_i^c, 0.33, conditional on $p_i^c > 0$. If consolidation causes the procedure rate to decrease, the effects should be more pronounced in procedures with a greater propensity for consolidation. To examine whether the estimate on p_i^c increases monotonically in magnitude as the level of p_i^c increases, Equation (1.4) can be modified as follows:

$$\begin{aligned}\text{Procedure rate}_{it} = &\left(\alpha \cdot \Delta \ln P_i + \sum_{j=1}^{2} \beta_j \cdot 1(i \in \text{Group } j) + \gamma \cdot p_i^d\right) \times \text{reform}_t \\ &+ I_i + T_t + \epsilon_{it}.\end{aligned} \tag{1.5}$$

where $1(i \in \text{Group } j)$ is an indicator of whether procedure i is in Group j, $j = 0, 1, 2$.[16] I omit Group 0 in Equation (1.5) so that β_j gauges the reform effects on Group j relative to Group 0. If the relationship between consolidation and the procedure rate is causal, then β_1 and β_2 are expected to be negative, with β_2 being greater in magnitude.

[16] $1(\cdot)$ is the indicator function.

1.5.2 Event Study

To check for pre-existing trends that drive the baseline estimate and to examine how the baseline coefficient on p_i^c (β in Equation 1.4) evolves in the post-reform period, I estimate the following leads and lags regression:

$$\text{Procedure rate}_{it} = \left(\alpha \cdot \Delta \ln P_i + \gamma \cdot p_i^d\right) \times \text{reform}_t + \sum_{k=2015\ \text{Q1}}^{2018\ \text{Q4}} \beta_k \cdot p_i^c \cdot 1(t = k) + I_i + T_t + \epsilon_{it}. \tag{1.6}$$

In Equation (1.6), I omit $t =$ 2017 Q2, the quarter immediately preceding reform. Consequently, β_k was estimated relative to that quarter. For the baseline estimate (β in Equation 1.4) to be valid, estimates of β_k's in the pre-reform period should not exhibit a trend that appears to be correlated with the timing of the reform. Given that the baseline estimate is valid, estimates of β_k's in the post-reform period show how the effect of the reform evolves over time.

1.6 Results

1.6.1 Baseline Estimates

Table 1.3 shows coefficients from estimating various specifications. Standard errors are clustered at the EAPG level and reported in parentheses.[17] All specifications control for procedure fixed effects and year-quarter fixed effects except column (5), which controls for EAPG/year-quarter fixed effects instead of year-quarter- fixed effects.

Columns (1)–(3) separately include each policy variable, that is, full-payment shock ($\Delta \ln P_i$), propensity for consolidation (p_i^c), and propensity for discount (p_i^d), as interacted with an indicator for the reform. Column (4) estimates Equation (1.4) of

[17]For each coefficient, the status of whether it is statistically significant at the 5% level preserves when standard errors are clustered at the pre-reform group level.

Section 1.5, including all three policy variables. Comparing columns (1) through (4), column (4) preserves the pattern found in columns (1) to (3), suggesting that these policy variables affect the outcome fairly independently. While the estimates on the full payment shock and propensity for discount are not statistically significant, the estimate on the propensity for consolidation is negative and significantly significant. The negative significant effect on the propensity for consolidation indicates that the procedure rate for procedures expected ex ante to be more likely to be consolidated (and hence receive zero payment) is reduced more due to the reform. Based on the estimates in column (4), the reform reduces "always-consolidated" procedures ($p_i^c = 1$) by 0.722 relative to "never-consolidated" procedures ($p_i^c = 0$), a 49.38% decrease compared to the pre-reform mean of the procedure rate, 1.462. As consolidated procedures incur costs but receive no payment, this finding is consistent with the notion that physicians consider financial incentives when prescribing procedures and points to an important role of payment schemes in physicians' treatment decisions. Contrarily, the null effects on the full-payment shock and propensity for discount suggest that they do not lead to demand inducement or procedure substitution.

Column (5) controls for EAPG/year-quarter fixed effects, instead of year-quarter fixed effects. The estimates in column (5) are qualitatively similar to those in column (4), except for the coefficient on the full-payment shock, which flips the sign from negative to positive but is still statistically insignificant. This suggests that the reform effects are not driven by variables at the EAPG/year-quarter level (e.g., demographics of Medicaid FFS patients with similar conditions).

Column (6) estimates the alternative specification (Equation 1.5 in Section 1.5). Instead of including p_i^c, column (6) includes two dummy variables each for a procedure group (as interacted with the indicator for the reform), "low p_i^c" (Group 1) and "high p_i^c" (Group 2), using procedures with $p_i^c = 0$ (Group 0) as the base group. Table 1.4 shows the number of unique procedures and value or range of p_i^c in each group. While

the coefficient on "low $p_i^c \times$ reform" is -0.0197 and is significantly insignificant, the coefficient on "high $p_i^c \times$ reform" is -0.433 and is significantly significant. Thus, procedures with a higher level of p_i^c were more responsive to the reform and decreased after the reform. This supports the causal negative significant effect on p_i^c in column (4); that is, the consolidation under the new system reduces the procedure rate after the reform. I hereafter refer to column (4) estimates as the "baseline estimates."

An alternative explanation for these findings is that they are due to the underreporting of consolidated procedures. Since consolidated procedures receive no payment under the new system, providers may have less incentive to report these procedures for reimbursement purposes, reducing the consolidated procedures observed in the discharge data after the reform. However, an analysis in the Appendix for ancillary procedures shows that ancillary procedures receiving zero separate payments through "ancillary packaging" do not respond to the reform compared to other ancillary procedures. Under the EAPG, while a significant procedure requires a majority of the time and resources during the medical episode, ancillary procedures are usually proscribed by physicians to assist in diagnosis or treatment. Here, the EAPG refers to ancillary packaging as the combination of the payment of certain ancillary procedures into the payment for a significant procedure. Both packaged ancillary and consolidated significant procedures are not paid separately. However, ancillary packaging differs from consolidation in that a packaged ancillary procedure is paid through an increase in the payment for its associated significant procedure by its expected payment (see the Appendix for a numerical example), whereas a consolidated significant procedure is not paid whatsoever. (The increased payment for the significant procedure remains the same whether or not the ancillary is performed or reported.) As such, providing a packaged ancillary procedure is not expected to affect providers adversely, but providing a consolidated significant procedure is. Therefore, the insignificant result for ancillary procedures indicates that the baseline estimates are not due to underreport-

ing because if providers underreported separately unpaid procedures, the observed procedure rate of packaged ancillary procedures would also decrease. Together, these findings are more in line with the role of financial incentives in care provision than with underreporting.

To visually inspect the relationship between the pre- and post-reform changes in the procedure rate and the propensity for consolidation (p_i^c), I first obtained residuals from regressing the procedure rate on all the independent variables in Equation (1.4) in Section 1.5, except $p_i^c \times$ reform. Here, I refer to these residuals as the "adjusted procedure rate." Then, I calculate the pre- and post-reform changes in the adjusted procedure rate for each procedure. Figure 1.4 shows the median of the change at each value of p_i^c. Procedures with a low propensity for consolidation (i.e., p_i^c close to zero) do not appear responsive to the reform as their changes primarily cluster around zero. However, the reform seems to reduce procedures with a high propensity (i.e., p_i^c close to one), as their changes are mostly below zero. For procedures with propensities in between, the changes generally center around zero. As the fitted regression line with a slope of -337.06 ($p = 0.002$) illustrates, the relationship was negative overall. Figure 1.4 indicates that the baseline estimates mainly stem from the reform's effects on procedures with high propensities for consolidation.

1.6.2 Event Study Estimates

In this section, I conduct an event study to examine whether there is a pre-trend that drives the baseline estimate. Figure 1.5 shows the leads and lags estimates from estimating Equation (1.6) in Section 1.5 for each year-quarter during the study period, with standard errors clustered at the EAPG level. None of the pre-reform period coefficients are statistically different from zero compared to the coefficient in the last quarter before the reform (2017 Q2), which is anchored at zero. There appears to be no pre-existing trend that is correlated with the timing of the reform, suggesting that

the propensity for consolidation (p_i^c) is exogenous, given other covariates. An F-test with the null hypothesis that all pre-reform betas are jointly zero is not statistically significant ($p = 0.1298$). The procedure rate responds to the reform immediately after the reform, as the coefficient in the effective quarter (2017 Q3) drops below zero and is statistically significant. The effects during the post-reform period were maintained at a similar level around the baseline estimate of -0.722. The event study results ensure that the baseline estimate is driven by the reform and not by unobserved factors.

1.6.3 Heterogeneous Effects

In this section, I examine the reform's heterogeneous effects across (1) patient groups with different health statuses, (2) ASC groups with different levels of propensities to treat Medicaid FFS patients, and (3) procedure groups with different payment types.

First, in the terminology of principal-agent literature, physicians act as the "agent" on behalf of their patients (Ellis and McGuire 1986). Physicians' care supply decisions respond less strongly to financial incentives when they weigh more value on patients' health benefits (Clemens and Gottlieb 2014). As sick patients are likely to benefit more from care than healthy patients, physicians may respond less to financial incentives for sick patients than for healthier patients. To examine whether this holds in the case of Florida Medicaid's payment reform, I first classified patients into two groups based on their health status using the Charlson index, which gauges the level of mortality for a patient with co-morbidities (Charlson et al. 1987).[18] Patients with co-morbidities are sicker than those without co-morbidities. While a zero value of the Charlson index indicates no co-morbidities, a higher positive value indicates a higher chance that co-morbidities will result in death.[19] Then, for each patient group, I use the procedure rate for that group as the dependent variable in the base-

[18]The Charlson index calculation uses diagnosis codes in the discharge data and is implemented using the Stata module "CHARLSON."

[19]The vast majority (97%) of the patients in the discharge data during the study period have a zero Charlson index.

line specification (Equation 1.4). Figure 1.6 shows the coefficients of the full-payment shock, propensity for consolidation, and propensity for discount (as interacted with an indicator for the reform) separately for all patients, those with a zero Charlson index and those with a positive Charlson index. We observed that the reform effects are concentrated exclusively on patients with no co-morbidities, whereas the reform does not appear to impact patients with co-morbidities. Thus, consistent with previous literature (e.g., Clemens and Gottlieb 2014), physicians are more responsive to financial incentives when treating healthier patients in this case.

Second, since the reform applies to services for Medicaid FFS patients, we expect that ASCs with higher propensities for treating Medicaid FFS patients will be more affected by the reform because these ASCs are more financially dependent on Medicaid. To examine this, for each ASC, I computed the propensity for treating Medicaid FFS patients as the share of discharges paid by Medicaid FFS. Next, I define the high (or low) propensities for treating Medicaid FFS patients as being above (or below) the median of the propensities among all 317 ASCs in the analysis dataset. Table 1.5 lists the number of ASCs with high (or low) propensities besides the range of propensities in each ASC group. Then, for each ASC group, I use the procedure rate for that group as the dependent variable in the baseline specification (Equation 1.4). Figure 1.7 shows the coefficients for each ASC group, along with the coefficients for all ASCs. For comparison, all estimates were divided by the corresponding group's mean pre-reform procedure rate. Consistent with our expectations, the baseline estimates were exclusively driven by ASCs with higher propensities, whereas the reform had little effect on ASCs with low propensities.

Finally, I examine how the reform impacted procedures of each payment type (i.e., full-payment, consolidated, or discounted procedures). Specifically, for each payment type, I use the procedure rate for that payment type as the dependent variable in the baseline specification (Equation 1.4). Figure 1.8 shows the coefficients

by payment type, which can be interpreted as the reform's effects on either full-payment, consolidated, or discounted procedures. Since the total volume is equal to the sum of the volumes by payment type, the procedure rate is equal to the sum of procedure rates by payment type. As such, for each policy variable on the x-axis, the sum of the three point estimates equals the baseline estimate on the policy variable. None of the coefficients on the full-payment shock and the propensity for discount is statistically significant. For the propensity for consolidation (p_i^c), the coefficients of full-payment procedures and consolidated procedures are -0.186 ($p = 0.099$) and -0.524 ($p = 0.032$), respectively. These two coefficients make up almost the entire baseline estimate on the propensity for consolidation (-0.722). Accordingly, consolidated procedures account for the majority (72.58%), and full-payment procedures account for part (25.76%) of the baseline estimate. On the one hand, since "never-consolidated" ($p_i^c = 0$) procedures are rarely consolidated, the coefficient for consolidated procedures indicates that the reform reduced the use of consolidated procedures, with the reduction more pronounced in procedures expected ex ante to be more likely to be consolidated.[20] On the other hand, the coefficient for full-payment procedures indicates that there is limited evidence that the reform shifted full-payment procedures from low-intensive to high-intensive services (i.e., procedures with high propensities for consolidation to those with low propensities for consolidation).

1.6.4 Spillover Effects

Since ASCs typically receive patients with various payers, a reform targeted at one particular payer may spill over to affect patients with other payers. This "spillover effect" may stem from various avenues. For example, if the reform reduces the net rev-

[20]For "never-consolidated" procedures, the average number of consolidated procedures per discharge is 0 in the pre-reform period by definition and 0.006 in the post-reform period. As a comparison, the average is 0.269 for all procedures in the analysis dataset.

enue for providers, providers may seek to recoup profits from other reform-untargeted payers, for example, by increasing the volume of care if these other payers pay for additional services. Second, the reform may have induced physicians to change their practice patterns for one set of patients. In turn, they may carry that changed practice pattern over to patients with other payers.

To examine whether the Florida Medicaid payment reform has any spillover effect on another reform-untargeted payer, I estimated the baseline specification (Equation 1.4 in Section 1.5) using the procedure rate for that untargeted payer as the dependent variable and the same independent variables. Essentially, I relate the care provision of a reform-untargeted payer to the reform-induced financial incentives for Medicaid. Figure 1.9 depicts the baseline estimates on policy variables (as interacted with an indicator for the reform) separately for Medicaid FFS, private payers, and Medicare FFS. For comparison, procedures common to all three payers (927 in total) were used in the estimations. In addition, all the coefficients are normalized by the corresponding mean of the procedure rate in the pre-reform period. Among all the coefficients for private and Medicare FFS payers, none is statistically significant except for the private payer's coefficient on the propensity for consolidation. This significant coefficient indicates that, for private payers, the level of "always-consolidated" procedures ($p_i^c = 1$) decreases by 6.29% relative to "never-consolidated" procedures ($p_i^c = 0$), which is much smaller in magnitude than its Medicaid FFS counterpart (49.38%).

Thus, there is suggestive evidence of minor spillover effects on patients with private payers. One explanation for this finding is that physicians carry their altered practice patterns from Medicaid patients to private payer patients. However, a caveat of this analysis is that, due to data limitations, certain potential determinants of care provision (e.g., procedure-level reimbursement rates) for these other payers are omitted when estimating the specifications. Consequently, the estimates for the reform-

untargeted payers could be driven by omitted variables and thereby spurious.

1.7 Limitations

Ideally, my specifications should include the prices of substitutes or complement for the procedure as they may influence the provision of that procedure. Nonetheless, substitutes or complements are difficult to define for a given procedure and may depend on the patient's condition. Consequently, a caveat of this analysis is that the estimates could be biased if the prices of substitutes or complements are correlated with policy variables. However, the finding that among all payment types, only consolidated procedures are responsive and reduced more for procedures with higher propensities for consolidation provides us with confidence that the only significant baseline estimate, the estimate on the propensity for consolidation, is unlikely to be driven by substitute or complement prices. Otherwise, other payment types (i.e., full-payment and discounted procedures) could also be influenced similarly.

Moreover, while this study finds that care provision responds to reform, it does not identify the mechanisms behind that response. Figure 1.10 plots the number of Florida Medicaid FFS and managed care enrollees during the study period.[21] Since the number of FFS enrollees changes smoothly during the transition of the payment system, the effects found immediately after the transition (see Figure 1.5) were unlikely to be driven by factors on the demand side (i.e., changes in patients' demographics or preferences) but rather by the supply side. One scenario is that ACSs admit more low-cost patients (e.g., patients whose health conditions do not require additional, possibly consolidated procedures) while denying admissions to high-cost patients. Another scenario is that for a given patient's case mix, physicians change their practice styles by undersupplying certain services in response to prospective pay-

[21] Florida Medicaid enrollment data are obtained from https://ahca.myflorida.com/medicaid/finance/data_analytics/enrollment_report/index.shtml.

ments. Still, the reform effects could stem from a combination of the two scenarios. Since different scenarios have drastically different policy implications, it is important to determine which scenario or whether both are at work.[22]

Finally, policymakers should also be cautious about overshooting payment policies, leading to the underprovision of care. However, as the discharge data do not contain and cannot be linked to quality-of-care measures, this study did not examine the reform effects on access to care, patients' health outcomes, and the quality of care. More comprehensive data are required to examine these topics, and this remains an important direction for future research.

1.8 Conclusion

As the federal and state governments are expanding health insurance access through the Affordable Care Act (ACA) Medicaid expansion and exchange marketplace, financing and delivering expanded services cost-efficiently is crucial to maintain sustainable cost growth. The traditional fee-for-service financing model is generally believed to lead to the overprovision of unnecessary services without improving health outcomes. Therefore, it is important to empirically examine whether an alternative financing model can remedy the shortcomings of fee-for-service.

This study contributes to the understanding of how financial incentives affect care provision for Medicaid outpatient services by exploiting a 2017 Florida Medicaid's payment reform that shifted from a fee-for-service to an EAPG-based prospective payment system as a natural experiment. In the empirical specification, I relate the procedure rate to three policy exposure measures (full-payment shock and propensities for consolidation and discount), as interacted with an indicator of the timing of the

[22]The baseline estimates are essentially unchanged when, in addition to the baseline specification, I control the number of distinct attending physicians who had treated Medicaid FFS patients with a procedure i at quarter t. Thus, the baseline estimates are not driven by the variation in the number of physicians practicing in ASCs.

reform.

I find evidence that EAPG consolidation, which provides no payment for consolidated procedures, has effectively reduced the use of procedures that are expected ex ante to be consolidated more frequently. Since consolidated procedures receive no payment but incur costs, this finding implies that physicians weigh financial costs and benefits when prescribing treatments. However, I did not find evidence that the other two aspects of EAPG have affected care provision. This suggests that either the reform induces no income or substitution effects or that income and substitution effects offset each other. Moreover, the reform effects were concentrated on healthier patients (i.e., patients with no co-morbidities). Conversely, physicians did not seem to change practice patterns for sick patients (i.e., patients with co-morbidities). This is consistent with the notion in previous literature that physicians respond less to financial incentives when they value patients' health gains. Furthermore, the reform effects are driven exclusively by ASCs with above-median propensities for treating Medicaid FFS patients (suggesting that facilities respond more to the incentives when they are more financially dependent on Medicaid) and primarily by consolidated procedures. Finally, there is limited evidence of spillover effects that the reform also affects untargeted patients with private-payer.

The findings imply that certain healthcare providers could respond substantially to financial incentives for Medicaid. Therefore, a Medicaid payment policy has the potential to influence physicians' procedure choices and thereby contain Medicaid expenditure. In particular, providing zero payments for clinically wasteful procedures could curb the use of such procedures. Accordingly, similar Medicaid reforms (e.g., prospective, bundled payments), which have generated a growing interest among states in adoption, may prove effective in promoting cost efficiency.

Appendix

In this appendix, I analyze how the 2017 Florida Medicaid payment reform affected ancillary services with no separate payments. The EAPG payment system classifies services into three types: (1) significant procedures, (2) medical visits, and (3) ancillary services.[23] Under the EAPGs, ancillary services refer to ancillary tests and procedures, which may or may not be performed along with a significant procedure or a medical visit during an outpatient episode. An ancillary service may be "packaged" to a significant procedure or medical visit, which means that the ancillary service is included in the EAPG payment for the significant procedure or medical visit instead of being separately paid. For example, anesthesia may be packaged into a total knee arthroplasty, chest X-ray, or pneumonia visit. Based on clinical grounds, the EAPG developer defines a suggested list of ancillary services that are always packaged when an associated significant procedure or medical visit occurs (hereafter referred to as the EAPG-packaged ancillaries). However, this list can be modified by the payer. Table 1.6 shows an example of applying the EAPG payment system to a fictitious episode's service items, adapted from the EAPG Definitions Manual.

The packaging does not imply that packaged services receive zero payment. Rather, the expected cost of packaged services is included in the payment for its associated significant procedure or medical visits. For example, if a packaged service costs \$10 and is performed on 10% of patients with one of its associated significant procedures, then \$1 (10% of \$10) would be included in the payment for that significant procedure during the design phase. (That significant procedure is reimbursed the same payment regardless of whether the ancillary procedure is performed or reported.) While packaging only applies to inexpensive ancillaries that are routinely performed alongside the significant procedure or medical visit, expansive and rarely-performed

[23] A medical visit refers to an outpatient episode during which the patient receives treatment, with no significant procedures performed.

ancillaries receive separate payments (e.g., the procedure with a CPT code of 84233 in Table 1.6). This is because the packaging of expansive, rarely-performed ancillaries would put providers at financial risk and discourage them from performing these, often valuable, services. For example, a provider would receive only $1 from a packaged test that costs $1,000 but occurs once every 1,000 visits.

A priori, how packaging affects the level of packaged EAPG-packaged ancillaries remains ambiguous. On the one hand, EAPG-packaged ancillaries receive no separate payments but require resources, whereas non-EAPG-packaged ancillaries receive separate payments. This may incentivize physicians to substitute non-EAPG-packaged ancillaries with EAPG-packaged ancillaries, increasing the former relative to the latter. On the other hand, a definite list of EAPG-packaged ancillaries could prevent providers from performing other ancillaries, given that payments for their associated significant procedures or medical visits sufficiently account for packaged ancillaries' costs. Still, the payment reform could have no significant impact on providing packaged ancillaries for the following two reasons. First, since only routine ancillaries can be packaged, this may imply that packaged ancillaries are inelastic to payments. Second, whether or not to provide packaged ancillaries may not significantly impact reimbursements as only inexpensive ancillaries are selected to be packaged.

