Mass Incarceration and the War on Drugs*

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Abstract

US incarceration rates quintupled from the early 1970s to the present, leading to the US becoming the most incarcerated OECD country in the world. A driving cause behind this growth was a nationwide shift to more punitive criminal justice policy, particularly with respect to drug related crimes. This movement has since been characterized as the "war on drugs." In this manuscript, we investigate the impact of rising incarceration rates on drug use and drug markets by exploiting a natural experiment in the Texas penitentiary system. In 1993, Texas made massive investments into its prison infrastructure which led to an over doubling of the state's prison capacity. The effect was that Texas's incarceration rates more than doubled, due in large part to declining paroles. We use this event to study the effect that mass incarceration had on drug markets. We find no effect on drug arrests, drug prices or drug purity. We also find no effect on self-referred cocaine or heroin treatment admissions. However, we do find large negative effects on criminal justice referrals into treatment for cocaine and heroin, suggesting that mass incarceration reduces drug use in the population. Furthermore, our results indicate that this decline is driven by incapacitation effects as opposed to deterrence effects.

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1 Introduction

The United States is the most highly incarcerated OECD country in the world. In 2013, the US Bureau of Justice Statistics estimated that there were more than 2.2 million adults incarcerated in state or federal prisons or local jails. This result is largely derivative of a monumental growth in incarceration rates over the past half-century. From the early 1970s to 2009, incarceration and prison admission rates over doubled in size. This change comes in spite of the fact that crime and arrest rates were constant or falling for more than a decade (Neal and Rick, 2014). The reason for such a large increase in the US prison population, while complex, is at least partially due to tightening drug policy.

Over the final decades of the 20th century, United States policymakers enacted increasingly stringent drug enforcement policy in what has been characterized as the "war on drugs". As a result, the number of annual drug arrests grew from 580,900 in 1980 to 1,579,600 in 2000 (FBI, 2017). Over this same time period, the Drug Enforcement Agency's annual budget grew from $207 million to $1,587 million, signalling a substantial growth in drug enforcement spending (DEA, 2017). However, these costs reflect only a small portion of the total cost that society incurs from drug markets, which was estimated to be more than $193 billion in 2007, including costs for criminal justice, health, and productivity losses among other things (NDIC, 2011). The magnitude of this considerable burden and the increasingly prominent role of drug enforcement in criminal justice policy raises the question: What effect, if any, has society generated in return for its war on drugs?

In this paper, we attempt to answer that question by exploiting a natural experiment in Texas in which the prison population evolved between two steady states. The first steady state may be characterized by a heavy reliance on discretionary parole releases in order to alleviate overcrowding pressures. The second steady state may be characterized by a substitution from paroles to incarceration under the context of ample prison capacity. This evolution is derivative of a massive investment in operational prison capacity that led to Texas doubling its number of prison beds from 1993-1995. This was followed by a massive increase in incarceration rates. We used this event to study the impact that

mass incarceration had on drug-related outcomes: drug arrests, illegal drug prices, and drug treatment admissions.

Our results are interesting. First, we found no evidence that an increase in incarceration rates had any effect on cocaine or heroin prices, thus suggesting that an increase in effective sentencing did not deter drug use via higher drug prices. This finding stands in contrast to Kuziemko and Levitt (2004) who associate higher incarceration rates with higher cocaine prices. We also found no evidence that incarceration reduced the number of self-admissions into treatment for heroin or cocaine. However, we did find a difference in criminal justice referrals into treatment for heroin and cocaine. Criminal justice referral channels for both drugs decrease when incarceration rates rise relative to estimated counterfactuals. We suggest that higher rates of incarceration have shifted people with substance abuse problems out of treatment and into imprisonment, rather than deterring general population drug use.

This article is organized as follows: The next section reviews the endogeneity problems associated with studying crime and proposes a natural experiment solution. The third section introduces the reader to the data we use in our analysis, as well as the empirical models. The fourth section discusses our results. The fifth section concludes.