To examine how the provision of EAPG-packaged ancillaries responds to the reform, I estimate a difference-in-differences (DiD) model that relates the service rate (defined as the number of services per 1,000 discharges) for ancillary service i at year-quarter t in the following form:

$$\text{Service rate}_{it} = \alpha \cdot \text{package}_i \times \text{reform}_t + I_i + T_t + \epsilon_{it}. \tag{1.7}$$

where package_i is an indicator of the EAPG-packaged ancillaries, that is, 1 if i is always packaged under the EAPGs and 0 otherwise. α is the coefficient of interest

and measures the effect of packaging on the service rate. The other notations and variables remain the same as those in the main text. Ideally, covariates should include the full-payment shock interacted with an indicator for the payment reform (see the main text for the definition of the full-payment shock). However, only four ancillaries performed for Medicaid FFS patients during the study period have available pre-reform payment rates. Thus, one limitation of this analysis is that the variable is omitted from the model.

For this analysis, I constructed an analysis dataset with the unit of observation at the service and year-quarter level. Table 1.7 presents the summary statistics for this dataset. An ancillary service was selected if it was performed for Medicaid FFS patients during the study period (2015 Q1–2018 Q4). Using this criterion, 74 unique ancillary services categorized into 26 EAPGs were selected. Among these, 39 were EAPG-packaged and the rest were non-EAPG-packaged. With 16 year-quarters, the total number of observations was 1,186. On average, 0.41 services per 1,000 discharges were performed per procedure and year-quarter combination.

Table 1.8 reports the estimate of Equation 1.7, which is not statistically significant with standard errors clustered at the EAPG level. If anything, the packaging increases the service rate by 0.0249 (or 4.2%, relative to the pre-reform mean of the service rate, 0.5922), which is also economically insignificant. In conclusion, I found no evidence that the reform significantly impacts the provision of the EAPG-packaged ancillaries. This result also suggests that the finding in the main text is not due to underreporting. Both packaged ancillary and consolidated significant procedures are not paid separately. (Packaging differs from consolidation in that, while a packaged ancillary procedure is paid via an increase in the payment for its associated significant procedures, there is no such payment for a consolidated significant procedure.) As such, if healthcare providers altered their coding behavior to underreport procedures that are not separately paid, both packaged ancillary procedures and more consoli-

dated significant procedures would reduce after the reform. However, the insignificant result for packaged ancillaries shows that this is not the case for packaged ancillaries. Therefore, the findings are inconsistent with the notion that healthcare providers underreported procedures that are not separately paid, and accordingly, the finding in the main text is unlikely driven by underreporting.

Tables

Table 1.1: An Application of the EAPG Payment System to an Episode's Service Items

CPT code	EAPG assigned	Payment element	Payment type	Consolidation/Discount factor
31545	063 Level II Endoscopy of Upper Air Way	Significant Procedure	Full Payment	100%
31525	062 Level I Endoscopy of Upper Air Way	Related Procedure	Consolidated	0%
41821	252 Level I Facial and ENT Procedures	Unrelated Procedure	Discounted	50%

Table 1.2: Summary Statistics

	mean	min	max
# of procedures per 1,000 discharges	1.45	0	218.11
Propensity for consolidation	0.19	0	1
Propensity for discount	0.11	0	1
Pre-reform payment	880.90	100	3,000
Post-reform full payment	962.80	119.77	13,137.02
# of discharges	4,009.19	3,091	5,079
EAPG weight	3.47	0.43	47.02
# of unique procedures	965		
# of EAPGs	101		
# of year-quarters	16		
Observations	15,440		

Notes: This table shows the summary statistics of the analysis sample for the study period (2015 Q1–2018 Q4) in terms of the mean, maximum, and minimum. The unit of observation is at the procedure and year-quarter level. The propensities for consolidation and discount are described in the text.

Table 1.3: Effect of Florida Medicaid's Payment Reform on the Procedure Rate

	Procedure rate (# of procedures per 1,000 discharges)					
	(1)	(2)	(3)	(4)	(5)	(6)
Full-payment shock × reform	-0.115			-0.0781	0.0519	-0.0817
	(0.0732)			(0.0690)	(0.118)	(0.0842)
Propensity for consolidation × reform		-0.791***		-0.722***	-0.823**	
		(0.233)		(0.227)	(0.298)	
Propensity for discount × reform			0.630	0.491	0.602	0.529
			(0.366)	(0.372)	(0.458)	(0.376)
Low propensity for consolidation × reform						-0.0197
						(0.251)
High propensity for consolidation × reform						-0.433**
						(0.171)
Baseline mean	1.462	1.462	1.462	1.462	1.462	1.462
EAPG-quarter fixed effects	No	No	No	No	Yes	No
Observations	15,440	15,440	15,440	15,440	15,440	15,440

Notes: This table reports estimates from various specifications in which the procedure rate, i.e., # of procedures per 1,000 discharges, is the dependent variable. The data are a balanced panel with the unit of observation at the procedure/quarter level from 2015 to 2018. "Reform" is an indicator for the timing of Florida Medicaid's payment reform. The "full-payment shock," "propensity for consolidation," and "propensity for discount" are defined in the text, capturing three aspects of the new payment system. With procedures unlikely to be consolidated as the base group, "low propensity for consolidation" refer to procedures that are more modestly likely to be consolidated. "High propensity for consolidation" are procedures most likely to be consolidated. "Baseline mean" presents the pre-reform average procedure rate. All columns include procedure and year-quarter fixed effects, except for column (5), which includes procedure and EAPG/year-quarter fixed effects. Standard errors are clustered at the EAPG level and reported in parentheses. *$p < 0.1$ ** $p < 0.05$ *** $p < 0.01$.

Table 1.4: Groups of Procedures by the Propensity for Consolidation

	# of unique procedures	value/range of the propensity
No propensity for consolidation	542	0
Low propensity for consolidation	225	(0, 0.33]
High propensity for consolidation	198	(0.33, 1]

Table 1.5: ASC Groups by the Propensity for Treating Medicaid FFS Patients

	# of facilities	range of the propensity
ASCs with low propensities	159	(0, 0.0025]
ASCs with high propensities	158	(0.0025, 1]

Table 1.6: An Application of the EAPG Payment System to an Episode's Service Items

CPT Code	EAPG assigned	Payment element	Payment type	Payment discount
31545	063 Level II Endoscopy of Upper Air Way	Significant Procedure	Full Payment	100%
31525	062 Level I Endoscopy of Upper Air Way	Related Procedure	Consolidated	0%
41821	252 Level I Facial and ENT Procedures	Unrelated Procedure	Discounted	50%
88331	390 Level I Pathology	Routine Ancillary	Packaged	0%
82435	402 Basic Chemistry Tests	Routine Ancillary	Packaged	0%
93000	413 Cardiogram	Routine Ancillary	Packaged	0%
322	380 Anesthesia	Routine Ancillary	Packaged	0%
84233	399 Level II Endocrinology Tests	Non Routine Ancillary	Full Payment	100%

Table 1.7: Summary Statistics

	mean	min	max
# of services per 1,000 discharges	0.41	0	24.71
# of unique ancillaries	74		
# of unique EAPG-packaged ancillaries	39		
# of unique non-EAPG-packaged ancillaries	35		
# of EAPGs	26		
# of year-quarters	16		
Observations	1,186		

Notes: This table shows the summary statistics of the analysis dataset for the study period (2015 Q1–2018 Q4) in terms of the mean, maximum, and minimum. The unit of observation is at the ancillary service and year-quarter level.

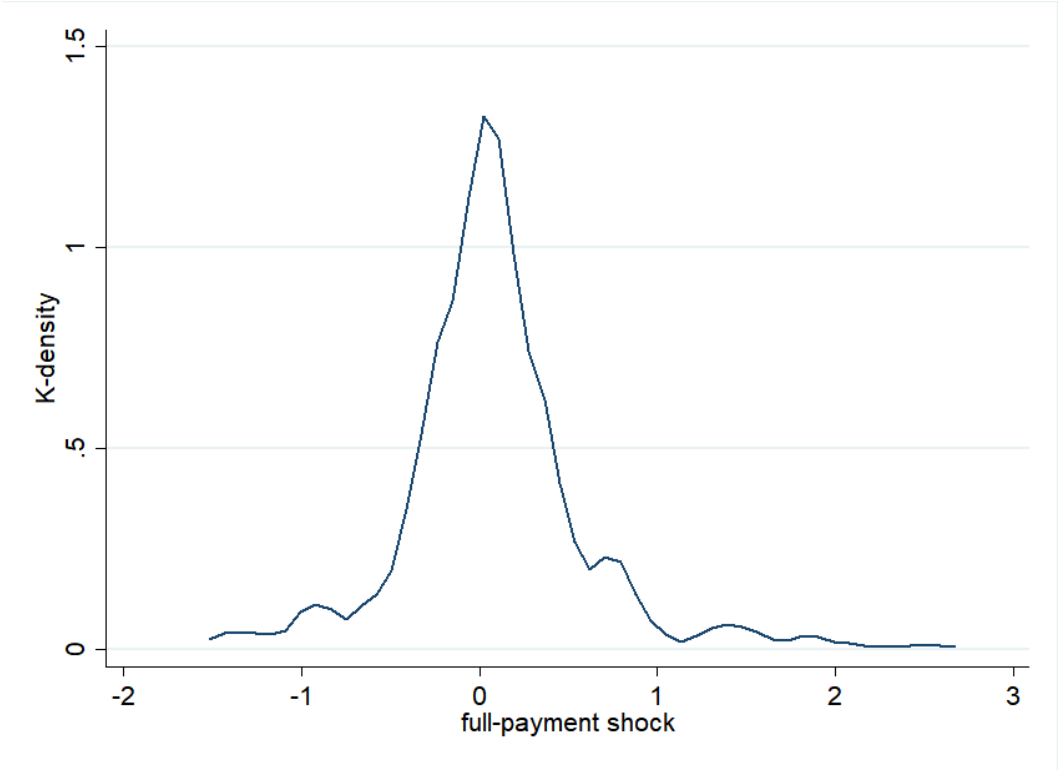
Table 1.8: Effect of the Reform on the Ancillary Service Rate

	Service rate (# of services per 1,000 discharges)
Package × reform	0.0249
	(0.280)
Baseline mean	0.5922

Notes: This table reports estimates from a difference-in-differences regression in which the ancillary service rate (# of services per 1,000 discharges) is the dependent variable. Standard errors are clustered at the EAPG level and are shown in parentheses. Data are balanced panel with the unit of observation at the service and year-quarter level from 2015 Q1 to 2018 Q4. "Package" is an indicator of the EAPG-packaged ancillaries. "Reform" is an indicator for the timing of the 2017 Florida Medicaid's payment reform. The model includes service and year-quarter fixed effects. "Baseline mean" presents the pre-reform average service rate.

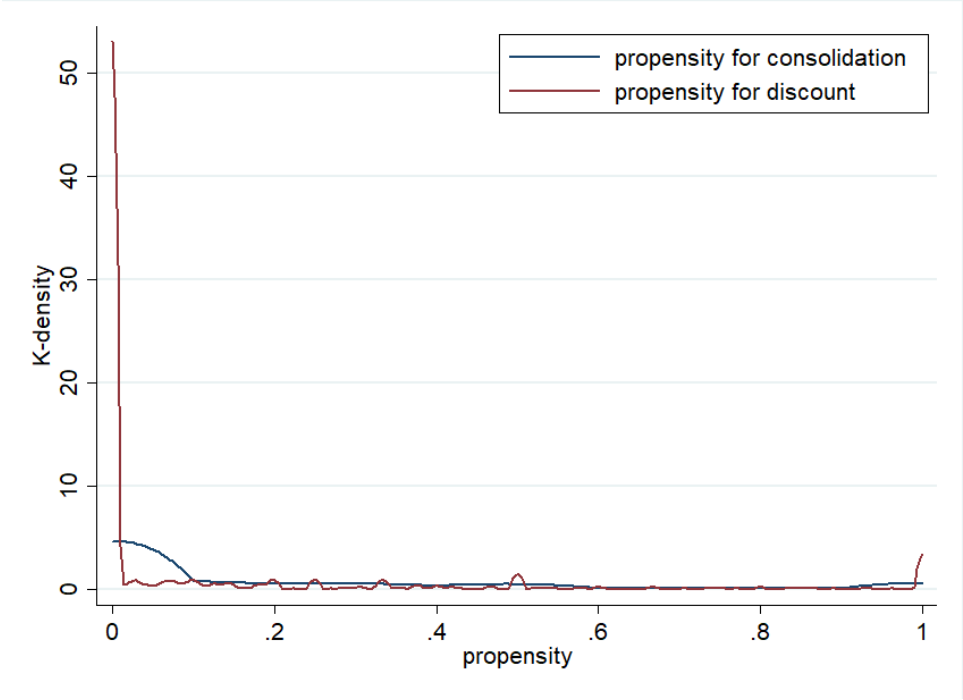
Figures

Figure 1.1: Distribution of the Full-Payment Shock



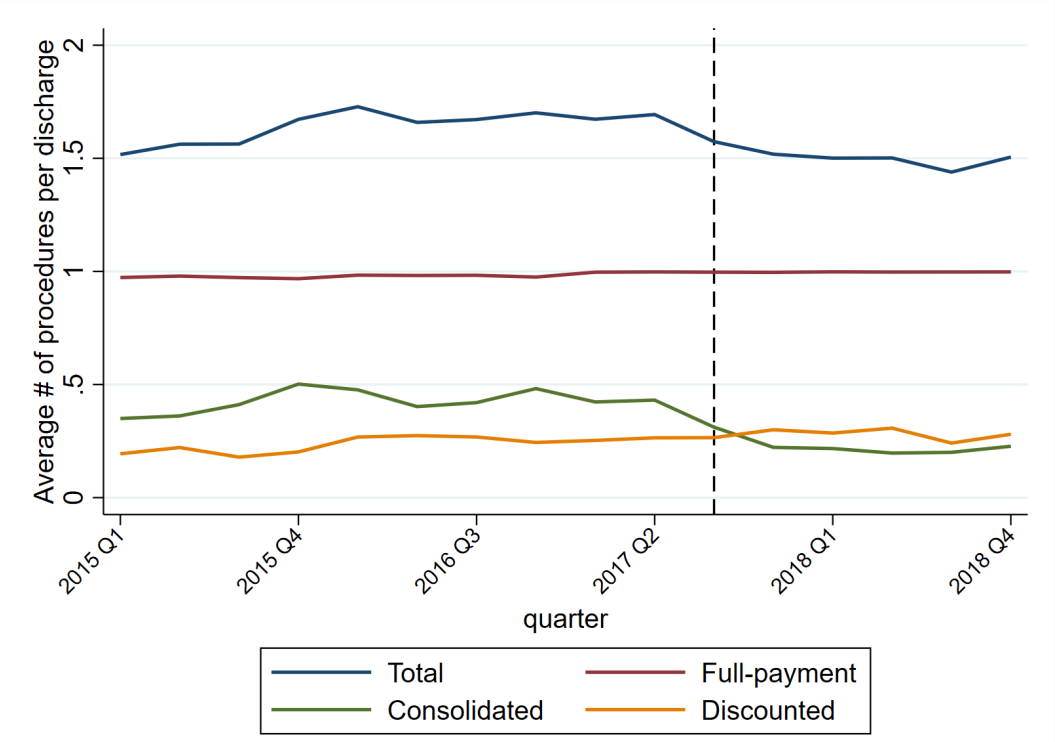
Notes: This figure shows the distribution, estimated by the kernel density (K-density), of the full-payment shock ($\Delta \ln P_i$), which is calculated as the log difference between the full payment in the first year of the post-reform period and the pre-reform payment.

Figure 1.2: Distributions of Propensities for Consolidation and Discount



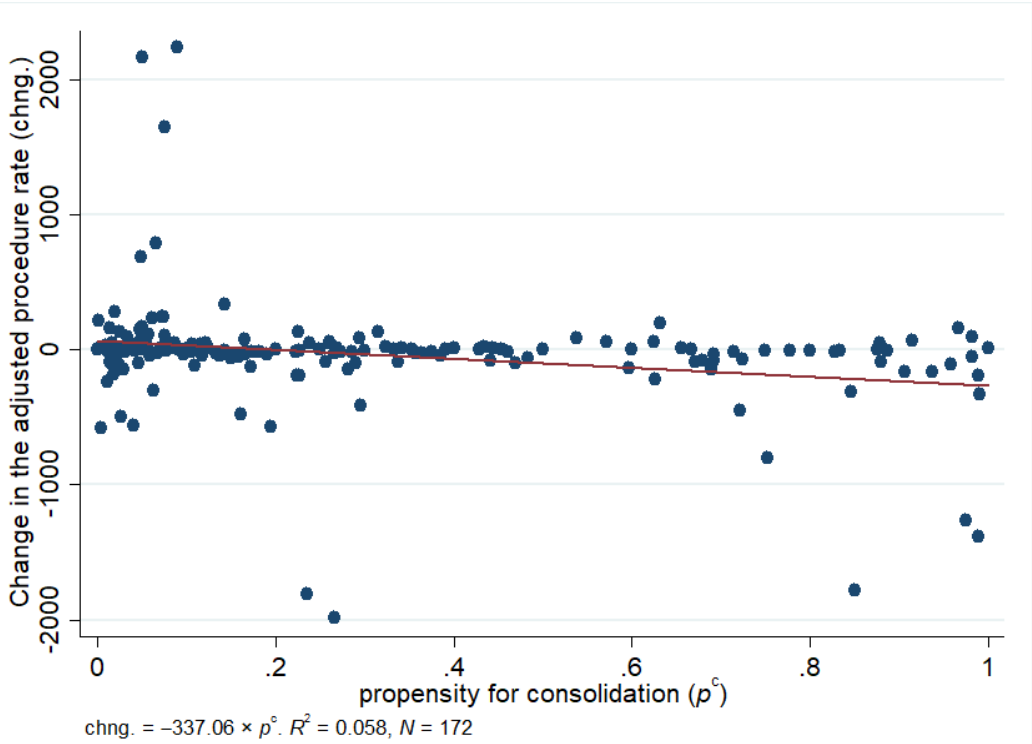
Notes: This figure shows the distributions, estimated by the kernel density (K-density), of the propensities of consolidation and discount. With pre-reform data, the propensity for consolidation of a procedure was computed as the total number of procedures performed that would be consolidated under the new system divided by the total number of procedures performed. The propensity for discount was computed analogously.

Figure 1.3: Average Number of Significant Procedures per Discharge



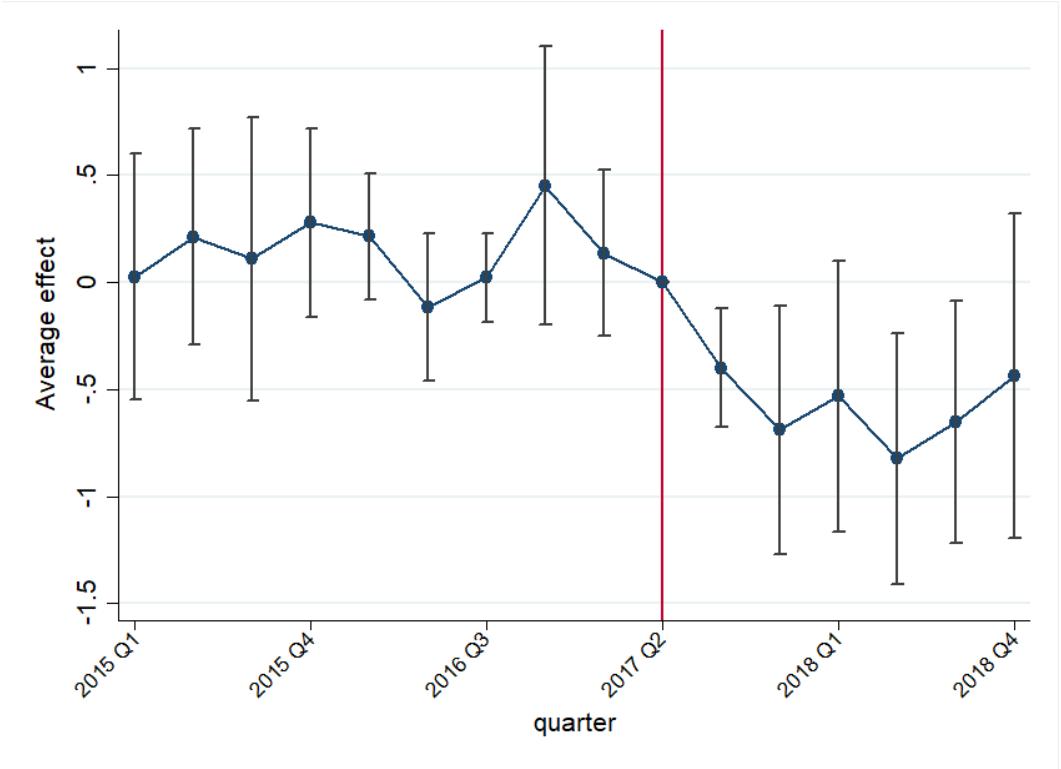
Notes: The vertical dashed line indicates the effective quarter of 2017 Florida Medicaid's payment reform in 2017 Q3. Under the new system based on EAPGs, a full-payment procedure receives the full payment (base rate × EAPG weight), a consolidated procedure receives no payment, and a discounted procedure receives 50% of the full payment.

Figure 1.4: Relationship Between the Pre- And Post-reform Change in the Adjusted Procedure Rate and the Propensity for Consolidation



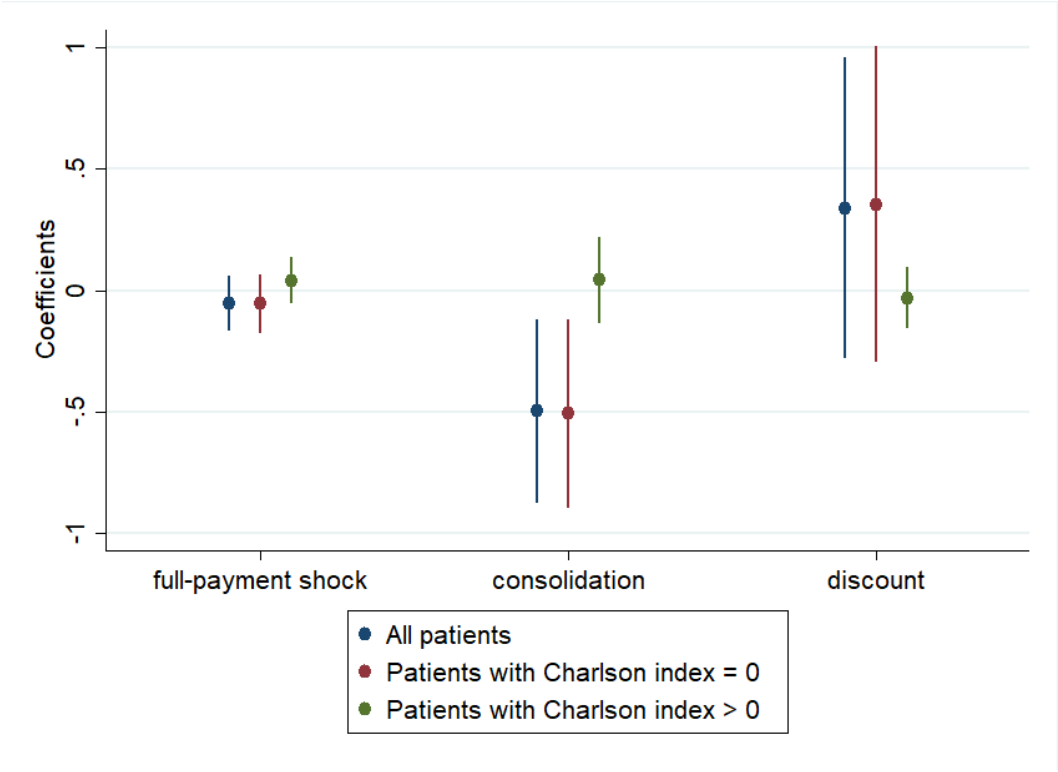
Notes: The above figure plots the median of the pre- and post-reform changes in the adjusted procedure rate at each value of the propensity of consolidation. Here, the adjusted procedure rate refers to the residuals from regressing the procedure rate on the full-payment shock and the propensity of discount, as interacted with an indicator for Florida Medicaid's payment reform, as well as procedure and year-quarter fixed effects.

Figure 1.5: Event Study Estimates



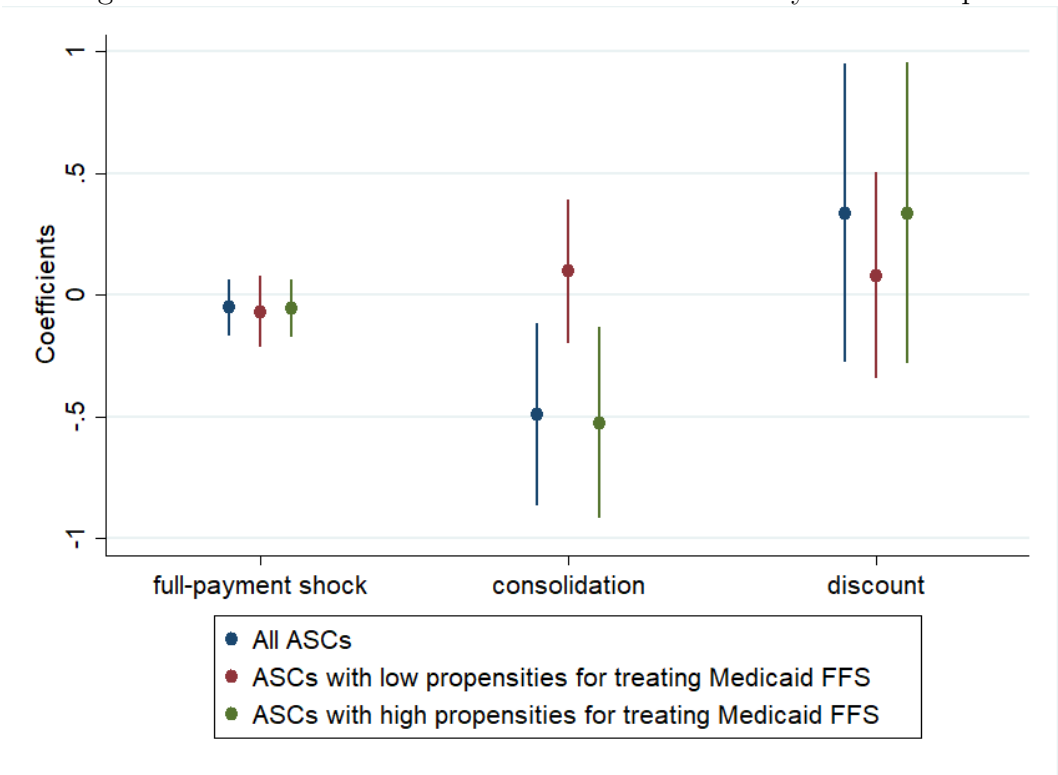
Notes: This figure shows estimates from a lead and lag regression, which regresses the procedure rate on the propensity for consolidation, as interacted with the indicators for each quarter. Dots show point estimates; vertical bars, 95% confidence intervals using standard errors clustered on the EAPG level. The controls include the full-payment shock and the propensity for discount, as interacted with an indicator for Florida Medicaid's payment reform, as well as procedure and year-quarter fixed effects. The solid vertical line indicates the effective quarter of reform.

Figure 1.6: Effect of the Reform on Care Provision by Health Statuses



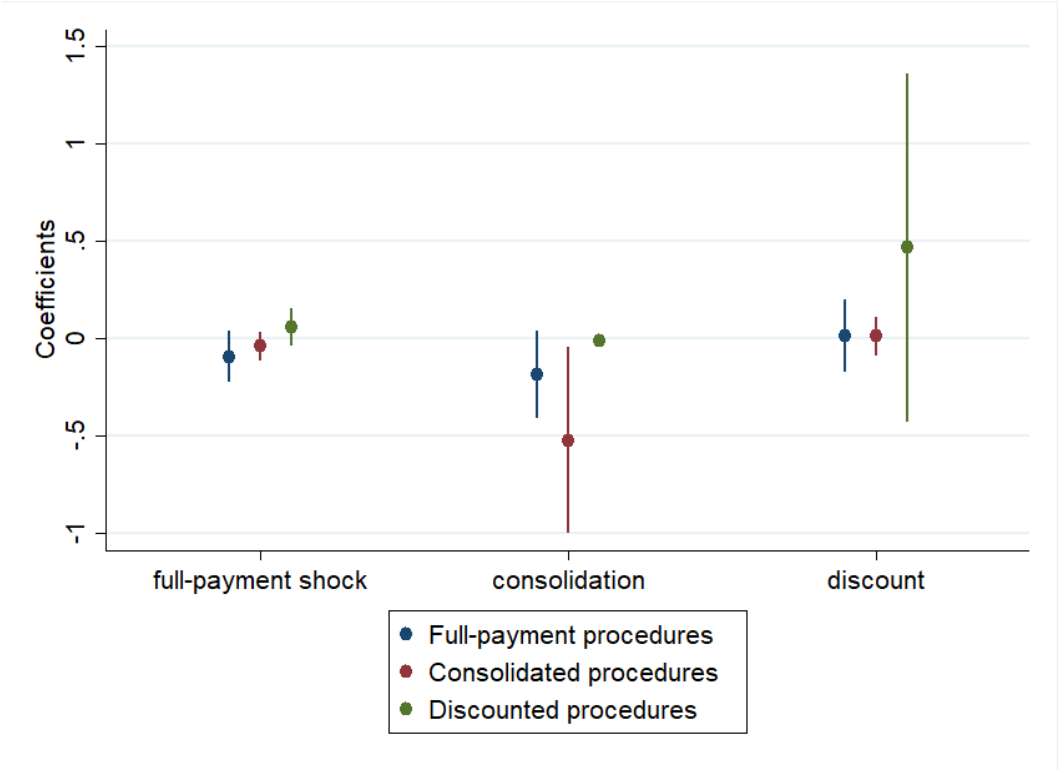
Notes: For each patient group indicated in the legend, this figure shows estimates from regressing the procedure rate for the patient group on the three policy variables. As indicated on the x-axis, the three policy variables consist of full payment and the propensities of consolidation and discount, as interacted with an indicator for Florida Medicaid's payment reform. All the models include procedure and year-quarter fixed effects. Dots show point estimates; vertical bars, 95% confidence intervals with standard errors clustered on the EAPG level.

Figure 1.7: Effect of the Reform on Care Provision by ASC Groups



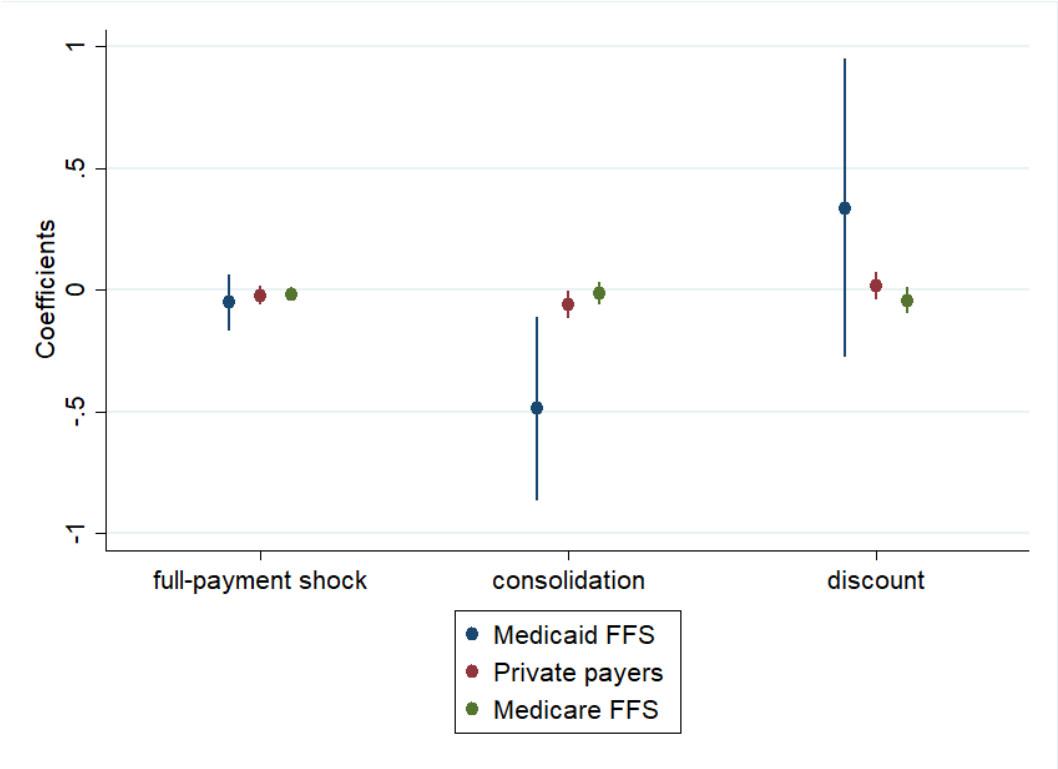
Notes: In the legend, "ASCs with low propensities for treating Medicaid FFS" refers to ASCs with below-median baseline propensity for treating Medicaid FFS patients, and "ASCs with low propensities for treating Medicaid FFS" refers to other ASCs. For each ACS group indicated in the legend, this figure shows estimates from regressing the procedure rate for the ASC group on the three policy variables. As indicated on the x-axis, the three policy variables consist of the full-payment shock and the propensities of consolidation and discount, as interacted with an indicator for Florida Medicaid's payment reform. All the models include procedure and year-quarter fixed effects. Dots show point estimates; vertical bars, 95% confidence intervals with standard errors clustered on the EAPG level.

Figure 1.8: Effect of the Reform on Care Provision by Payment Types



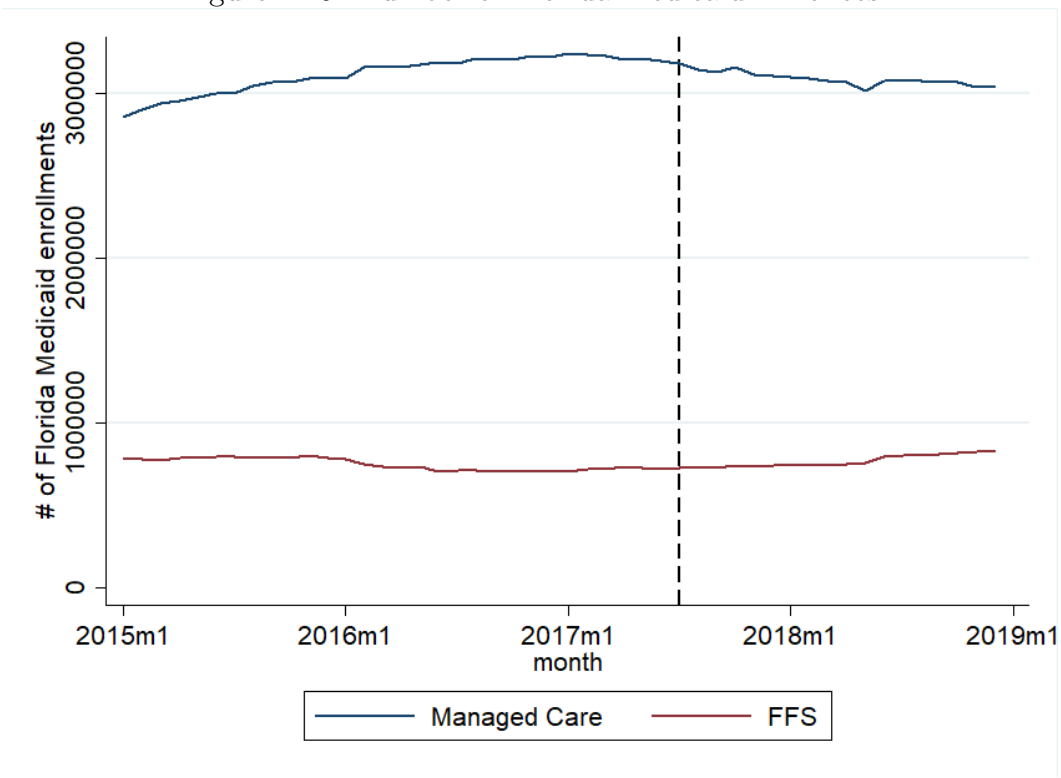
Notes: For each payment type of procedures indicated in the legend, this figure shows estimates from regressing the procedure rate for that payment type on the three policy variables. As indicated on the x-axis, the three policy variables consist of the full-payment shock and the propensities of consolidation and discount, as interacted with an indicator for Florida Medicaid's payment reform. All the models include procedure and year-quarter fixed effects. Dots show point estimates; vertical bars, 95% confidence intervals with standard errors clustered on the EAPG level.

Figure 1.9: Effect of the Reform on Care Provision by Payers



Notes: For each payer indicated in the legend, this figure shows estimates from regressing the procedure rate for that payer on the three policy variables. As indicated on the x-axis, the three policy variables consist of the full-payment shock and the propensities of consolidation and discount, as interacted with an indicator for Florida Medicaid's payment reform. All the models include procedure and year-quarter fixed effects. Dots show point estimates; vertical bars, 95% confidence intervals with standard errors clustered on the EAPG level.

Figure 1.10: Number of Florida Medicaid Enrollees



Notes: YYYYmX stands for month X of year YYYY. The vertical dash line indicates the effective month of Florida Medicaid's payment reform, July 2017.

Chapter 2

Drug Overdose Mortality and Medicaid Expansion under the Affordable Care Act

2.1 Introduction

As one of the largest health insurance programs in the U.S., Medicaid provides free or low-cost health coverage to low-income people, the disabled, children, and pregnant women. Passed in March 2010, the Patient Protection and Affordable Care Act (ACA) Medicaid expansion extended the eligibility for Medicaid coverage to adults with incomes up to 138% of the federal poverty level (FPL).[1] To date, 39 states (including the District of Columbia) have expanded their Medicaid programs (Kaiser Family Foundation 2019). Studies have shown that the expansion significantly increased insurance coverage for the poor. For example, Miller and Wherry (2017) estimated that, in the second year of the expansion, it reduced uninsurance rates by 8.2 percentage points and increased Medicaid coverage by 15.6 percentage points among U.S. citizens aged 19 to 64 years with incomes below 138% of the FPL.

Over the past two decades, deaths from drug overdose, particularly opioid overdose, have increased dramatically in the U.S. The substantial rise in opioid overdose

[1]The expansion was intended to mandate all states to expand their eligibility for Medicaid. However, a 2012 Supreme Court decision effectively ruled that the expansion was optional for states.

deaths is dubbed the "opioid epidemic." In 1999, the unintentional opioid overdose mortality rate in the United States was about two per 100,000 people; by 2017, it had increased to about 13 per 100,000 people (CDC WONDER). A priori, the impact of expansion on drug overdose deaths remains unclear. On the one hand, the expansion may facilitate newly insured beneficiaries to obtain prescription opioids, which could be addictive and lead to increased drug dependence and overdose; namely, prescription opioids could be a "gateway" to addiction.[2] Patients who develop addiction using prescription drugs and can no longer obtain sufficient prescriptions to meet their demand may resort to illicit drugs. This increases the risk of overdose because users cannot easily assess drug safety and quality in the underground markets (Goodman-Bacon and Sandoe 2017; Miron, Sollenberger, and Nicolae 2019).[3] As such, policies intended to curb opioid addiction by restricting access to prescription opioids could inadvertently drive users to switch from prescription drugs to illicit drugs and increase drug overdose deaths. Restrictions on prescription drugs may also lead to pain undertreatment, reduce users' quality of life, and result in more suicides (Kertesz, Gordon, and Satel 2018). Surveys indicated that the regulations discourage physicians from prescribing opioids, potentially leading to undertreatment of pain (Gilson and Joranson 2001). Moreover, facilitating access to prescription opioids may lead to nonmedical use of opioids among individuals without prescriptions. About half of the respondents who misused prescription opioid pain relievers reported obtaining them from a friend or relative (Lipari and Hughes 2017).

On the other hand, the ACA includes substance use disorder (SUD) services as an essential health benefit, requiring Medicaid health insurance provided to newly

[2]Multiple studies found no increase in opioid prescriptions with the expansion (Saloner et al. 2018; Sharp et al. 2018; Cher, Morden, and Meara 2019).

[3]For example, illicit opioids obtained from underground markets do not have warning labels, and thereby users are more likely to combine opioids with alcohol or other drugs, increasing the risk of respiratory depression (Miron, Sollenberger, and Nicolae 2019). Moreover, illicit opioids are produced without adhering to appropriate manufacturing measures, leading their potency to vary considerably and unpredictably (Abouk et al. 2021).

eligible adults to cover SUD services.[4] Medicaid is the only insurance option available to many patients with SUD to obtain affordable treatment (Goodman-Bacon and Sandoe 2017).[5] Health conditions requiring pain relief and demand for drug abuse treatment are more common among Medicaid recipients than among non-recipients, especially those with disabilities and chronic diseases. Olfson et al. (2018) found that the uninsured rate among low-income people with SUD in expansion states decreased more than that in non-expansion states. Therefore, the expansion could reduce drug overdose deaths by increasing access to treatment.[6] Further, the expansion may also help curb drug overdoses by increasing the accessibility to prescription opioids for individuals who are susceptible to illicit drug use.

As drug overdose claims a substantial number of lives every year, and many states have adopted the expansion, the a priori ambiguous effect of the expansion on drug overdose mortality rates warrants empirical investigation. Existing studies examining this issue have reported mixed results (Yan et al. 2021; Abouk et al. 2021; Averett, Smith, and Wang 2019; Maclean and Saloner 2019). However, much of this literature suffers from various identification issues such as a lack of statistical power.

This study investigated how the expansion affects drug overdose mortality rates. It contributes to the literature in two ways. First, using a difference-in-differences (DiD) framework and more granular data than the previous research at the county-quarter level, I find evidence that the expansion was associated with an increase in drug overdose mortality rates. Nearly half of these effects were attributable to opi-

[4]Source: https://obamawhitehouse.archives.gov/ondcp/healthcare.

[5]About 37% of the respondents to the National Survey on Drug Use and Health (2010–2013) cited the lack of health insurance as their main reason for not receiving treatment (Grooms and Ortega 2019).

[6]The current standard care to treat opioid addiction is medication-assisted treatment (MAT), which involves using medications (e.g., methadone, buprenorphine, and naltrexone) along with counseling and other support services (Abouk et al. 2021). All state Medicaid programs cover at least one of these medications. Evidence shows that MATs are effective in reducing illicit drug use, opioid dependence, and drug and opioid-related deaths. Prior studies found evidence that the expansion improved access to SUD treatment (Maclean and Saloner 2019; Andrews et al. 2019; Cher, Morden, and Meara 2019; Clemans-Cope et al. 2019; Sharp et al. 2018; Meinhofer and Witman 2018; Saloner et al. 2018; Wen, Hockenberry, Borders, et al. 2017).

oids. Second, I conducted mediation analyses to identify potential channels through which the effects occurred. I found that the expansion increased insurance rates for individuals with incomes below 138% of the FPL, which in turn helped reduce the mortality rate. This suggests that expanding insurance itself is not responsible for the increase in mortality but protects against mortality. However, there is suggestive evidence that the expansion exacerbated the increase in the prevalence of illicit drugs, thereby increasing the mortality rate. Moreover, despite the expectation that the expansion would increase the demand for prescription opioids, opioid prescribing rates in expansion states did not significantly increase compared to those in non-expansion states after the expansion. Additional analyses showed that opioid prescribing rates in some expansion states even decreased than those in non-expansion states after the expansion, suggesting that these expansion states are restrictive in prescribing opioids. Furthermore, the overall expansion effect on drug overdose mortality was mainly driven by expansion states experiencing post-expansion decreases in opioid prescribing rates. Based on these findings, I hypothesize that restrictions on opioid prescriptions along with the expansion led to more people with unmet demands for prescription opioids to resort to illicit substitutes, which are more dangerous than the legal versions, thus leading to more drug overdose deaths.[7] As such, my results are consistent with growing evidence that policies limiting access to licit opioids increased heroin deaths rates (Kim 2021) and heroin possession and dealers (Mallatt 2020).