2 Conceptual Framework, Theory, Literature Discussion

Incarceration could theoretically reduce individual-level drug use through a combination of deterrence and higher drug prices. These potential mechanisms are staples in arguments used to justify the war on drugs (MacCoun and Reuter, 2001). One study supporting this theory is Kuziemko and Levitt (2004) who find that cocaine prices rose by 5-15% as a consequence of more severe punishments for drug offenses. Using these estimates and previously estimated elasticities, they speculate that higher incarceration rates likely reduced drug consumption. However, evidence for an actual response in population drug use is thin. Friedman, Cleland and Cooper (2011) test whether higher arrest rates lower injection drug use, but fail to find any evidence supporting this hypothesis.

Another mechanism through which incarceration may reduce drug use is incapacita-

tion. This concept posits that the incarceration of drug users will reduce population drug consumption levels merely by moving users into an environment where they do not have access to illicit substances. In their analysis of the 2006 Italian Clemency Bill, Buonanno and Raphael (2013) found that, on average, the imprisonment of one inmate prevents 0.435 drug offenses. If this is true, given the monetary costs and documented negative effects of incarceration on violent behavior and labor market opportunities (Mueller-Smith, 2015), these incapacitation effects are likely much more costly than deterrence.

Estimating the effect of incarceration on drug use and drug markets is complicated due to the endogeneity of incarceration with crime. Incarceration is a function of crime, and thus changes in incarceration rates may themself be a response to changes in crime rates. This presents an econometric problem of reverse causation and omitted variables. To circumvent this problem, we exploit an important event in the history of Texas imprisonment. In 1980, the Texas Department of Corrections (TDC) lost the civil action lawsuit *Ruiz v. Estelle* in which TDC prison conditions and management practices were deemed to be unconstitutional. Of particular relevance to this study was Texas's practice of housing inmates in overcrowded prison cells. As part of the final judgment, TDC was thereafter required to adhere to fixed capacity constraints as determined by their operational prison capacity (H-78-987-CA, 1985). To ensure compliance, TDC was placed under court supervision until 2003. This effectively transformed the Texas short-run prison capacity from flexible to fixed, and, in the ensuing years, this event would place tremendous stress on the Texas prison system (Perkinson, 2010).

Within the context of the *Ruiz vs. Estelle* decision, the state could manage the growing demands on its prison infrastructure by either excessively paroling criminals or expanding its prison capacity. As long-term investment in prison expansion would be delayed and erratic, Texas was forced to rely on paroles for short-term capacity easing in the years immediately following the enforcement of the overcrowding stipulations.[1] Figure 1 reveals this dependency. It may be seen that the Texas parole rate began to rise

[1]Following the *Ruiz v. Estelle* decision, TDC entered into a series of negotiations to determine the stipulations that TDC management would be required to adhere while under court supervision. The stipulations governing prison overcrowding constraints was determined in the 1985 negotiation *Ruiz v. Procunier*.

in 1985, reaching its peak in 1990. Initial prison construction started in the late 1980s under Governor Bill Clements, who managed to expand the state's operational capacity. However, the Clements prison expansion was seemingly insufficient, and it would not be until Ann Richards entered the governor's office that satisfactory investments in prison capacity were made. Under Governor Richards, Texas would approve a billion dollar prison construction project in 1993 that over doubled the state's count of prison beds (Perkinson, 2010). Figure 2 shows the annual level of prison beds in Texas for 1983-2004 (left axis and solid line) as well as the year-to-year percentage change (right axis and dashed line). While the Clements expansion increased capacity slightly in the late 1980s, this growth was dwarfed by the 1993 Richards expansion, as prison capacity grew from just under 60,000 beds to 130,000 beds in only two years.

The effect of the Richards prison expansion was dramatic. Figure 1 shows the parole, prison admission and prison release rates for the years 1980 to 2003. The reference line designates 1993, the year of the Richards prison expansion. It may be seen that the parole rate began to decline following the Clements build out in the late 1980s, though interestingly paroles continue to constitute the majority of total releases until 1993. As well, admissions and releases begin to diverge following the Clements construction, however this gap is largest in the years during the Richards prison expansion. This led to a dramatic increase in the incarceration rate as shown in Figure 3, which presents the time series for total incarcerated per 100,000 population for both Texas and the average of all other states. In the two years following the Richards expansion, the Texas incarceration grew by over 200 individuals per 100,000 state population. Such a substantial increase in incarceration provides us with a unique opportunity to better understand the effect that increases in incarceration have on the drug markets, as well as on drug use and drug treatment admissions.