In this study, I use a stylized model based on the supply and demand of prescription drugs to illustrate that, in the presence of restrictions, the expansion of insurance eligibility may lead more people to use illicit drugs. In light of this model, I examined the heterogeneous effects among counties. I found that the effects on drug overdose

[7]In Louisiana, Governor John Bel Edwards implemented limitations on the dosage of opioid prescriptions for all people alongside the state's Medicaid expansion on July 1, 2016. Although Medicaid has covered many more prescriptions since the expansion was implemented, the Louisiana Board of Pharmacy reported that the numbers of opioid prescriptions and their doses have dropped by 2% and 3%, respectively (O'Donoghue 2017).

deaths were less pronounced in expansion counties with higher increases in insurance or opioid prescribing rates, and they were more pronounced in expansion counties with more severe drug problems. Furthermore, the interaction of the expansion and pain clinic laws decreased opioid prescribing rates, escalated the prevalence of illicit drugs, and fueled drug overdose mortality. These findings are consistent with the implications of the stylized model, suggesting that policymakers should consider costs and benefits when restricting access to legal opioids.

The remainder of this paper is organized as follows. Section 2.2 reviews the relevant literature. Section 2.3 illustrates the stylized model. Section 2.4 presents the empirical methods. Section 2.5 describes the data used in this study. Section 2.6 discusses the results. Finally, Section 2.7 concludes the paper with a consideration of policy implications.

2.2 Related Literature

Previous literature found the sign of the ACA's Medicaid expansion effect on drug-related mortality to be ambiguous or positive. Several studies noted that drug-related deaths grew more rapidly in expansion states than in non-expansion states before the expansion among all states (Goodman-Bacon and Sandoe 2017; Yan et al. 2021) or only among states east of the Mississippi River (Abouk et al. 2021). These studies claimed that evidence from DiD models was not credible because of the pre-trend in drug-related deaths, which violates the parallel trend assumption of the DiD model. However, my analysis showed that the pre-trend became statistically insignificant after controlling for confounding covariates, particularly other statewide drug-related policies.

Two studies found imprecise estimates (Averett, Smith, and Wang 2019; Borgschulte and Vogler 2020). In the first, Averett, Smith, and Wang (2019) used a DiD approach

with state-year level data and found a positive and insignificant effect of the expansion on opioid deaths. However, state-year level data may be underpowered to detect reasonable effects.[8] In the second study, Borgschulte and Vogler (2020) first used the double-lasso method described by Belloni, Chernozhukov, and Hansen (2014) and Urminsky, Hansen, and Chernozhukov (2016) to select variables to be included in a propensity score model that matches the counties in expansion and non-expansion states. They then used propensity-score weighting DiD models and found no significant overall effects of the expansion on opioid overdose mortality rates. However, they found sizable and significant effects—a 35.63% increase—for people aged 20 to 24 years, whereas they found no significant effects among other age groups. One limitation of their study was that they did not account for other drug-related policies, which could be correlated with the expansion and affect drug overdose deaths. While propensity-score matching attempts to address the differences in observables between the expansion and non-expansion counties, it may introduce bias to the estimate (Daw and Hatfield 2018; King and Nielsen 2019).

In contrast, two other studies found positive and significant effects. Using a DiD approach, Yan et al. (2021) estimated that drug overdose mortality increased by 10.3% in expansion states relative to non-expansion states after the expansion. The authors attributed this result to the opioid epidemic and concluded that it mitigated the life-saving impact of the expansion. However, the authors did not investigate the mechanisms underlying the mortality increase and their relationship with the expansion. Like Borgschulte and Vogler (2020), the authors did not control for other drug-related policies. Abouk et al. (2021) used DiD models to separately examine the association between expansion and drug-related mortality east and west of the Missis-

[8]Using variables at the state-quarter level in this study, I conducted a power analysis in à la Black et al. (2019), who defined the minimum detectable effect (MDE) as the minimum effect detectable at the 95% confidence level for a two-tailed test 80% of the time. I found an insignificant effect below the MDE, indicating a lack of power to detect a significant effect.

sippi River.[9] They found a statistically significant association between the expansion and an increase in drug-related mortality in the east (attributable to synthetic opioids other than methadone and heroin) and found no association in the west. The authors also argued that the estimate in the east was not valid because of the pre-trend, whereas there appeared to be no pre-trend in the west. After controlling for state-specific time trends to address the pre-trend in the east, the estimate for the east became insignificant. However, Meer and West (2016) illustrated that if the treatment affects the outcome gradually over time rather than having an immediate effect on the outcome in a discrete manner, controlling for state-specific time trends will mechanically attenuate the estimated treatment effect toward zero. Moreover, a power analysis à la Black et al. (2019) suggested that data restricted to the west, where the DiD design was valid, were underpowered to detect a reasonable effect.

To address the identification issues in existing studies, such as not accounting for other relevant state policies, non-parallel pre-trends, and lack of power, I use finer data at the county-quarter level and a richer set of covariates. In addition, I conduct mediation analyses to examine the potential mechanisms of the identified positive and significant effects.

2.3 Conceptual Framework

Previous studies suggest that the cause of overdose deaths shifts from legal to illicit drugs when the access to prescription opioids is restricted (Goodman-Bacon and Sandoe 2017). In this section, I used a stylized model based on supply and demand to illustrate how the expansion can exacerbate deaths from illicitly manufactured drugs in the presence of prescription opioid restrictions.

[9]This separation attempted to account for the coinciding rise in the supply of illicitly manufactured fentanyl. According to the authors, black and brown powder heroin was sold primarily west of the Mississippi River, whereas white powder heroin was sold primarily east of the Mississippi River. While illicitly manufactured fentanyl is white and can be easily mixed with eastern white heroin, it is difficult to mix it with western black or brown heroin; therefore, it is used much less in the west.

Figure 2.1 illustrates the supply (S) and demand (D) for prescription opioids, where p and q denote the out-of-pocket price and quantity of prescription opioids, respectively. Suppose that the supply is restricted to be fixed at $\overline{s}$. Initially, the price and quantity demanded of the prescription drugs are p_0 and q_0, respectively. As $q_0 > \overline{s}$, there is a shortage of prescription drugs of $q_0 - \overline{s}$. People with unmet demand may opt to use illicit drugs from underground markets, which are more hazardous than legally prescribed versions because their drug potency is not easily accessible, increasing the risk and incidence of overdose and death.

After the expansion, the price decreased from p_0 to p_1 for newly insured Medicaid recipients and those who could easily obtain prescription drugs from others with Medicaid. As the demand is downward-sloping, the quantity demanded increases from q_0 to q_1. Because the supply is fixed, the shortage increases to $q_1 - \overline{s}$ by $q_1 - q_0$. This increase in shortage pushes more people with unmet demand to use illicit drugs, thus exacerbating overdoses and deaths.

This model makes the following three predictions about the heterogeneous effects of the expansion across counties, given that everything else is equal. First, as health insurance rates increase due to the expansion, more people are being treated for opioid addiction, decreasing the demand for prescription opioids (D shifts left). This lowers the shortage increase, thereby decreasing overdose deaths. Thus, expansion counties with higher increases in insurance rates due to the expansion are expected to have fewer exacerbating effects on overdose deaths. Second, as opioid prescribing rates increase, the supply of prescription opioids increases (S shifts right), lowering the shortage increase. Therefore, expansion counties that saw higher increases in opioid prescribing rates due to the expansion are also predicted to have reduced mortality as there will be less diversion to illicit drugs in these counties. Third, a decrease in the price of prescription opioids creates a greater increase in drug shortage for counties with more severe drug problems (having more drug addicts) because these counties

have a more elastic demand (D is flatter). The intuition is that, as individuals more addicted to drugs spend a larger proportion of their income on drugs, the quantity of drugs they demand is more sensitive to drug price changes (National Research Council 2010). Accordingly, these individuals have a more elastic demand curve. As such, expansion counties with more severe drug problems, proxied by higher drug overdose mortality rates before the expansion, are expected to experience greater effects of exacerbation in overdose deaths. In a later section, I empirically examine these implications by estimating the heterogeneous effects among counties.

Moreover, policies restricting or regulating prescription opioids (e.g., pain clinic laws, prescription drug monitoring programs) may reduce the supply of prescription opioids (S shifts left). In expansion states, implementing such opioid-restricting policies may further widen the shortage of prescription drugs, inducing more substitution to illicit drugs. Therefore, the stylized model also implies that the interaction of the expansion and opioid-restricting policies may lead to even more overdose deaths than without opioid-restricting policies. I defer an investigation of policy interaction effects to a later section.

2.4 Empirical Strategy

2.4.1 Baseline Specification

I exploit the variation in the timing of states' adoption of the Medicaid expansion using a DiD framework to identify the causal effect of the expansion on an outcome variable. The baseline estimation equation is constructed as follows:

$$\mathrm{Y}_{cst} = \alpha \cdot \mathrm{Exp}_{st} + X'_{cst}\beta + C_c + T_t + \epsilon_{cst}. \tag{2.1}$$

where Y_{cst} is a dependent variable (e.g., the number of drug overdose deaths per 100,000 people) in county c, state s, and quarter t (from 2010Q1 to 2018Q4). A dummy variable for the expansion status, Exp_{st}, is 1 if state s implemented the expansion in quarter t, and 0 otherwise. X_{cst} is a vector comprising the following control variables: (1) dummies for other statewide drug-related policies that could correlate with the expansion dummy and may impact the dependent variable (constructed in the same manner as the expansion dummy), including the prescription drug monitoring program, pain clinic law, naloxone access law, "Good Samaritan" law, medical marijuana law, and recreational marijuana law; (2) county-level and time-varying demographics, including percentages by gender, race, origin, and age group; and (3) economic indicators, including unemployment rates, poverty rates, and median household income.[10] C_c and T_t are county and quarter fixed effects, which control for county and year-quarter fixed heterogeneity, respectively. ϵ_{cst} is an idiosyncratic error term. α captures the causal impact of the expansion on the dependent variable. Finally, standard errors are clustered at the state level to allow for arbitrary autocorrelation of the errors in each state.

2.4.2 Event Study

A key assumption of the DiD model—the parallel trends assumption—holds that the dependent variable in expansion states would trend in a way similar to that in non-expansion states in the absence of expansion (after controlling for covariates). Otherwise, the DiD estimates are driven by unobserved trends and are thereby invalid.

To examine the pre-expansion differential trend in drug overdose mortality be-

[10]Prescription drug monitoring programs involve statewide electronic databases that track prescriptions of controlled substances. Pain clinic laws impose regulations on pain clinics to restrict prescriptions of controlled substances (including opioids) without medical indication. Naloxone access laws allow lay responders to administer naloxone, an opioid antagonist. "Good Samaritan" laws protect people from prosecution for possessing controlled substances in the event of a drug overdose. Medical marijuana laws allow for marijuana use to treat certain medical conditions. Recreational marijuana laws legalize marijuana use for recreational purposes.

tween the expansion and non-expansion states, as well as the evolution of the treatment effect in the post-expansion period, I perform event studies by running a leads-and-lags regression as follows:

$$\mathrm{Y}_{cst} = \sum_{r=-16}^{19} 1[r(s) = r] \cdot \alpha_r + X'_{cst}\beta + C_c + T_t + \epsilon_{cst}. \tag{2.2}$$

where, for expansion states, $r(s)$ is a function that returns the quarter relative to state s' expansion quarter in quarter t; for non-expansion states, $r(s) = -1$. In the estimation, the indicator for the quarter preceding the expansion quarter, $1[r(s) = -1]$, is omitted from the model. Conditional on other variables, α_r is the difference in the dependent variable in relative quarter r between the expansion and non-expansion states relative to the difference in the quarter preceding the expansion quarter. The other variables are defined in Equation (2.1).

2.4.3 Mediation Analysis

Mediation analysis is used to estimate the role of pathways or mechanisms by which a treatment variable (e.g., policy) affects an outcome; it explains why a relationship exists between two variables (Hicks and Tingley 2011). Here, I further investigated how the expansion may affect drug overdose mortality rates by performing mediation analyses.

To illustrate, let T and Y denote the treatment and outcome, respectively. Let M denote a potential mechanism (called mediator) that transmits the effect of T on Y. Following the steps suggested by Baron and Kenny (1986), the mediation analysis comprises three regressions as follows:

$$M = b_0 + b_1 \cdot T + \upsilon, \tag{2.3}$$

$$Y = \phi_0 + \phi_1 \cdot T + e, \tag{2.4}$$

$$Y = \theta_0 + \theta_1 \cdot T + \theta_2 \cdot \ M + u. \tag{2.5}$$

where v, e, and u are the error terms. In Equation (2.3), which relates mediator M with treatment T, b_1 needs to be significant for M to be a mediator; otherwise, T and M have no relationship. In Equation (2.4), which relates outcome Y to treatment T, ϕ_1 gauges the total effect of treatment T on outcome Y. In addition to Equation (2.4), Equation (2.5) includes mediator M as an explanatory variable. If the magnitude of θ_1 is significantly smaller than that of ϕ_1, this indicates mediation via mediator M because the inclusion of M explains some of the treatment effect on the outcome (VanderWeele 2016). The difference between these two coefficients is often interpreted as a mediated or indirect effect (IE), that is, IE $= \phi_1 - \theta_1$. The remaining treatment effect in Equation (2.5), θ_1, is often taken as a measure of the direct effect (DE), that is, DE $= \theta_1$ (VanderWeele 2016).

In a later section, I examine four sets of time-varying channels that may respond to the expansion and influence drug overdose mortality rates: (1) insurance rates (below 138% of the FPL), (2) distributed controlled substance rates, (3) opioid prescribing rates, and (4) illicit drug seizure rates. These variables are described in the next section.

2.5 Data and Variables

2.5.1 Data Source

Table 2.1 lists variables used in the analyses along with their units of observation and data sources.

2.5.2 Drug Overdose Mortality Rates

Mortality-related variables were derived from restricted-use, individual-level multiple causes of death (MCOD) data from the Centers for Disease Control and Prevention (CDC), which provides information on the universe of death certificates.

The MCOD data provide the following information relevant to my analyses: the decedent's year and month of death, cause of death coded by the International Classification of Diseases Version 10 (ICD-10), and the age and county of residence at the time of death.[11] However, the MCOD data do not provide information on the decedent's health insurance coverage, income, or other socioeconomic statuses relevant to identifying their Medicaid eligibility. Since the expansion only applies to non-elderly adults, I limited the sample to U.S. decedents between the ages of 20 and 64 years. While Medicare covers nearly all elderly people aged 65 or over, adults aged 18 and 19 years were also excluded because the Children's Health Insurance Program provides coverage to eligible children up to the age of 19 years.

I used the CDC's definition based on ICD-10 to identify drug overdose deaths. For opioid overdose deaths, the involved opioids were further identified using the ICD-10 "T-codes." Table 2.2 lists the ICD-10 codes for drug overdose deaths and the opioids involved (Ahmad, Rossen, and Sutton 2021). When analyzing overdose deaths due to prescription opioids, the CDC looks at natural opioids (e.g., morphine and codeine), semisynthetic opioids (e.g., oxycodone, hydrocodone, hydromorphone, and oxymorphone), and methadone. Because information on overdose deaths does not distinguish between legally and illicitly manufactured fentanyl, the CDC separates synthetic opioids (other than methadone) from prescription opioids (CDC 2022).

Drug overdose mortality rates (per 100,000 population) are calculated by dividing the number of drug overdose deaths by the county population aged 20–64 and mul-

[11] I used the county of residence rather than the county of the occurrence of death as the deceased person's county.

tiplying by 100,000. Overdose mortality rates due to specific drug categories (e.g., heroin, synthetic opioids) were defined similarly. For example, mortality rates due to heroin were calculated as the number of heroin deaths (identified by the ICD-10 code in Table 2.2) per 100,000 people. Figure 2.2 illustrates the trends in drug overdose mortality rates at the county and quarter level separately by expansion status during the study period (2010–2018). While in 2010, expansion and non-expansion states (as of the end of 2018) have similar levels of drug overdose mortality rates, the mortality rate in expansion states surpassed that in non-expansion states around 2011. Since then, the mortality-rate gap has been widening, especially since 2014, the year when most expansion states implemented the expansion. These trends have led some to hypothesize that the expansion may play a role in escalating drug overdose deaths, not curbing them.

2.5.3 State Policies

States' ACA Medicaid expansion statuses were drawn from the Kaiser Family Foundation. Policy statuses of other statewide drug-related policies were obtained from the Prescription Drug Abuse Policy System. Table 2.3 lists effective quarters of the expansion (ACA) and other state policies, including the prescription drug monitoring program (PDMP), pain clinic law (PLC), naloxone access law (NAL), Good Samaritan law (GSL), medical marijuana law (MML), and recreational marijuana law (RML). If a state never adopted the policy or adopted the policy after the end of 2018, I do not list an effective quarter.

Table 2.4 presents the number of expansion states by expansion quarter as of the end of the study period, 2018Q4. Most expansion states implemented the expansion on January 1, 2014.

2.5.4 Potential Mediators

Time-varying variables–such as insurance rates, opioid prescription rates, distributed controlled substance rates, and illicit drug seizure rates–could be potential mediators through which the expansion affects drug overdose mortality rates.

In this study, insurance rates refer to countywide health insurance rates for individuals between 18 and 64 years of age with an income at or below 138% of the FPL, that is, the population whose health insurance coverage was most affected by the expansion. Opioid prescribing rates are the number of retail opioid prescriptions dispensed per 100 people, which are confined to the initial or refill prescriptions dispensed at retail pharmacies; thus, these rates do not capture illicitly manufactured opioids (Shakya and Harris 2022). Distributed controlled substance rates are the statewide retail drug distribution rates in terms of grams per 100,000 people; these rates were obtained from Report 3 (Quarterly Distribution in Grams per 100K Population) from the Automation of Reports and Consolidated Orders System (ARCOS), which is a data collection system through which manufacturers and distributors of controlled substances report their transactions to the Drug Enforcement Administration (DEA). However, ARCOS data does not provide information regarding illicit drug purchasing or licit drug purchasing that individuals transport across state borders. To capture the prevalence of illicitly manufactured and distributed opioids, I define illicit drug seizure rates as the number of cases of drug seizure by law enforcement operations per 100,000 people. Drug seizure data are obtained from the DEA's National Forensic Laboratory Information System (NFLIS). The NFLIS collects drug identification results from forensic laboratories that analyze drugs seized by law enforcement agencies (NFLIS n.d.). I calculated two illicit drug seizure rates for fentanyl and heroin. Note that drug seizures may vary in drug volume.

2.5.5 Summary Statistics

Table 2.5 shows units of observations, mean, and standard deviation for variables used in the analysis separately by expansion status (as of the end of 2018) in the pre-expansion period between 2010 and 2013. For each variable, the mean and standard deviation are aggregated within its unit of observation.

2.6 Results

2.6.1 Baseline Specification

Table 2.6 shows the results from estimating variations of the baseline specification (Equation 2.1 in Section 2.4) with drug overdose mortality rates as the dependent variable. To examine how robust the estimate is with various controls, I progressively included more controls in columns (1) to (3). Estimates from columns (1) to (3) are qualitatively similar and are all statistically significant. In addition to column (3), column (4) weights the regression by the county population aged 20–64, obtaining a similarly significant estimate. Based on the estimate in column (4), the expansion increased drug overdose mortality rates by 0.881. This represents a 15.4% increase compared to the average mortality rate of 5.714 in the expansion states in 2013, the last year before the expansion for most expansion states.

Goodman-Bacon (2021) indicated that estimates from traditional two-way fixed effects are biased if the timing of treatment varies across states (which is the case with the expansion) and if the treatment effect is heterogeneous over time. To check whether the estimate in column (4) suffers such bias, column (5) excludes expansion states implementing the expansion after January 1, 2014. The estimate in column (5) remains similar in magnitude to that in column (4), but with reduced precision ($p = 0.057$). Therefore, the estimate in column (4) is robust to heterogeneity in the

timing of treatment and is hereafter referred to as the baseline estimate.[12]

2.6.2 Event Study

Figure 2.3 shows estimates for the leads-and-lags regression (Equation 2.2 in Section 2.4), weighted by the county's population aged 20–64 years. The estimate for the last quarter before the expansion quarter (the first lag) is anchored at zero. Therefore, the estimate for a lead or lag relative to the expansion quarter can be interpreted as an estimated mortality rate difference between the expansion and non-expansion states compared to that difference at the first lag, controlling for other covariates. None of the estimates before the expansion are statistically significant. Therefore, there is no pre-trend of mortality rates in the expansion versus non-expansion states, which is consistent with the parallel trends assumption of the DiD model. Moreover, the difference increased over time after the expansion, indicating that the effects of the expansion on drug overdose mortality rates have been rising over time.

2.6.3 Effects by Drug Category

To investigate which drug categories were responsible for the baseline effects, I separately estimated the effects of the expansion on mortality rates for different drug categories with the baseline specification. Figure 2.4 shows the results. Opioids accounted for more than half of the baseline effects of all drugs. Within this class of opioids, heroin and synthetic opioids (other than methadone) accounted for nearly all the effects of opioids. None of the estimates for the other drug categories was economically or statistically significant.

Prescription opioids mainly consist of natural/semi-synthetic opioids and methadone.

[12]While this study used start dates of PDMPs that allow authorized providers to use the PDMP database voluntarily (referred to as voluntary-access PDMPs), must-access PDMPs require providers to access the database before prescribing or dispensing controlled substances. The baseline estimate is also robust to controlling start dates of must-access PDMPs obtained from Kim (2021) and Mallatt (2020) instead of voluntary-access PDMPs.

However, as shown in Figure 2.4, none of these drug categories led to deaths associated with expansion. Moreover, heroin is manufactured and distributed illegally, and synthetic opioids (e.g., fentanyl and tramadol) are prescribed under the supervision of licensed medical professionals as well as the oversight of the DEA. Mortality rates from synthetic opioids are primarily driven by illicitly manufactured rather than legally prescribed drugs. As such, the effects of the expansion on opioid overdose deaths are likely to be attributed to illicitly manufactured opioids rather than to prescription opioids.

2.6.4 Mechanisms

In Sections 2.6.1 to 2.6.3, I found that the expansion significantly increased drug overdose mortality rates. To investigate why the expansion led to a drug overdose mortality increase, I conducted mediation analyses to examine the potential mechanisms in this section. Specifically, I separately controlled for potential mediators, in addition to the baseline specification, and observed how the estimate on the expansion dummy changed.

Table 2.7 shows the results. In the first column, the baseline estimate is reproduced for comparison. In column (6), in addition to the baseline specification, I controlled for insurance rates, which are widely accepted to have increased significantly because of the expansion. The estimate for insurance rates was negative and not statistically significant. Once the insurance rates were controlled for, the estimate of the expansion increased. This indicates that the expansion increased insurance rates, which in turn reduced mortality rates. To see this, using the terminology of mediation analysis in Section 2.4.3, the expansion's effect through insurance rates (the mediated or indirect effect) equals the total effect (the baseline estimate) minus the direct effect (the Column 6 estimate). Thus, the mediated effect was estimated at $0.881 - 1.112 = -0.231 < 0$, indicating that the expansion reduces drug overdose mortality rates

through expanding insurance. This may occur if more drug addicts are insured and receive treatment for drug addiction, thereby reducing the mortality rate.

Additionally, in column (7), I controlled for distributed controlled substance rates. The estimate for the expansion remained unchanged, and the estimate for distributed controlled substance rates was statistically insignificant. This suggests that distributed controlled substance rates do not explain the baseline estimate.

Moreover, in column (8), I included opioid prescribing rates. The estimate for the expansion was essentially unchanged, and the estimate for opioid prescribing rates was statistically insignificant. This suggests that opioid prescribing rates do not account for the baseline estimate. Taken together, columns (7) and (8) indicate that the effects of the expansion on drug overdose mortality rates are unlikely to be attributed to legally manufactured and prescribed opioids, consistent with the findings in Section 2.6.3.

The findings in Section 2.6.3 suggest that illicitly manufactured opioids—synthetic opioids (e.g., fentanyl) and heroin in particular—might account for the effects of the expansion on drug overdose mortality. To examine this suggestion, in column (9), I controlled for illicit seizure rates for fentanyl and heroin as proxies for illicitly manufactured fentanyl and heroin, respectively. Once these illicit seizure rates were controlled for, the estimate of the expansion was significantly reduced and became statistically insignificant. That is, these illicit seizure rates explained most of the estimated effect. In addition, both illicit seizure rates significantly increased drug overdose mortality rates.

Furthermore, I investigated whether the relationships between these sets of potential mediators and the expansion were causal, a necessary condition for these variables to be channels by which the expansion affected the mortality rates. To this end, I conducted event studies using the baseline specification with each potential mediator separately as the dependent variable. Figure 2.5 shows the estimates from these

leads-and-lags regressions. Panel (a) shows that the expansion discretely and significantly increased insurance rates. The DiD estimate with insurance rates as the dependent variable was 5.395 and statistically significant ($p = 0.000$). In panels (b) and (c), the expansion did not seem to influence distributed controlled substance and opioid prescribing rates. Panel (d) indicates that, in the pre-expansion period, the trend of illicit drug seizure rates (defined as the summation of illicit seizure rates for fentanyl and heroin) in the expansion states was similar to that in the non-expansion states after controlling for other covariates. However, after the expansion, the trend of illicit seizure rates started to rise dramatically in the expansion states compared to the non-expansion states. The DiD estimate with illicit seizure rate as the dependent variable was 7.982, which was statistically significant ($p = 0.015$). Together with the estimates in column (9) of Table 2.7, Panel (d) suggests that the expansion increased the amount of illicitly manufactured and distributed opioids, which in turn increased mortality rates. These results further strengthen the notion that illicitly manufactured opioids are at least partially responsible for the effects of the expansion on drug overdose mortality. A plausible explanation for this notion is that more stringent restrictions on prescription opioids following the expansion caused people to switch from legally to illicitly manufactured drugs, leading to more deaths.

Overall, the results in this section indicate that insurance rates and illicit drug seizure rates, whose effects on mortality rates were in opposite directions, were mechanisms by which the expansion impacted drug overdose mortality.

2.6.5 Heterogeneous Effects

To test the implications of the stylized model in Section 2.3, I examined the heterogeneous effects among the counties. In addition to the baseline specification, I included the interaction terms between the dummy for expansion and other variables, as described below.

Table 2.8 shows the results. In column (10), "Δinsurance rates" are defined as the increase in insurance rates (below 138% of the FPL) between 2013 and 2017 (i.e., before and after the expansion). I use this variable to gauge the extent of the expansion; that is, the expansion has a greater treatment dosage in counties with greater insurance rate increases. The estimate on "Δinsurance rates" is negative and statistically significant. This indicates that the effects of the expansion on overdose mortality are less pronounced in expansion counties with a higher increase in insurance rates due to the expansion compared with the other expansion counties. In column (11), "Δinsurance rates" is replaced by "insurances rates before 2014" (average insurance rates in the pre-expansion period, 2010–2013). Since counties with low pre-expansion insurance rates are expected to be more affected by the expansion, this measure is used as an alternative to "Δinsurance rates" to gauge the extent of the expansion. Namely, the expansion is expected to have a greater treatment dosage in counties with lower "insurance rates before 2014" than others. An advantage of "insurance rates before 2014" is that they are predetermined, whereas "Δinsurance rates" involve post-expansion data and thus could be endogenous. The estimate on the interaction term in column (11) is positive and statistically significant. Therefore, counties with low "insurance rates before 2014" (thus are expected to see greater increases in insurance rates after the expansion and thus to be most influenced by the expansion) are associated with lower drug mortality increases, consistent with the result in column (10). Moreover, in column (12), "Δprescription rates" are defined as the increase in opioid prescription rates between 2013 and 2017. The estimate on "Δprescription rates" is negative and statistically significant. This indicates that the effects of the expansion are lower in expansion counties with a higher increase in prescription rates after the expansion compared with the other expansion counties. Furthermore, in column (13), the estimate on the interaction between the expansion and drug overdose mortality rates before 2014 (i.e., before the expansion) is positive and statistically

significant. This indicates that the effects of the expansion on overdose mortality are more pronounced in expansion counties with previously high overdose rates (or more severe drug overdose problems) than those in other expansion states. Finally, I included all three interaction terms in column (14). The patterns in columns (10) to (12) are preserved in column (14). Hence, the results in this section are consistent with the implications of the stylized model in Section 2.3.

Figure 2.6 shows the relationship between the pre- and post-expansion differences in drug overdose mortality and opioid prescribing rates in expansion states. For each expansion state, indicated by the dot labeled with the state's abbreviation, each difference was estimated using Equation (2.1) in Section 2.4 by comparing the outcome variable, either drug overdose mortality rates or opioid prescribing rates, in the expansion state with those in non-expansion states. Overall, there is a negative relationship between the two differences. The change of mortality rate in expansion states with an opioid prescribing rate increase had no general pattern, either increase or decrease. However, most expansion states with an opioid prescribing rate decrease experienced an increase in the mortality rate. Thus, the total effect (the baseline estimate) was primarily driven by expansion states with an opioid prescribing rate decrease. A plausible explanation is that restrictions in licit opioid prescriptions led more people to substitute illicit for licit opioids, fueling overdose deaths. Since the expansion lowered the out-of-pocket price of prescription opioids, the quantity demanded of prescription opioids was expected to increase, given no restrictions. An opioid prescribing rate decrease may indicate that the expansion state had taken measures to restrict prescription opioids. In comparison, evidence shows that the 2006 implementation of Medicaid Part D, which provided coverage of prescription drugs to the Medicare population, significantly reduced prices and increased the use of prescription drugs (Powell, Pacula, and Taylor 2020). Therefore, there may be greater unmet demand for prescription opioids in expansion states with an opioid

prescribing rate decrease, pressing more people to use illicit drugs and leading to more overdose deaths.

2.6.6 Policy Interaction Effects

As illustrated in Figure 2.1, the expansion reduces the price of prescription opioids and increases the quantity demanded for prescription opioids. Also, supply-side policies imposing regulations on prescription opioids (e.g., prescription drug monitoring programs, pain clinic laws) may reduce the supply for prescription opioids. As such, the interaction of the expansion and restrictive supply-side policies may increase the shortage of prescription opioids, leading more people to use illicit drugs with a greater risk of overdose and death. To examine this, I estimate how the expansion, pain clinic laws, and their interaction affect opioid prescribing rates, illicit drug seizure rates, and drug overdose mortality rates in this section.[13] As all expansion states have a prescription drug monitoring program before the expansion, I only consider pain clinic laws to represent restrictive supply-side policies. States adopting the expansion and pain clinic laws during the study period include Arizona, Kentucky, Louisiana, Ohio, and West Virginia.[14]

Table 2.9 reports the results. Column (15) shows that, in states with both the expansion and pain clinic laws, there was a significant reduction of opioid prescribing rates by 8.647 ($p = 0.000$) or 8.8% compared to the mean in these states, consistent with Shakya and Harris (2022). This indicates that expansion states with more stringent opioid restrictions experienced significant decreases in opioid prescribing rates. In addition, the estimate on the expansion was still statistically insignificant.

[13]Pain clinic laws target clinics with an extraordinarily high volume of prescriptions of controlled substances (e.g., opioids). These laws impose oversight on pain clinics, such as routine inspections, limitations on dispensing and clinic ownership, as well as civil and criminal penalties for violations (Rutkow, Vernick, and Alexander 2017).

[14]Arizona implemented pain clinic laws in 2018; Kentucky in 2012; Louisiana in 2006; Ohio in 2011; and West Virginia in 2012. All these states adopted the expansion in 2014, except Louisiana, which implemented the expansion in 2016.

Column (16) estimated that the interaction between the expansion and pain clinic laws was associated with a marginally significant increase in illicit drug seizure rates by 30.544 ($p = 0.052$) or a 162.9% increase relative to the mean in states with both policies. With the interaction of both policies included, the expansion itself was not associated with a significant rise in the seizure rates, suggesting that expansion states with pain clinic laws mainly drove the previously found association between the two. In column (17), I found that states with both policies saw an increase in drug overdose mortality rates by 2.036 ($p = 0.004$) or 20.9% compared with the mean. Meanwhile, the estimate on the expansion became statistically insignificant, indicating that the baseline estimate was mainly driven by these states with both policies. Finally, in addition to column (17), column (18) controlled for illicit drug seizure rates. While illicit drug seizure rates were significantly associated with an increase in drug overdose mortality rates, the estimate on the interaction between the two policies became insignificant. This suggests that the policy interaction effects found in column (17) were mainly driven by the seizure rates, i.e., the seizure rates constitute a major mediator. Across all columns, estimates on pain clinic laws and expansion were statistically insignificant but consistent in sign.

Together, the effects in states with a combination of the expansion and pain clinic laws were more prominent than those in states with only one of the two policies, consistent with the stylized model in Section 2.3. These findings suggest that the expansion further reduced opioid prescribing rates when coupled with opioid restrictions, escalating the prevalence of illicit drugs and leading to greater drug overdose mortality. They also suggest that illicit drug seizures are endogenous and a function of the restrictions.

2.7 Conclusion

Deaths due to drug overdose have been rising dramatically in the U.S. over the past two decades. There are two competing views on the cause of this dramatic rise, "more prescriptions, more deaths" and "more restrictions, more deaths" (Miron, Sollenberger, and Nicolae 2019). The conventional explanation places blame for the rise on the expansion of prescriptions and advertising of opioids in the 1990s. This "more prescriptions, more deaths" explanation has spurred federal and state governments to take measures to curtail opioid prescriptions and increase the cost of opioid production. Proponents of this view argue that policies restricting the supply of prescription opioids would reduce deaths due to overdose. However, "more restrictions, more deaths," holds that stringent restrictions on prescription opioids compel people to use illicitly manufactured drugs, which are more dangerous than legally prescribed versions, increasing the risk of overdose and death. Over the past decade, drug overdose mortality rates from heroin and synthetic drugs such as fentanyl have continued to rise despite reduced prescriptions. In addition, stringent restrictions on prescription opioids can result in undertreatment of pain, harm patients' quality of life, and even drive some to commit suicide. Accordingly, loosening access to prescription opioids would disincentivize the use of illicitly manufactured drugs, curbing overdose deaths.

In this study, I investigated how the ACA's Medicaid expansion affects drug overdose mortality rates. On the one hand, the expansion lowered the cost of opioid addiction treatment, helping to alleviate drug opioid dependence and overdose deaths. On the other hand, it reduced the cost of opioid prescriptions and thereby made opioids more accessible, possibly leading to increased addiction and deaths. Using a DiD framework, I estimated that the expansion increased drug overdose mortality rates by 0.881 per 100,000 people at the county and quarter level, over half of which were driven by opioids. This represents a 15.4% increase compared with the average

drug overdose mortality rate in the expansion counties prior to the expansion–a sizable and statistically significant effect. An event study showed no significant trend in drug overdose mortality rates in the expansion counties relative to the non-expansion counties before the expansion, given other controls. However, this trend steadily increased after the expansion. Further analyses of the involved drugs suggested that the effects on opioid overdose deaths were almost all driven by illicitly manufactured opioids, such as heroin and synthetic opioids, rather than legally provided opioids. Moreover, potential mechanisms that connected the expansion to mortality rates were investigated. I found evidence that the expansion increased insurance rates (below 138% of the FPL), which reduced the mortality rates. I also found evidence suggesting that the expansion fueled the prevalence of illicitly manufactured drugs, thus raising mortality rates in the expansion counties compared with non-expansion counties.

To illustrate how the expansion can exacerbate overdose deaths from illicitly manufactured drugs in the presence of prescription opioid restrictions, in Section 2.3, I constructed a stylized model that has implications for the heterogeneous effects and policy interaction effects across counties. To test the stylized model, I found that the effects of the expansion were less pronounced in expansion counties with higher increases in insurance or opioid prescribing rates after the expansion compared to those in other expansion counties. In contrast, the effects were more pronounced in expansion counties with higher drug overdose mortality rates (more severe drug overdose problems) before the expansion. Further- more, the interaction of the expansion and pain clinic laws decreased opioid prescribing rates and increased the prevalence of illicit drugs and drug overdose mortality. These results are consistent with the implications of the stylized model.

Taken together, this study's findings support the view of "more restrictions, more deaths" rather than the conventional view of "more prescriptions, more deaths." This study highlights the importance of policymakers assessing and weighing the

costs and benefits of restricting legal access to opioids. Although greater access to prescription opioids may fuel opioid dependence and overdose, it may reduce pain, improve patients' quality of life, and curtail the prevalence of underground drug consumption. In addition, policymakers could consider a mixed strategy that targets the improper use of prescription opioids, simultaneously meeting the demand for prescription opioids and increasing the treatment for SUD.

However, this study does not provide conclusive evidence indicating whether the dramatic post-expansion increase in illicit fentanyl seizure rates in expansion states relative to non-expansion states was a coincidence or a consequence of the expansion. If it was a consequence, then the question of whether the expansion fuelled drug overdose deaths through the stringent accompanying prescription restrictions remains unanswered. Moreover, this study does not rule out alternative explanations. For example, Abouk et al. (2021) found a substantial pre-expansion increase in heroin mortality in expansion states relative to non-expansion states. As heroin users may blend heroin with fentanyl, expansion states could be more susceptible to illicitly manufactured fentanyl, increasing its prevalence and resulting deaths. Moreover, the adoption of Medicaid expansion could have been motivated by drug-related mortality trends. However, the statistically insignificant differential pre-trend found in the event study renders this explanation unconvincing.

In line with this study's argument, future research needs to explore (1) whether the finding that the expansion did not increase opioid prescribing rates as expected was due to opioid prescription restrictions, (2) whether the expansion heightened the demand for prescription opioids exceeding the limits on prescriptions, and (3) whether more people with unmet demand resorted to underground markets and thus faced greater risk of overdose.

Tables

Table 2.1: Variables, Units, and Data Sources

Variable(s)	Unit	Data Source
Mortality rates	county/quarter	National Vital Statistics System Multiple Causes of Death (MCOD) restricted-use data
Medicaid expansion	county/quarter	Kaiser Family Foundation
Other statewide drug-related policies	county/quarter	Prescription Drug Abuse Policy System
Demographics	county/year	National Cancer Institute's Surveillance, Epidemiology, and End Results Program (SEER)
Unemployment rates	county/quarter	Bureau of Labor Statistics
Poverty rates	county/year	U.S. Census Bureau Small Area Income and Poverty Estimates (SAIPE)
Median household income	county/year	U.S. Census Bureau Small Area Income and Poverty Estimates (SAIPE)
Insurance rates	county/year	Small Area Health Insurance Estimates (SAHIE)
Opioid prescribing rates	county/year	Centers for Disease Control and Prevention (CDC)
Distributed controlled substance rates	state/quarter	Automated Reports and Consolidated Ordering System (ARCOS)
Illicit drug seizure rates	state/year	National Forensic Laboratory Information System (NFLIS)

Table 2.2: ICD 10 Codes for Drug Overdose Deaths and the Involved Opioids

Cause of Death	Codes
Drug overdose	X40–X44, X60–64, X85, or Y10–Y14
Opioid overdose	
Opium	T40.0
Heroin	T40.1
Natural/Semisynthetic opioids	T40.2
Methadone	T40.3
Synthetic opioids (other than methadone)	T40.4
Other and unspecified opioids	T40.6

Table 2.3: Effective Quarters of Statewide Drug-Related Policies

state	ACA	PDMP	PCL	NAL	GSL	MML	RML
Alaska	2015Q3	2008Q3		2016Q1	2008Q3	1999Q1	2015Q1
Alabama		2005Q2	2013Q2	2015Q2	2015Q2		
Arkansas	2014Q1	2011Q1		2015Q3	2015Q3	2016Q4	
Arizona	2014Q1	2007Q3	2018Q2	2016Q3	2018Q2	2010Q4	
California	2014Q1	1939Q1		2008Q1	2013Q1	1996Q4	2017Q1
Colorado	2014Q1	2005Q2		2013Q2	2012Q2	2000Q4	2014Q3
Connecticut	2014Q1	2006Q2		2003Q4	2011Q4	2012Q2	
District of Columbia	2014Q1	2014Q1		2013Q1	2013Q1	2010Q3	2014Q3
Delaware	2014Q1	2010Q3		2014Q3	2013Q3	2011Q3	
Florida		2009Q2	2010Q4	2015Q2	2012Q4	2017Q1	
Georgia		2011Q2	2013Q3	2014Q2	2014Q2		
Hawaii	2014Q1	1943Q1		2016Q2	2015Q3	2000Q2	
Iowa	2014Q1	2006Q2		2016Q2	2018Q3		
Idaho		1967Q1		2015Q3	2018Q3		
Illinois	2014Q1	1961Q1		2010Q1	2012Q2	2014Q1	
Indiana	2015Q1	1997Q1		2015Q2	2014Q1		
Kansas		2008Q3		2017Q3			
Kentucky	2014Q1	1998Q3	2012Q3	2013Q2	2015Q1		
Louisiana	2016Q3	2006Q3	2006Q1	2015Q3	2014Q3		
Massachusetts	2014Q1	1992Q1		2012Q3	2012Q3	2013Q1	2016Q4
Maryland	2014Q1	2011Q2		2013Q4	2009Q4	2013Q4	
Maine		2003Q2		2014Q2		1999Q4	2017Q1
Michigan	2014Q2	1988Q1		2014Q4	2017Q1	2008Q4	
Minnesota	2014Q1	2007Q3		2014Q2	2014Q3	2004Q2	
Missouri				2016Q3	2017Q3		
Mississippi		2005Q1	2011Q2	2015Q3	2015Q3		
Montana	2016Q1	2011Q3		2017Q2	2017Q2	2004Q4	
North Carolina		2005Q3		2013Q2	2013Q2		
North Dakota	2014Q1	2005Q4		2015Q3	2015Q3	2016Q4	
Nebraska		2011Q2		2015Q2	2017Q3		
New Hampshire	2014Q3	2012Q2		2015Q2	2015Q3	2013Q3	
New Jersey	2014Q1	2008Q1		2013Q3	2013Q2	2010Q4	
New Mexico	2014Q1	2004Q3		2001Q2	2007Q2	2007Q3	
Nevada	2014Q1	1995Q2		2015Q4	2015Q4	2001Q4	2017Q1
New York	2014Q1	1972Q1		2006Q2	2011Q3	2014Q3	
Ohio	2014Q1	2005Q2	2011Q2	2014Q1	2016Q3	2016Q3	
Oklahoma		1990Q2		2013Q4			
Oregon	2014Q1	2009Q3		2013Q2	2016Q1	1998Q4	2015Q3
Pennsylvania	2015Q1	1972Q1		2014Q4	2014Q4	2016Q2	
Rhode Island	2014Q1	1978Q1		2012Q2	2012Q2	2006Q1	
South Carolina		2006Q2		2015Q2	2017Q2		
South Dakota		2010Q1		2016Q3	2017Q3		
Tennessee		2003Q1	2011Q2	2014Q3	2015Q3		
Texas		1981Q3	2009Q3	2015Q3			
Utah		1995Q1		2014Q2	2014Q1		
Virginia		2002Q2		2013Q3	2015Q3		
Vermont	2014Q1	2006Q2		2013Q3	2013Q2	2004Q3	
Washington	2014Q1	2007Q3		2010Q2	2010Q2	1998Q4	2014Q2
Wisconsin		2010Q2	2016Q1	2014Q2	2014Q2		
West Virginia	2014Q1	1995Q3	2012Q2	2015Q2	2015Q2		
Wyoming		2003Q1		2017Q3			

Notes: ACA stands for ACA's Medicaid expansion; PDMP, prescription drug monitoring program; PLC, pain clinic law; NAL, naloxone access law; GSL, Good Samaritan law; MML, medical marijuana law; RML, recreational marijuana law.

Table 2.4: Number of Expansion States by Expansion Quarter

Expansion quarter	Number of states
2014Q1	25
2014Q2	1
2014Q3	1
2015Q1	2
2015Q3	1
2016Q1	1
2016Q3	1

Table 2.5: Summary Statistics by Expansion Status between 2010 and 2013

Variables	Unit of observation	Expansion states	Non-expansion states
		Mean (Standard deviation)	Mean (Standard deviation)
Outcomes			
Drug overdose deaths per 100,000 (ages 20–64)	county/year-quarter	5.42 (8.25)	4.65 (8.43)
Opioid overdose deaths per 100,000 (ages 20–64)	county/year-quarter	1.85 (4.83)	1.56 (4.51)
Potential mediators			
Insurance rates (below 138% FPL)	county/year	63.59 (8.75)	57.24 (8.43)
Distributed controlled substances (grams per 100,000)	state/year-quarter	17,950.35 (4,182.82)	22,734.28 (18,748.41)
Opioid prescriptions (per 100)	county/year	89.63 (44.43)	97.73 (53.32)
Illicit fentanyl seizures (per 100,000)	state/year	0.26 (0.25)	0.35 (0.30)
Illicit heroin seizures (per 100,000)	state/year	0.04 (0.12)	0.00 (0.00)
Controls			
% Male	county/year	0.50 (0.02)	0.50 (0.02)
% White	county/year	0.90 (0.14)	0.84 (0.18)
% Black	county/year	0.06 (0.10)	0.13 (0.17)
% Asian	county/year	0.02 (0.05)	0.01 (0.02)
% Hispanic	county/year	0.07 (0.11)	0.10 (0.15)
% Population ages under 20	county/year	0.25 (0.03)	0.26 (0.04)
% Population ages 20–24	county/year	0.06 (0.02)	0.06 (0.03)
% Population ages 25–34	county/year	0.12 (0.02)	0.12 (0.02)
% Population ages 35–44	county/year	0.12 (0.02)	0.12 (0.02)
% Population ages 45–54	county/year	0.15 (0.01)	0.14 (0.02)
% Population ages 55–64	county/year	0.14 (0.02)	0.13 (0.02)
Unemployment rate	county/year-quarter	8.57 (3.09)	8.13 (3.15)
Poverty rate	county/year	15.96 (6.00)	18.16 (6.72)
Median household income	county/year	46,343.19 (11,535.84)	42,401.05 (10,155.84)
Number of counties		1,502	1,642
Number of states		32	19

Table 2.6: Effects of the Affordable Care Act's Medicaid Expansion on Drug Overdose Mortality Rates

	Drug overdose mortality rates				
	(1)	(2)	(3)	(4)	(5)
Expansion	1.168***	1.137***	0.859***	0.881***	0.823*
	(0.403)	(0.346)	(0.285)	(0.299)	(0.421)
Mean (2013)	5.653	5.653	5.653	5.714	5.700
Demographics	No	Yes	Yes	Yes	Yes
Other drug-related policies	No	No	Yes	Yes	Yes
Weighted by the population aged 20–64 years	No	No	No	Yes	Yes
Exclude later expansion states	No	No	No	No	Yes
Observations	113,044	112,888	112,888	112,888	98,568

Notes: The data are panel data with the unit of observation at the county/quarter level from 2010 to 2018. The dependent variable was the drug overdose mortality rate per 100,000 people aged 20–64 years. All columns include county and quarter fixed effects. The row "Mean (2013)" shows the average mortality rates in the expansion states in 2013. "Later expansion states" refer to expansion states that implemented the expansion after January 1, 2014. Standard errors are clustered at the state level and reported in parentheses. *$p < 0.1$ ** $p < 0.05$ *** $p < 0.01$.

Table 2.7: Effects of the Expansion on Drug Overdose Mortality Rates Controlling for Potential Mediators

	Drug overdose mortality rates				
	baseline	(6)	(7)	(8)	(9)
Expansion	0.881***	1.112***	0.879***	0.863***	0.301
	(0.299)	(0.321)	(0.299)	(0.294)	(0.229)
Insurance rates (below 138% of the FPL)		-0.0427			
		(0.0398)			
Distributed controlled substance rates (grams per 100,000 people)			-0.00000268		
			(0.00000329)		
Opioid prescribing rates (per 100 people)				-0.0138	
				(0.00863)	
Illicit fentanyl seizure rates (per 100,000 people)					0.0537***
					(0.00680)
Illicit heroin seizure rates (per 100,000 people)					6.151***
					(1.585)
Observations	112,888	112,888	112,888	102,720	112,888

Notes: The data are panel data, with the unit of observation at the county/quarter level from 2010 to 2018. The dependent variable was the drug overdose mortality rate per 100,000 people aged 20–64 years. All columns include the control variables, county fixed effects, and quarter fixed effects. The regressions are weighted by the county's population aged 20–64 years. Standard errors are clustered at the state level and reported in parentheses. *$p < 0.1$ ** $p < 0.05$ *** $p < 0.01$.

Table 2.8: Heterogeneous Effects

	Drug overdose mortality rates				
	(10)	(11)	(12)	(13)	(14)
Expansion × Δinsurance rates	-0.105**				-0.118***
	(0.0413)				(0.0324)
Expansion × Insurance rates before 2014		0.0738***			
		(0.0224)			
Expansion × Δprescribing rates			-0.0651***		-0.0373***
			(0.0182)		(0.0127)
Expansion × Mortality rates before 2014				0.285**	0.238*
				(0.126)	(0.121)
Expansion	2.848***	-3.749***	-0.437	-0.726	0.983
	(0.858)	(1.345)	(0.377)	(0.642)	(0.914)
Observations	112,888	112,888	98,716	112,788	98,676

Notes: The data are panel data, with the unit of observation at the county/quarter level from 2010 to 2018. The dependent variable was the drug overdose mortality rate per 100,000 people aged 20–64 years. All columns include the control variables, county fixed effects, and quarter fixed effects. The regressions are weighted by the county population aged 20–64 years. Standard errors are clustered at the state level and reported in parentheses. "Δinsurance rates" is the increase in insurance rates (below 138% of the FPL) between 2013 and 2017. "Insurance rates before 2014" are the average insurance rates before 2014. "Δprescription rates" is the increase in opioid prescription rates between 2013 and 2017. "Mortality rates before 2014" are the average drug overdose mortality rates before 2014. *$p < 0.1$ ** $p < 0.05$ *** $p < 0.01$.

Table 2.9: Effects of the Interaction of the Expansion and Pain Clinic Laws on Various Variables

	Opioid prescribing rates	Illicit drug seizure rates	Drug overdose mortality rates	Drug overdose mortality rates
	(15)	(16)	(17)	(18)
Expansion × Pain clinic laws	-8.647***	30.544*	2.036***	0.522
	(2.007)	(15.37)	(0.667)	(0.461)
Expansion	0.831	2.035	0.485	0.384
	(1.157)	(3.848)	(0.317)	(0.263)
Pain clinic laws	-3.197*	-3.241	-0.349	-0.188
	(1.817)	(2.676)	(0.258)	(0.278)
Illicit drug seizure rates				0.0496***
				(0.00825)
Mean	98.523	18.749	9.743	9.743
Observations	102,720	112,888	112,888	112,888

Notes: Data are panel with the unit of observation at the county/quarter level from 2010 to 2018. For each column, the dependent variable is indicated in the first row. All columns include control variables, county, and quarter fixed effects. The row "mean" shows averages of the corresponding dependent variable in states with the expansion and a pain clinic law. Standard errors are clustered at the state level and reported in parentheses. Regressions are weighted by the county population aged between 20 and 64. *$p < 0.1$ ** $p < 0.05$ *** $p < 0.01$.

Figures

Figure 2.1: Supply and Demand of Prescription Opioids Before and After the Expansion

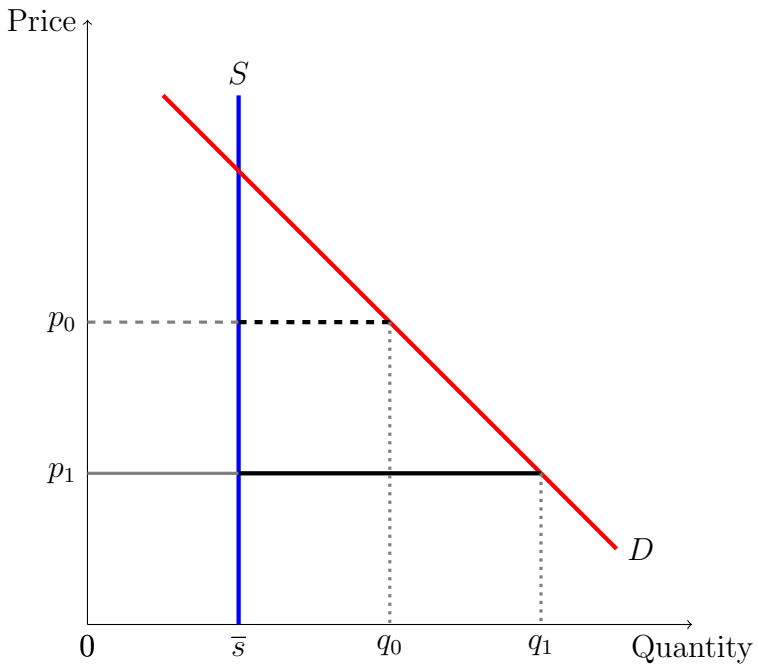
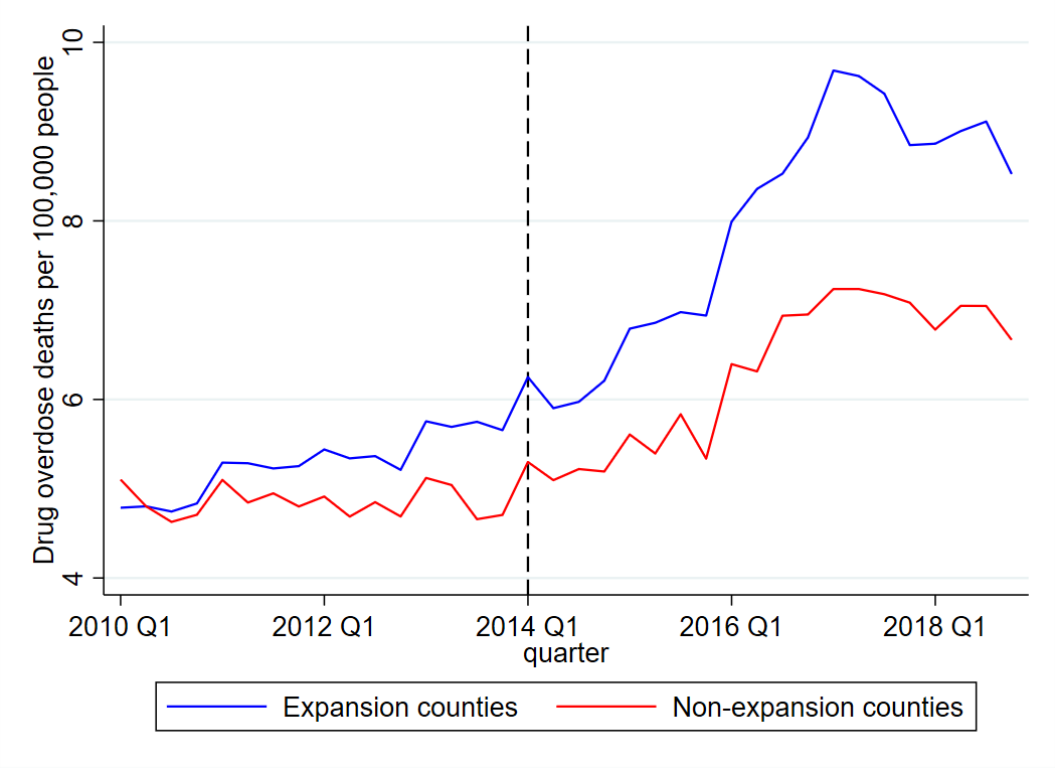
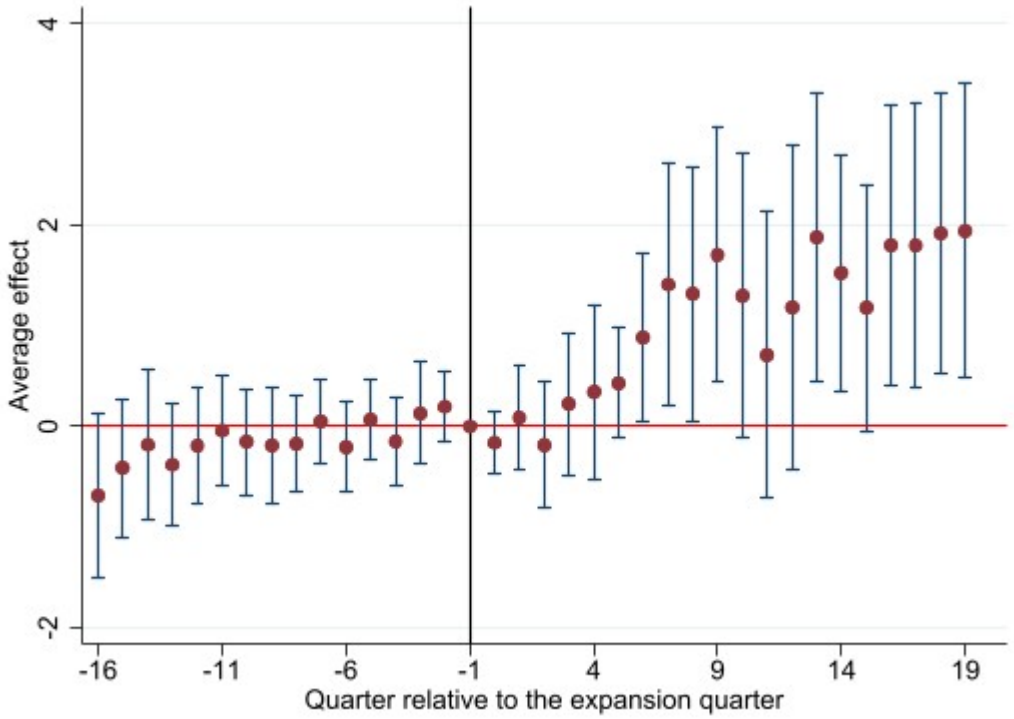


Figure 2.2: Averages in Drug Overdose Mortality Rates per 100,000 People at the County and Quarter Level by Expansion Status Between 2010 and 2018



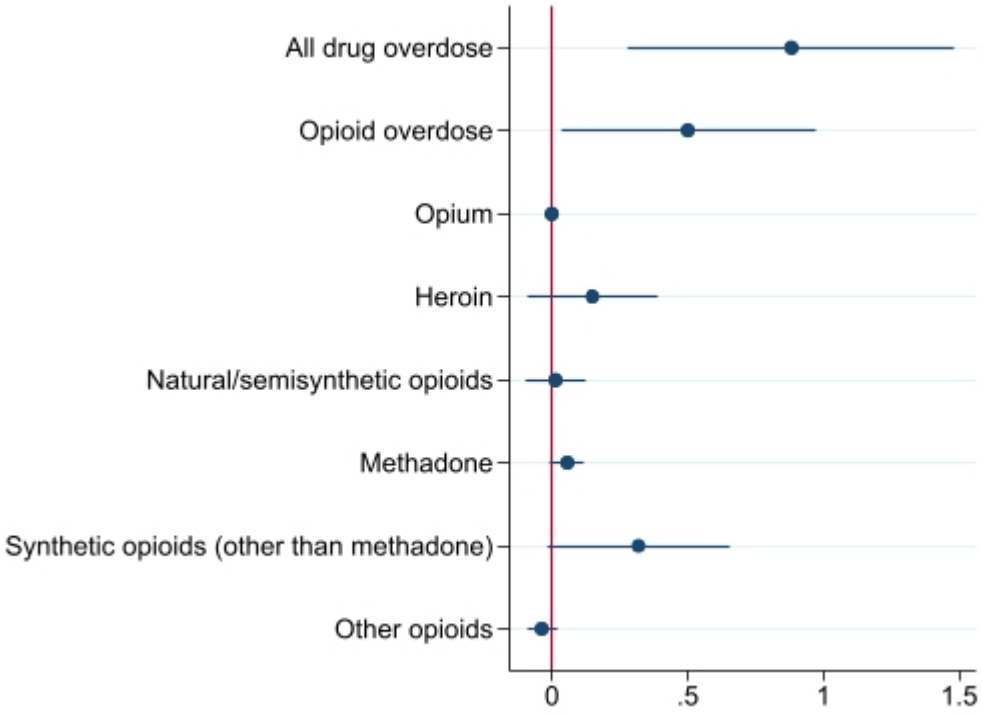
Notes: The dashed vertical line at the 2014 Q1 mark indicates the quarter in which the expansion went into effect in most expansion states.

Figure 2.3: Lead and Lag Estimates for the Effect of the Affordable Care Act's Medicaid Expansion on Drug Overdose Mortality Rates



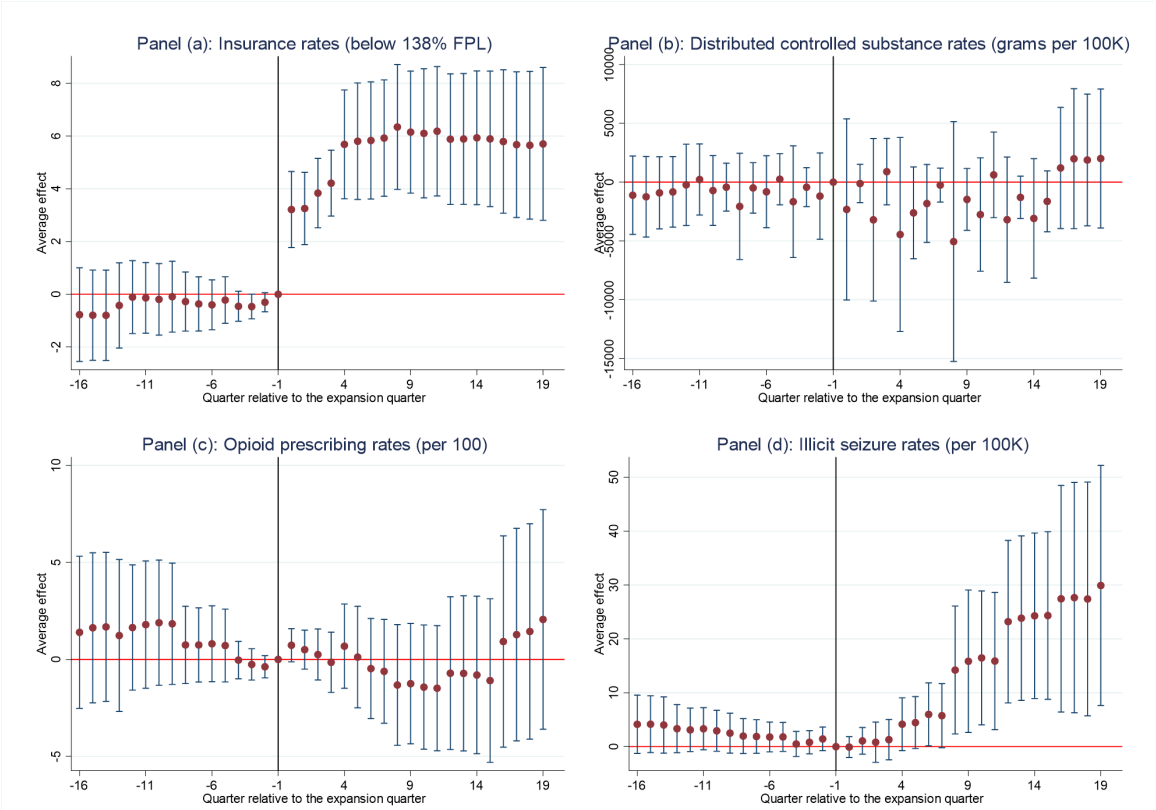
Notes: Dots show the lead-and-lag estimates. Vertical bars show 95% confidence intervals using standard errors clustered at the state level. The dependent variable is drug overdose mortality rates per 100,000 people aged 20–64 years. Covariates include control variables, county fixed effects, and quarter fixed effects. The regression was weighted by the county population aged 20–64 years. The estimate for the first lag (the last quarter before the expansion quarter) is anchored at zero, as indicated by the vertical line.

Figure 2.4: Effects of the Affordable Care Act's Medicaid Expansion on Mortality Rates Due to Different Drug Categories



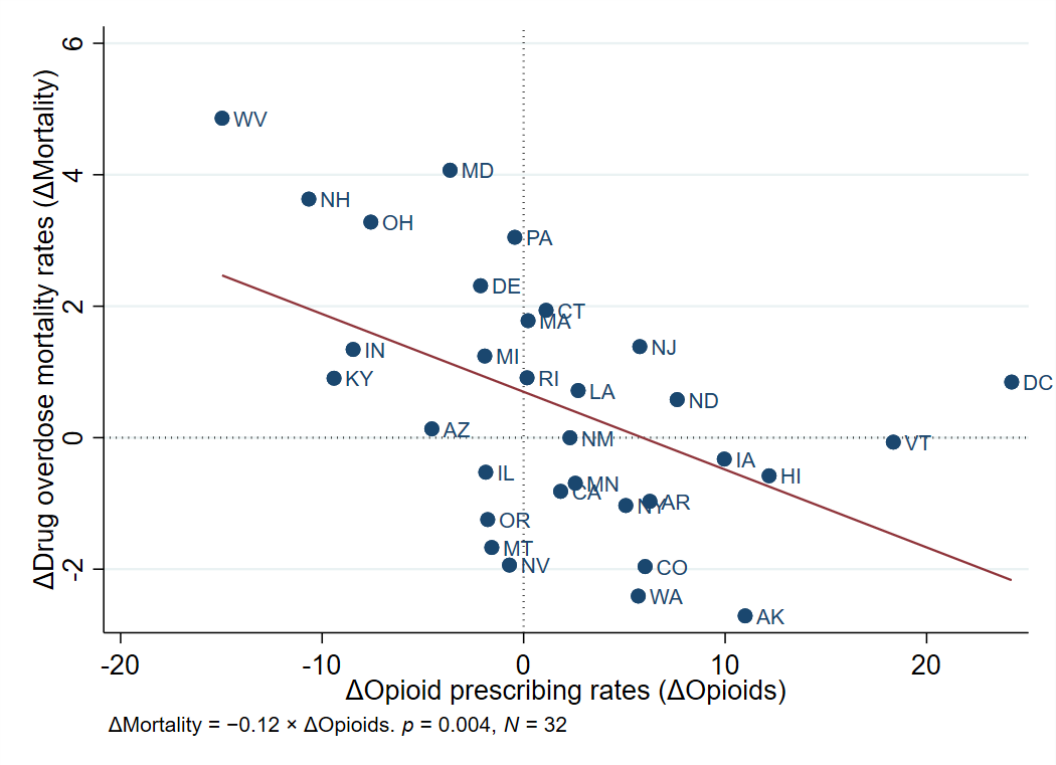
Notes: Dots show the separately estimated effects of the expansion on mortality rates for different drug categories with the baseline specification. Horizontal bars show 95% confidence intervals using standard errors clustered at the state level. Covariates include control variables, county fixed effects, and quarter fixed effects. Each regression was weighted by the county population aged 20–64 years.

Figure 2.5: Lead and Lag Estimates With Different Potential Mediators as the Dependent Variable



Notes: Dots show the lead-and-lag estimates. Vertical bars show 95% confidence intervals using standard errors clustered at the state level. In each panel, the title indicates the dependent variable. Covariates include control variables, county fixed effects, and quarter fixed effects. Each regression was weighted by the county population aged 20–64 years. The estimates for the first lag (the last quarter before the expansion quarter) are anchored at zero, as indicated by the vertical lines.

Figure 2.6: Relationship Between the Pre- And Post-expansion Differences in Drug Overdose Mortality and Opioid Prescribing Rates



Notes: In this figure, each expansion state corresponds to a dot labeled by the state's abbreviation. "Δdrug overdose mortality rates" and "Δopioid prescribing rates" are pre- and post-expansion differences in drug overdose mortality rates and opioid prescribing rates, respectively. For each expansion state, each difference is estimated using Equation (2.1) in Section 2.4 by comparing the outcome variable, either drug overdose mortality rates or opioid prescribing rates, in the indicated expansion state with those in non-expansion states.

Chapter 3

Is Marijuana a Gateway Drug? Evidence From the 1997 National Longitudinal Survey of Youth

3.1 Introduction

Marijuana is the most widely used federally illicit drug in the U.S. In 2020, among people aged 12 years or older in the U.S., 17.9% (or about 49.6 million people) reported using marijuana in the past 12 months (NIH 2020). Studies have consistently found that marijuana use commonly precedes the use of "hard drugs" (e.g., cocaine, heroin), which can seriously harm the user's health. In contrast, "soft drugs," such as alcohol and marijuana, pose fewer health risks. For example, some medical professionals have used marijuana to treat certain medical conditions (e.g., cancer, seizures, chronic pain). Also, heroin and cocaine score higher than marijuana in terms of physical harm, dependence, and social harm in a scoring proposed by Nutt et al. (2007). The use of any hard drugs, even occasionally, can significantly increase the risk of adverse health effects (e.g., abuse, dependence, addiction, overdose), raising public health concerns.

The observed sequence of drug use can be explained by the gateway hypothesis, first proposed by Kandel (1975), which posits that experimentation with marijuana increases the likelihood that an individual will progress to hard drug use. Gateway

effects may arise via the following three channels. First, marijuana use may provide instant pleasure to users, causing them to crave more potent hard drugs in the hopes of experiencing heightened gratification. If this is the case, legalizing marijuana use, which is likely to increase marijuana usage, would increase the consumption of hard drugs. Second, interactions with drug dealers and peer users may increase the chance for marijuana users to interact with suppliers of hard drugs.[1] To remedy this, drug policies aimed at separating the markets for marijuana and hard drugs may be effective in reducing the number of individuals who progress to the use of hard drugs. Such policies exist in the Netherlands, where the sale of a limited quantity of marijuana is permitted in "coffee shops," attempting to keep marijuana experimenters and users away from hard drugs. Meanwhile, the sale of hard drugs is subject to severe punishment. The logic is that strict prohibition of soft drugs would stimulate the underground market, pushing soft drug users into hard drugs (Van Ours 2003). In addition, by legalizing marijuana, the government can oversee and track the sale of marijuana, thereby improving the transparency, safety, and quality of the product being sold. Furthermore, the legalization of marijuana would introduce competition to the marijuana market, thus deterring drug cartels that only exist in underground markets and possibly curbing hard drug use. Third, light to moderate marijuana use has few adverse health effects (Sabia et al. 2021). Therefore, those who started using marijuana first may have formed a false perception that other illicit drugs are also safe, leading them to experiment with hard drugs. If so, creating additional and enhancing current health warnings relating to the adverse effects of hard drugs may help correct users' false perceptions and reduce hard drug usage.

However, the observed progression from marijuana to hard drug use does not necessarily imply that usage of marijuana causes hard drug use. Certain characteristics

[1]Bretteville-Jensen and Jacobi (2011) reported that 23% and 19% of non-marijuana users claimed to be able to obtain heroin and cocaine respectively within three days, whereas the corresponding figures for marijuana users were 57% and 33% respectively. In addition, more marijuana users than non-users claimed that they had been offered hard drugs (80% versus 26%).

that could cause some individuals to be more susceptible to soft and hard drug usage include genetic predisposition, psychological problems (e.g., stress, depression, and childhood trauma), family background, peer environment, and accessibility.[2] Regardless of their history of marijuana use, individuals with these characteristics could start using marijuana because it is cheaper and more readily available than hard drugs (Pudney 2003). Pudney (2003) also suggested that the relative costs between hard drugs and marijuana may attribute to the observed sequence of drug use. Since people tend to have few resources early in life, they may only afford marijuana at first and progress to hard drugs later as their earnings increase.

Ideally, randomized human trials should be conducted to investigate the gateway hypothesis, but such trials have been deemed to be unethical and illegal worldwide. The existing medical and economic literature on the gateway hypothesis relies mainly on observational data (e.g., self-reported survey data) and researchers disagree on whether marijuana is a gateway drug. See Nöel and Wang (2018) for a review of this literature. Currently, the mechanism underlying the observed sequence of drug use is not well understood, with many studies not distinguishing correlation from causation. This limitation leads to results that can be explained by confounding unobserved heterogeneity (e.g., genetic predisposition, psychological problems, policy environment). However, despite limited empirical evidence, opponents of the legalization of marijuana often cite the gateway hypothesis in their argument. Currently, medical marijuana is legal in 38 states and D.C., and recreational marijuana is legal in 19 states and D.C. As the U.S. is considering decriminalizing marijuana at the federal level, which we submit would increase marijuana consumption, it is of great significance that the validity of the gateway hypothesis is investigated in order to predict whether such legislation will subsequently lead to the increased use of hard

[2]Bretteville-Jensen and Jacobi (2011) refer to accessibility as economic, cultural, and physical factors that affect an individual's access to drugs, such as monetary and non-monetary costs (e.g., transaction costs, social stigma).

drugs.

This study aims to disentangle the gateway effects from the results of unobserved heterogeneity using data collected in the National Longitudinal Survey of Youth 1997 (NLSY97). The research question is: does marijuana use hastens the onset of hard drug use among adolescents and young adults? The current study contributes to the literature in the following two aspects. First, while studies that model the intertemporal dependence between marijuana and hard drug use (e.g., DeSimone 1998; Deza 2015) answer how current marijuana use affects future hard drug use, this study investigates how marijuana use shortens the onset of hard drug initiation, which is more in line with the conventional notion of the gateway effect. Second, previous studies with a similar interpretation to ours (e.g., Beenstock and Rahav 2002; Van Ours 2003; Pudney 2003; Melberg, Jones, and Bretteville-Jensen 2010; Bretteville-Jensen and Jacobi 2011) all used cross-sectional data, which does not account for fixed and time-varying individual heterogeneity. Our method is different as we apply a bivariate survival panel model to the longitudinal survey data, allowing for time-varying observables as well as accounting for confounding fixed and time-varying unobservables. Moreover, the proposed model is a more flexible method for heterogeneity analysis, which informs about potential mechanisms that may cause the gateway effects, such as early marijuana initiation and the frequency of marijuana use.

I found strong evidence that marijuana use hastens hard drug initiation in the NLSY sample, even after controlling for individual characteristics. Based on my preferred specification, marijuana use significantly increased the risk of hard drug initiation by 139%, which is robust to different sets of covariates and similar to the result by Melberg, Jones, and Bretteville-Jensen (2010). Moreover, gateway effects are more prominent in those who first used marijuana early or more frequently, suggesting that the onset age and frequency of marijuana use may determine the gateway effects. Thus, these findings add to the literature in understanding mechanisms behind the

gateway effects. They also have policy implications regarding the importance of postponing the onset age and reducing the frequency of marijuana use.

3.2 Related Literature

Random animal trials generally support the gateway hypothesis (e.g., Pistis et al. 2004; Ellgren, Spano, and Hurd 2007; Panlilio et al. 2013). Results from animal studies have revealed potential biological mechanisms that give rise to the gateway effect. For example, studies have shown that exposing rats to THC (a component of marijuana that is responsible for most of the psychological effects of the drug) during a critical phase of brain development (e.g., adolescence) is associated with an altered brain reward system that increases the likelihood that the rats will self-administer other drugs, such as heroin (NIH 2020). To the extent that the findings of these studies are applicable to humans, this could help to explain why individuals who began marijuana use in youth were found to be more susceptible to hard drug usage than those who began later in life.

Another aspect of research on gateway effects examines how marijuana laws, either medical or recreational, affect hard drug use (e.g., Wen, Hockenberry, and Cummings 2014; Chu 2015; DeAngelo and Redford 2015; Sabia et al. 2021). Studies related to this component generally found that laws regarding the regulation of marijuana usage increased marijuana use, but had no impact on hard drug use, thus providing no support for the gateway hypothesis. However, as Shover et al. (2019) cautioned, one must refrain from drawing a causal connection at the individual level from data at the geographical level (e.g., state, county).[3] Moreover, since marijuana laws may

[3]An earlier study, Bachhuber et al. (2014), found that from 1999 to 2010, states with medical marijuana laws saw slower rates of increase in opioid overdose death rates than other states. However, extending Bachhuber et al.'s analysis through 2017 using the same method, Shover et al. (2019) show that the findings not only do not hold for the extended period, but the relationship reversed in sign, from -21% to 23%. From this, the authors deduced that the earlier found relationship were spurious, likely due to unobserved factors to researchers.

only affect a small subpopulation, insignificant estimates based on geographical-level data may mask the actual effect of policies in the affected subpopulation. In contrast, longitudinal survey data have the advantage of following a sample of individuals over a period of time, observing their demographics along with the evolution of their self-reported drug use behavior and other variables. In these circumstances, longitudinal survey data facilitates the analysis of heterogeneous effects among subgroups.

This study is similar to those that rely on econometric techniques to distinguish the gateway effects from observed heterogeneity using individual-level survey data. This class of research has generally found gateway effects to be statistically significant and has placed importance on controlling for unobservables (e.g., DeSimone 1998; Pudney 2003; Van Ours 2003; Melberg, Jones, and Bretteville-Jensen 2010; Bretteville-Jensen and Jacobi 2011; Deza 2015), except for Beenstock and Rahav (2002). Using the NLSY from the U.S., DeSimone (1998) estimated the effect of past marijuana use on current cocaine use with instrumental variable (IV) estimation and concluded that there was strong evidence for the gateway hypothesis. The author used two measures of state-level penalties for marijuana possession (i.e., maximum jail time and an indicator that no fines for first-time offenders) and two alcohol-related variables (i.e., beer tax and an indicator of parental alcohol problems) as IVs. However, it is questionable whether these IVs satisfied the exclusion restriction (i.e., these IVs influenced cocaine use only through marijuana use), which is a crucial assumption of the IV estimation but not empirically testable. Van Ours (2003) made use of a bivariate survival model to investigate the dynamics of marijuana and cocaine use in the Netherlands. He found limited, but significant gateway effects from first-time marijuana to first-time cocaine use, and this was mostly driven by unobserved heterogeneity. Pudney (2003) modeled the interdependency between multiple types of drug use in the UK and after considering unobserved heterogeneity, he found that the gateway effects from soft to hard drugs appeared to be modest, but statistically signif-

icant. Melberg, Jones, and Bretteville-Jensen (2010) used a bivariate survival model with shared frailty (i.e., unobserved characteristics) to examine how past marijuana initiation affected hard drug initiation in Norway. Utilizing a latent class approach (which assumes that each individual belongs to one of the subgroups or latent classes in the population), they identified two distinct groups of youth: a smaller group of youths who manifested gateway effects and a larger group of youths who demonstrated no gateway effects. The smaller group not only started consuming illicit drugs at a younger age but also reported more childhood problems with police, parents, friends, and school. Bretteville-Jensen and Jacobi (2011) employed a bivariate probit model and Bayesian estimation framework to analyze the effects and relative importance of previous marijuana use, proneness to drug use, and accessibility factors on hard drug initiation using 2006 Norwegian survey data. They found that these factors contributed to the observed higher hard drug use among marijuana users, with previous marijuana use having the largest effect. Deza (2015) modeled the intertemporal dependence of drug use (within and between drugs) using a dynamic discrete choice model of alcohol, marijuana, and hard drug use. She found statistically significant "stepping-stone" effects from soft to hard drugs and concluded that alcohol, marijuana, and hard drugs were complements in utility.[4] However, Beenstock and Rahav (2002), who applied various methods (e.g., two-stage logit, bivariate probit, survival analysis) to survey data from Israel, found gateway effects from cigarettes to marijuana, but not from marijuana to hard drugs. The authors acknowledged that their approach was somewhat *ad hoc* due to the absence of price data for marijuana and hard drugs and that their sample size may have insufficient power to detect gateway effects under their estimations.

[4]The author referred to the stepping-stone effect from drug i to drug j as the causal effect of the use of drug i in period t on the use of drug j in period $t+1$. Also, she referred to complements in utility in the sense that utility derived from consuming drug i and j together was higher than the summation of the utilities derived from consuming either drug alone.

3.3 Methodology

In this section, I constructed an empirical model that relates hard drug initiation to marijuana use, while accounting for unobserved heterogeneity to assess gateway effects.

For individual i in year t, D_{it} denotes an indicator of hard drug initiation, i.e., D_{it} equals 1 if hard drug initiation occurs and 0 otherwise. M_{it} denotes an indicator of marijuana use. I first specified a probit model associating latent hard drug initiation to marijuana use, as follows:

$$D_{it}^{*} = \alpha \cdot M_{it} + X_{it}'\beta + T_t + \eta_i^1 + \upsilon_{it}^1 \tag{3.1}$$

where D_{it}^{*} is latent hard drug initiation, which measures the propensity to initiate hard drugs. That is, $D_{it} = 1[D_{it}^{*} > 0]$, in which $1[\cdot]$ denotes the indicator function. The coefficient of interest, α, determines the gateway effects from marijuana to hard drugs. The sign of α indicates whether marijuana and hard drugs are complements ($\alpha > 0$), substitutes ($\alpha < 0$), or bear no relationship ($\alpha = 0$). X_{it} is a vector of controls that consists of pre-determined and time-varying observables (variables that can be measured). β is a vector of the coefficients. T_t is the year fixed effect. η_i^1 represents fixed unobservables (e.g., genetic predisposition, psychological problems) and υ_{it}^1, time-varying unobservables (e.g., hard drug prices, policy environment).

Since marijuana use (M_{it}) could have a mutual relationship with hard drug initiation via unobservables (η_i^1, υ_{it}^1) separately from any causal link, the estimated gateway effect ($\hat{\alpha}$) could be biased if Equation (3.1) was estimated in isolation. To address this endogeneity issue, I specified an auxiliary probit model for latent marijuana use, M_{it}^{*}, as follows:

$$M_{it}^{*} = X_{it}'\gamma + T_t + \eta_i^2 + \upsilon_{it}^2 \tag{3.2}$$

where the variables and coefficients are defined in a manner similar to Equation (3.1).

To control for endogeneity in Equation (3.1), I allowed η_i^1 and η_i^2 to be jointly normally distributed with mean 0 and covariance matrix Ω_η,

$$\begin{pmatrix} \eta_i^1 \\ \eta_i^2 \end{pmatrix} \sim \mathcal{N}\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \Omega_\eta = \begin{pmatrix} \sigma_1^2 & \sigma_1\sigma_2 \cdot \rho_\eta \\ \sigma_1\sigma_2 \cdot \rho_\eta & \sigma_2^2 \end{pmatrix} \right) \tag{3.3}$$

where σ_1 and σ_2 are the standard deviations of η_i^1 and η_i^2. ρ_η denotes the correlation between η_i^1 and η_i^2. Additionally, I allowed υ_{it}^1 and υ_{it}^2 to be jointly normally distributed with a mean of 0 and covariance matrix Ω_υ,

$$\begin{pmatrix} \upsilon_{it}^1 \\ \upsilon_{it}^2 \end{pmatrix} \sim \mathcal{N}\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \Omega_\upsilon = \begin{pmatrix} 1 & \rho_\upsilon \\ \rho_\upsilon & 1 \end{pmatrix} \right) \tag{3.4}$$

where υ_{it}^1 and υ_{it}^2 have a standard deviation of 1. ρ_υ is the correlation between υ_{it}^1 and υ_{it}^2.

Collectively, Equations (3.1)–(3.4) are referred to in the literature as a bivariate random-effects probit model (Roodman 2022).

3.4 Data and Variables

3.4.1 Sample Selection

The analyses were based on the NLSY97, an individual-level panel survey where information was collected from a nationally representative sample of 8,984 individuals born between 1980 and 1984 in the U.S. Surveys were conducted annually from 1997 to 2011 and biennially from 2011 onwards. NLSY97 started collecting information relating to individual's use of marijuana in 1997 and in respect of hard drug use in 1998. Hereafter let R stand for respondent and DLI stand for date of last interview. For example, NLSY97 started asking, "R ever use marijuana?" in 1997, "# days use marijuana in last 30 days?" in 1997, "Has R used marijuana since DLI?" in 1998, "R

ever use cocaine/hard drugs?" in 1998, and "Has R used cocaine/hard drugs since DLI?" in 1999.

This study ran from 1999 to 2015, which was the last year that the NLSY97 asked questions on marijuana and hard drug use. The study period begins in 1999 as it was the first year the NLSY97 posed the question, "Has R used cocaine/hard drugs since DLI?", which is used to derive the dependent variable (hard drug initiation) in later analyses. To fully investigate the self-reported drug use history of the participants, I selected a sample of 5,821 individuals who had never reported using marijuana or hard drugs before 1999. The sample selection is based on three NLSY97 questions: "R ever use marijuana?" in 1997, "Has R used marijuana since DLI?" in 1998, and "R ever use cocaine/hard drugs?" in 1998. The key and control variables along with the corresponding NLSY97 questions are listed in Table 3.1. Table 3.2 provides the summary statistics for the individuals in the sample. In line with the total population, 51% are female. 28% are Black, and 22% are Hispanic, which are much higher than the corresponding shares in the entire population due to the NLSY97's oversampling of African Americans and Hispanics. (The NLSY97 also provides a nationally representative but smaller sample.) In comparison, the percentages of the Black and Hispanic populations in 1997 were 12.8% and 11.1%, respectively. Prior to 1997, 26% were arrested, 28% had drunk alcohol, and 23% had smoked cigarettes. In 1997, 53% lived with both biological parents, about 25% of peers used illicit drugs on average, and 90% were religious. During the study period, 16% used hard drugs, and 45% used marijuana. Hazards of hard drug initiation were averaged at 2% in the study period. Finally, consistent with the gateway hypothesis, the average starting age for marijuana usage (19.68 years) was about one year lower than that for hard drugs (20.66 years).

Following Rabe-Hesketh and Skrondal (2008) and Melberg, Jones, and Bretteville-Jensen (2010), I constructed an indicator of hard drug initiation, D_{it}, which is used

in estimations as the dependent variable. For those who did not initiate hard drugs during the study period, D_{it} was equal to zero throughout the study period. In contrast, for those who started using hard drugs during the study period, D_{it} equals 1 for the year of initiation and 0 before that year. After the first instance of hard drug use, the individuals were no longer at risk of initiating hard drugs and were thus removed from the sample for subsequent years. Moreover, the NLSY97 indicated missing data with five negative values: (-1) refusal, (-2) don't know, (-3) invalid skip, (-4) valid skip, and (-5) noninterview. This study treats data with negative values as missing for all variables except for marital status. For marital status, teenagers with a value of -4 (valid skip) are assumed to be never married. As a result, the analysis panel data consisted of 61,799 observations at the individual and year levels.

3.4.2 Descriptive Survival Analysis

Malone et al. (2010) described the gateway hypothesis in terms of necessary conditions regarding risks, that is, the conditional probability of an event, given that the event has not yet occurred. Mathematically, hazard at year t can be expressed as $Pr[T = t|T \geq t]$, where $Pr[\cdot]$ is the probability function, and T is the year that the event under investigation occurs. A key necessary condition posits that compared to individuals who have never used marijuana and hard drugs, individuals who have used marijuana are more at risk of starting to use hard drugs.

To examine whether this condition is consistent with the empirical hazards of hard drug initiation, following the method of Rabe-Hesketh and Skrondal (2008), I assessed the discrete-time hazards as predicted probabilities by estimating a logistic regression:

$$\text{logit}\Big(Pr(D_{it} = 1|d)\Big) = \sum\nolimits_{s=0}^{T_i - T_i^0} \alpha_s \cdot d_s \tag{3.5}$$

where D_{it} is an indicator of hard drug initiation in year t for individual i, which

was constructed in the previous section. For those who started using hard drugs during the study period, T_i is the year of initiation. For those who did not initiate hard drugs during the study period, T_i was the last year before being right-censored. Here, right-censoring occurred in either of the following two scenarios. First, hard drug initiation did not occur before the end of the study period. Second, the subject ceased to be at risk of initiating hard drugs before the end of the study period due to dropping out or death. T_i^0 is the first year at risk of initiating hard drugs. s indexes the number of years at risk in year t, that is, $s = t - T_i^0$. d is a vector containing all dummy variables d_s, where s ranges from 0 to $T_i - T_i^0$. α's are coefficients.

Figure 3.1 shows the estimated hazards of hard drug initiation against years at risk of initiating hard drugs versus those conditional on having used marijuana. In estimating the hazards of hard drug initiation conditional on having used marijuana, I drop data before the year of initiating marijuana for those who used marijuana. In this figure, the x-axis represents the number of years since the first year at risk and the y-axis represents the hazard value. For those who never used marijuana and hard drugs, the first year at risk of initiating hard drugs is set at 1999, the first year of the study period. For those who had used marijuana, the first year at risk is set as the year they began using marijuana. Except for one year at risk, the risks for individuals who had used marijuana before were higher than the risks for all individuals. This observation raises the question: was this pattern driven by the causal effect of marijuana use on increasing the risk of initiating hard drugs or other factors? The main objective of this study is to determine the extent to which this pattern is due to gateway effects.

3.5 Results

3.5.1 Baseline Estimates

In this section, as our primary interest is the gateway effect from marijuana to hard drugs, only the estimates for the model of hard drug initiation, i.e., Equation (3.1) in Section 3.3, are presented and discussed.

Table 3.3 shows the estimates from various specifications in which standard errors are clustered at the individual level. In columns (1)–(3), controlling for pre-determined controls (listed in Table 3.1), I estimated Equation (3.1) in Section 3.3 alone with random-effects logit, random-effects complementary log-log, and random-effects probit, respectively. The semi-elasticity of hazards of hard drug initiation with respect to marijuana use was estimated at 2.69, 2.67, and 2.84, respectively.[5] Thus, the semi-elasticity estimate is robust across different model types. In column (4), I jointly estimated Equations (3.1) and (3.2) in Section 3.3, allowing the correlation of time-varying but not fixed unobservables in both equations. In column (5), I further allowed fixed unobservables in both equations to correlate. The column (5) estimate (0.49) was statistically significant and roughly half of the column (4) estimate (0.96), suggesting that fixed unobservables partly explain why marijuana use tends to precede hard drug use. Based on column (5), I excluded the pre-determined controls in column (6). The column (6) estimates were similar to the column (5) estimates, but only marginally significant, suggesting that the bivariate random-effect model is fairly robust with different sets of pre-determined controls. Nevertheless, controlling for pre-determined controls is preferred as it is more efficient. Finally, based on column (5), column (7) includes time-varying controls (listed in Table 3.1). Column (7) estimates were similar to those in column (5) and were statistically significant, suggesting that the bivariate random-effect model is robust with different sets of time-

[5]The semi-elasticity of y with respect to x is evaluated as the average of $\frac{\partial \hat{y}}{\partial x} \cdot \frac{1}{\hat{y}}$, where $\hat{y}$ is the expected value of y.

varying controls as well. However, missing values in time-varying controls reduce the size of the analysis sample. In addition, certain time-varying controls (e.g., employment status, marital status, educational attainment, health status) could be impacted by marijuana use and thus serve as channels by which marijuana use affects hard drug initiation. Therefore, it is preferable not to control for time-varying controls. Accordingly, the specification in column (5) is preferred and is hereafter referred to as the preferred specification. Also, the estimates in column (5) are hereafter referred to as the baseline estimates.

Taken together, the unobserved heterogeneity is partly attributable to the observed sequence of drug use from marijuana to hard drugs. However, after accounting for unobserved heterogeneity, the estimate of the gateway effect from marijuana to hard drugs is still statistically significant and robust with various sets of controls. The baseline semi-elasticity estimate indicates that marijuana use increases the risks of starting to use hard drugs by 1.39 times, which is comparable to the estimate indicated by Melberg, Jones, and Bretteville-Jensen (2010), who found that the risk of hard drug initiation more than doubled after marijuana initiation.

3.5.2 Robustness Checks

On the basis of the preferred specification, I conducted three robustness checks as follows. First, the semi-elasticity estimate remains statistically significant when standard errors are clustered at the region level (instead of the individual level). Second, the NLSY97 oversamples African Americans and Hispanics and provides a nationally representative subsample. After I removed individuals not in the nationally representative subsample from the analysis sample, with the remaining 45,016 observations, the estimate was 1.73 and statistically significant, which is comparable to the semi-elasticity estimate in the preferred specification (1.39). Finally, another concern is that the baseline estimates were driven by the right censoring due to participants

who never used hard drugs dropping out of the survey before the end of the study period. To investigate this issue, I removed individuals involved in that type of right-censoring. 4,747 out of 5,821 (81.5%) individuals and 53,573 out of 61,799 (86.7%) data points remain. With the remaining sample, the semi-elasticity estimate of the preferred specification is 1.40 and statistically significant, which is very close to the baseline semi-elasticity estimate (1.39). This indicates that the baseline estimates were not driven by non-hard drug users who exited the survey prematurely because even in the sample free from that type of right-censoring, the estimate is essentially unchanged.

3.5.3 Heterogeneous Effects

Experiences with marijuana are not universally pleasant. As such, marijuana use does not affect all individuals equally, offering scope for heterogeneity among subgroups. Instead of relaxation and euphoria, some people experience anxiety, fear, distrust, or panic (NIH 2020). Heterogeneity analyses can also help inform about potential mechanisms behind the observed gateway effects, such as early marijuana initiation and the frequency of marijuana use, which could be informative for public policy purposes.

To examine the heterogeneous effects, I included interaction terms in addition to the preferred specification. The results can be viewed in Table 3.4. In column (1), I explore whether gateway effects differ by gender. The estimate of the interaction between marijuana use and the indicator of being female is not statistically significant, suggesting that the gateway effects are similar whether the user is male or female. In column (2), I investigate whether gateway effects vary by ethnic group. While African Americans have significantly lower gateway effects compared to the reference group (non-Black/non-Hispanic), the gateway effects are similar across Hispanics, mixed race (non-Hispanic), and non-Black/non-Hispanic. Previous studies conducted (e.g.,

Bretteville-Jensen and Jacobi 2011) have suggested that individuals who started using marijuana during the development of their brain may be more vulnerable to the influence of marijuana. In column (3), to examine whether the gateway effects are more pronounced among people who started using marijuana early in life, I included the interaction between marijuana use and an indicator of "early marijuana users," defined as those who first used marijuana before the age of 18 years old. Consistent with previous literature, this study indicated that "early marijuana users" have greater gateway effects. To examine whether the gateway effects are more pronounced among people who used marijuana more frequently, column (4) includes the interaction between marijuana use and an indicator of "intense marijuana users," defined as those who used marijuana for more than three days in the last 30 days at the time of the survey.[6] I found that "intense marijuana users" have greater gateway effects. Finally, column (5) estimates suggest that gateway effects decline with age, that is, earlier marijuana use has a greater impact on hard drug initiation, which is consistent with the findings of Deza (2015). Figure 3.2 shows semi-elasticity estimates in different groups.

Although the onset age of and frequency of marijuana use may be endogenous (driven by unobserved factors that induce the use of hard drugs), the heterogeneity effects are informative in suggesting that early onset and frequency of marijuana use may be, at least, partly to blame for the rise of gateway effects.

3.6 Limitations

This study has several limitations. First, self-reported drug use may not accurately measure actual drug use as users may misreport and have difficulty remembering. Although this may be concerning with all survey data, it could be more so in this

[6]The indicator of "intense marijuana users" is derived from the NLSY97 question "# days use marijuana in last 30 days?"

context. NLSY97 interviews were conducted either face-to-face or via telephone. Despite measures to guarantee confidentiality, NLSY participants may have under-reported their drug use due to social stigma and the illegality of illicit drug use (DeSimone 1998). The participants were equally likely to have exaggerated their actual drug use (Bretteville-Jensen and Jacobi 2011).

Second, I assumed that the bivariate random-effects probit model accurately describes how marijuana use and hard drug initiation are determined, in which the error terms are bivariate normally distributed. However, if the bivariate normal distributions did not sufficiently account for unobserved heterogeneity, then the estimate could be biased and driven by certain omitted variables. For example, drug prices could potentially be important omitted variables, as previous studies have suggested that prices were important in explaining drug use (e.g., Van Ours and Williams 2007; Melberg, Jones, and Bretteville-Jensen 2010).

Finally, the nature of unobserved heterogeneity, which is found to partly account for the observed drug use sequence from marijuana to hard drugs, is unknown. In addition, this study does not discuss the mechanisms underlying the gateway effects, except that the age of onset and frequency of marijuana use may play a role. Understanding these mechanisms is crucial for policy purposes, as different mechanisms have different or even opposite policy implications. For example, as was noted in the Introduction section, if gateway effects arise mainly because marijuana "primes" the brains of users, then legalizing marijuana would stimulate hard drug use. However, if gateway effects arise mainly through interactions with drug dealers, then legalizing marijuana, which may aid in separating marijuana users from the hard drug market, may prove effective in curbing hard drug use.

3.7 Conclusion

The use of hard drugs, such as cocaine and heroin, imposes grave health risks to users, thus raising major public health concerns. Empirical studies have consistently found that marijuana use precedes hard drug usage. These findings led to the gateway hypothesis, which posits that marijuana use causes people to progress to experiment with and use hard drugs. As federal and state governments are considering legalizing marijuana, which may stimulate marijuana use, it is important to investigate the validity of the gateway hypothesis to inform the debate on legalizing marijuana.

The issue addressed in this study is whether marijuana use hastens hard drug initiation, consistent with the conventional notion of gateway effects. The major challenge is how to disentangle gateway effects from unobserved heterogeneity, which may also lead to the progression from marijuana to hard drugs, thus confounding correlation for causation. This study makes a methodological contribution to the current literature by proposing a bivariate survival panel model to isolate gateway effects from unobserved heterogeneity in explaining the initiation of hard drug use. The same framework can be applied to examine broader issues, such as how drug use impacts educational attainment, employment status, or crime.

In the analysis sample taken from the NLSY97, I found that unobserved heterogeneity partly explains the sequence of drug use from marijuana to hard drugs. Once accounting for unobserved heterogeneity, estimated gateway effects from marijuana to hard drugs substantially decrease but remain significant. This shows the importance of adjusting for unobserved heterogeneity, which, if left unadjusted, would be confounded as the actual gateway effects. With the preferred specification, marijuana use significantly increased the risk of hard drug initiation by 1.39 times, which is similar to the estimate by Melberg, Jones, and Bretteville-Jensen (2010). The estimate is qualitatively robust with various sets of covariates. Furthermore, gateway effects are more pronounced among those who first used marijuana before the age of 18 years,

as well as those who used marijuana more frequently, which speaks to the possible mechanisms underlying the gateway effects. Additionally, the effects were less pronounced in African Americans and were attenuated with age. These findings add to the literature in better understanding the mechanisms behind the gateway effects, suggesting that the age of onset and frequency of marijuana use may underpin the effects. Consequently, it may be effective in curbing hard drug use through policy measures such as (1) launching information campaigns on marijuana's adverse effects, (2) early identification of youths experiencing drug problems and providing help to prevent them from starting to use hard drugs, and (3) increasing the legal age of marijuana use and limiting the potency of marijuana in marijuana-legalized states.

Tables

Table 3.1: Key and Control Variables Along With Their Corresponding NLSY97 Questions

Key variables	NLSY97 question
Marijuana use	Has R used marijuana since DLI?
Hard drugs use	Has R used cocaine/hard drugs since DLI?
Pre-determined controls	
Female	R's gender
Race and ethnicity	Race and ethnicity
Black	
Hispanic	
Mixed race (non-Hispanic)	
Ever arrested (in 1997)	Total number of arrests
Ever drank alcohol (in 1997)	R ever drink alcohol?
Ever smoked (in 1997)	R ever smoked?
% Peers using illicit drugs (in 1997)	Percent peers use illegal drugs
Lived with both biological parents (in 1997)	Does R live with both biological parents?
Religious (in 1997)	What is R's current religious preference?
Time-varying controls	
Receive income from job	R receive income from job in past year?
Marital status	R's marital/cohabit status
Single	
Married	
Household size	Household size
Educational attainment	Highest degree received
Less than high school	
High school	
Some college	
Rural	Current residence in urban or rural area
General health	How is R's general health?
Region of residence	Census region of residence
Northeast	
North Central	
South	
Drank alcohol	Has R drank since DLI?
Smoked cigarettes	Has R smoked since DLI?

Notes: R stands for respondent; DLI stands for date of last interview. "% Peers using illicit drugs" is a categorical variable with five categories: (1) less than 10%, (2) about 25%, (3) about 50%, (4) about 75%, and (5) more than 90%. "General health" is a categorical variable with five categories: (1) excellent, (2) very good, (3) good, (4) fair, and (5) poor. "Household size" is the number of family members living together. The other variables are indicators of the indicated event or status.

Table 3.2: Summary Statistics

	mean	SD
Birth year	1982.22	1.38
% Female	0.51	0.50
% Black	0.28	0.45
% Hispanic	0.22	0.41
% Mixed race (non-Hispanic)	0.01	0.09
% Non-black/non-Hispanic	0.50	0.50
% Arrested (before 1997)	0.26	0.44
% Drank alcohol (before 1997)	0.28	0.45
% Smoked cigarettes (before 1997)	0.23	0.42
% Lived with both biological parents (in 1997)	0.53	0.50
% Peers using illicit drugs (in 1997)	1.97	1.16
% Religious (in 1997)	0.90	0.30
% Used hard drugs (during study period)	0.16	0.37
% Used marijuana (during study period)	0.45	0.50
Hazards of hard drug initiation	0.02	0.12
Age of first hard drug use (in those who used hard drugs)	20.66	3.87
Age of first marijuana use (in those who used marijuana)	19.68	3.73
N	5,821	

Notes: SD represents standard deviation. "% Peers using illicit drugs" is a categorical variable with five categories: (1) less than 10%, (2) about 25%, (3) about 50%, (4) about 75%, and (5) more than 90%.

Table 3.3: Baseline Estimates

	Hazards of hard drug initiation						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Model	logit	cloglog	probit	probit	probit	probit	probit
Marijuana use	2.76***	2.71***	1.17***	0.96	0.49**	0.45*	0.54***
	(0.082)	(0.078)	(0.047)	(1.03)	(0.29)	(0.25)	(0.17)
$\hat{\rho}_v$				0.11	0.24	0.27	0.19
$\hat{\sigma}_1$					0.55	0.71	0.16
$\hat{\sigma}_2$					1.27	1.39	1.04
$\hat{\rho}_\eta$					0.77	0.78	1.00
Semi-elasticity	2.69	2.67	2.84	2.40	1.39	1.29	1.48
Pre-determined controls?	Yes	Yes	Yes	Yes	Yes	No	Yes
Time-varying controls?	No	No	No	No	No	No	Yes
N	61,799	61,799	61,799	61,799	61,799	61,799	57,221

Notes: Pre-determined and time-varying controls are listed in Table 3.1. The estimates of the covariates are omitted for clarity. Standard errors are clustered at the individual level and reported in parentheses. *$p < 0.1$ ** $p < 0.05$ *** $p < 0.01$.

Table 3.4: Heterogeneous Effects

	Hazards of hard drug initiation				
	(1)	(2)	(3)	(4)	(5)
Marijuana use × female	0.04				
	(0.067)				
Marijuana use × Black		-0.29***			
		(0.083)			
Marijuana use × Hispanic		-0.062			
		(0.084)			
Marijuana use × mixed race		0.56			
		(0.41)			
Marijuana use × early user			0.11**		
			(0.052)		
Marijuana use × intense user				0.20***	
				(0.050)	
Marijuana use × age					-0.018**
					(0.0079)
Marijuana use	0.51*	0.59**	0.41**	0.35**	0.89***
	(0.26)	(0.24)	(0.18)	(0.17)	(0.28)
Pre-determined controls?	Yes	Yes	Yes	Yes	Yes
N	61,799	61,799	61,799	61,799	61,799

Notes: All models included pre-determined and time-varying controls, as listed in Table 3.1. The estimates of the covariates are omitted for clarity. Standard errors are clustered at the individual level and reported in parentheses. "Early user" is an indicator of whether the subject first used marijuana before age 18. "Intense user" is an indicator of whether the subject used marijuana for more than three days in the last 30 days at the time of the survey. *$p < 0.1$ ** $p < 0.05$ *** $p < 0.01$.

Figures

Figure 3.1: Discrete-Time Hazards of Hard Drug Initiation

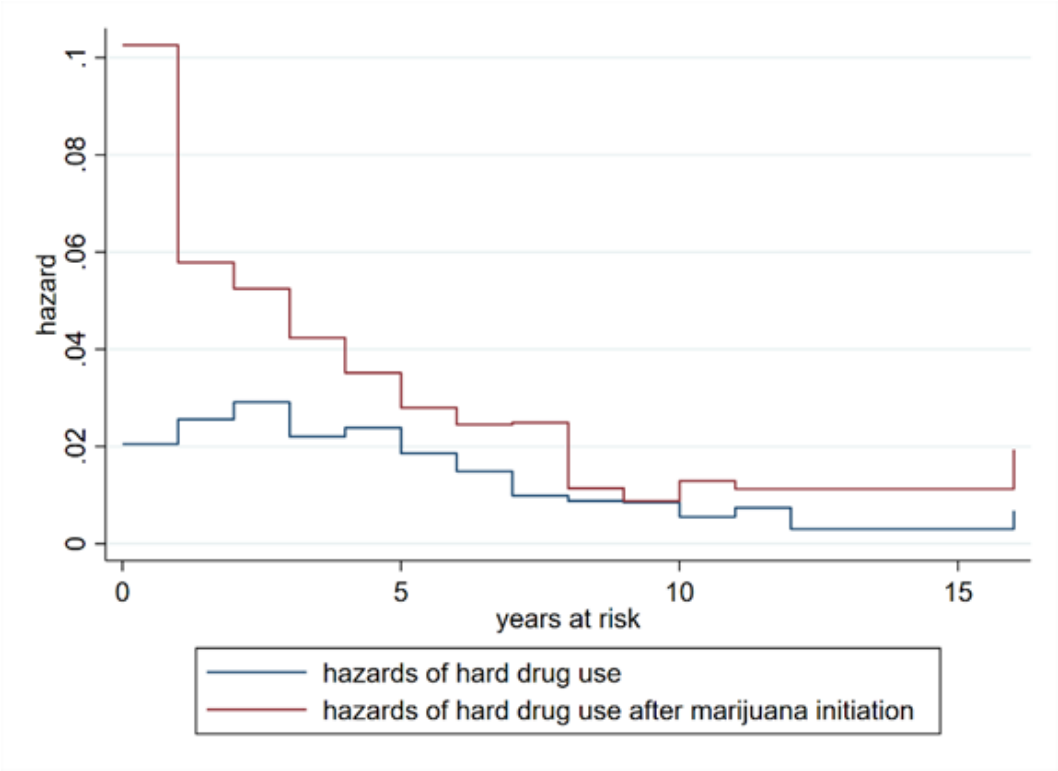
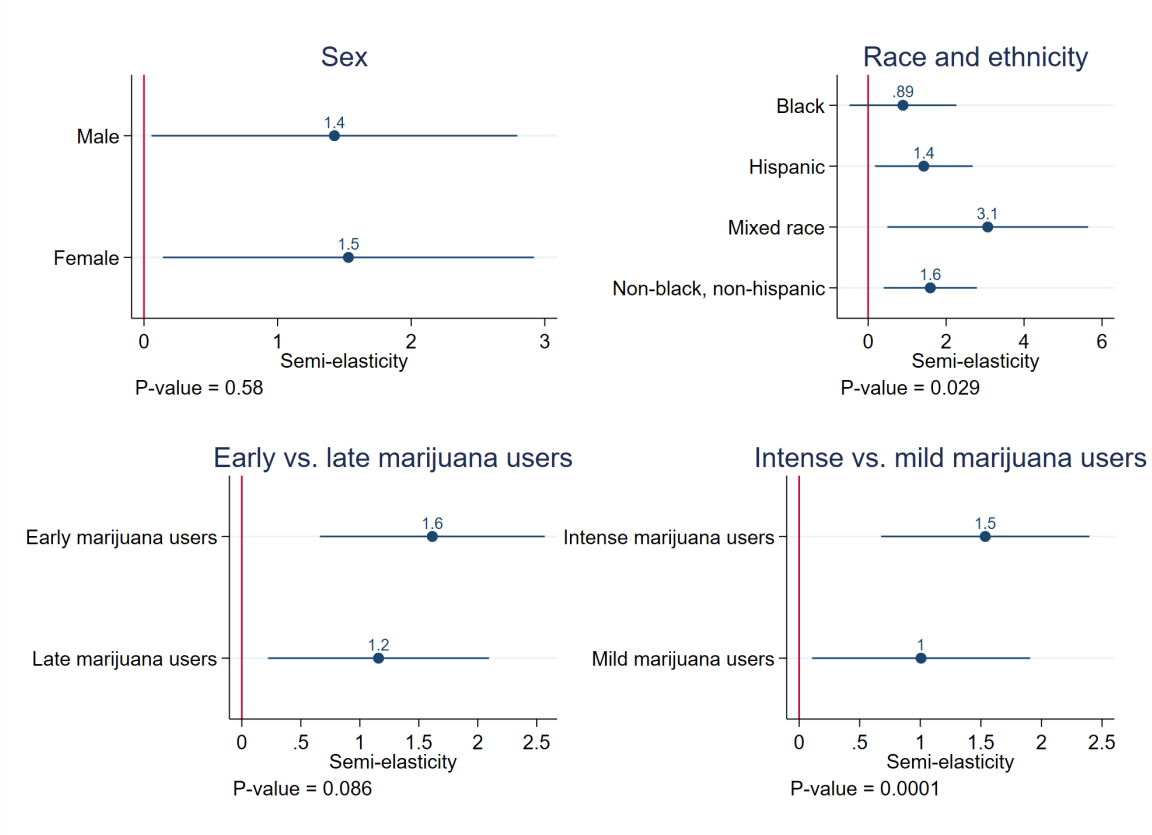


Figure 3.2: Semi-elasticity Estimates in Different Groups



Notes: Dots show point estimates and horizontal bars show 95% confidence intervals using standard errors clustered at the individual level. In each subplot, the p-value indicates whether the estimates are equal.

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Biography

Jin Xing was born and raised in Dalian, China. He graduated from Beijing Jiaotong University with a bachelor's degree in applied physics in 2011. In the fall of the same year, he came to the United States to study at Case Western Reserve University. After obtaining an M.S. in electrical engineering in 2014, he worked as an operations analyst in New York. Through reading economics textbooks in his leisure time, he became interested in economics and determined to study it systematically should an opportunity presents itself. Such an opportunity occurred one year later when Drexel University accepted his application for M.S. in economics. Following his graduation in 2017, he started the journey of pursuing a Ph.D. degree in economics at Tulane University in the fall of that year. His research fields are health economics and applied microeconomics.