The details above may be represented by the theoretical framework derived in Raphael and Stoll (2009), which shows that the steady-state rates for incarcerated and nonincarcerated individuals may be expressed by the following function:

$$Inc_t \approx \frac{c_t p_t}{c_t p_t + \theta_t} \tag{1}$$

$$Noninc_t \approx \frac{\theta_t}{c_t p_t + \theta_t} \tag{2}$$

where t indexes time periods, c is the number of offenses, p is the probability of conviction (for an individual who commits a crime), and θ is the release rate. This equation provides the means for understanding why incarceration rates fluctuate over the period of our study. Admissions and arrests are the product cp and releases and paroles are θ. However, according to Raphael and Stoll (2009), cp is very small in practice. Furthermore, they find that $\frac{1}{\theta}$ is approximately equal to the expected time served for an offender. Thus, Equation (1) may be approximated by

$$Inc_t \approx E(T_t) c_t p_t \tag{3}$$

where c and p are as they are in Equations (1) and (2) and $E(T)$ indicates the expected time served for an incarcerated offender.

Equation (3) offers several insights. First off, it highlights the problem of reverse causation. The deterrence and incapacitation effects associated with incarceration would be expected to be deterministic the parameter c; however, it is also clear that c is simultaneously deterministic of incarceration rates. Thus, unbiased estimation of the effect of incarceration on crime requires one to first identify a shock to either p or θ that is unrelated to c. In this paper, we posit that the 1993 Richards prison expansion was such a shock. Our reasoning is that this expansion was primarily a response to the ruling and subsequent overcrowding stipulations of *Ruiz v. Estelle*, and the ensuing changes in incarceration rates are driven by the Texas parole boards' manipulation of θ. As a result, Texas evolved between two steady states, as depicted in Figure 1. The first steady state exists approximately from 1983 to 1987, and may be characterized as being "artificial" since Texas's excessive paroling inflated total prison releases to roughly equate to total prison admissions. The second steady state appears to arise in the year 2000, and may be characterized as being "natural" since paroles do not constitute nearly as large of a proportion of total releases as observed under the "artifical" steady state. With this understanding in mind, we may use this experiment to study how drug markets respond to

exogenous shocks to incarceration rates.

3 Data

Our paper utilizes data from seven sources: prison measures from the National Prison Statistics (NPS); male drug arrests recorded in the Summary Uniform Crime Reports Part II offenses database (UCR); sworn officer employment counts from the Law Enforcement Officers Killed and Assaulted dataset (LEOKA); drug treatment admission counts in the Treatment Episode Data Set (TEDS); drug prices and purity measures from undercover purchases recorded in the Drug Enforcement Agency's System to Retrieve Information from Drug Evidence (STRIDE); state level covariates from the Current Population Survey (CPS); and state population sizes estimated by the Center for Disease Control's Surveillance, Epidemiology, and End Results Program (SEER).

Annual counts of state prison admissions, prison releases, incarcerated persons, and discretionary paroles are collected in the National Prison Statistics (NPS) database for every state from 1978 to 2003. Prison admissions, prison releases, and discretionary paroles are constructed by summing together the respective male and female measures in order to create respective total-count measures. The NPS has several measures of incarceration divided across race and gender. We constructed our incarceration counts by summing together the male and female counts of their "total race" incarceration measure, which encompasses all individuals held within state or federal prisons within the state's borders. SEER annual state population estimates are used to transform each measure into annual rates per 100,000 state population. These measures were used in the first stage of our analysis to identify the exact mechanism through which the Texas prison experiment impacts drug markets.

We collected monthly local law enforcement agency drug arrests counts from the Uniform Crime Reporting Part II (UCR) data set for 1985 to 2003. We aggregated male drug arrests to the state level on an annual basis. A rate per 100,000 state population measure was then constructed by dividing counts of male drug arrests by respective SEER state annual population estimates. The male drug arrest rate serves as a reflection of both law

enforcement utilization and the abundance of drug use. Consequently, the drug arrest rate is not a perfect measure of either factor; however, it provides insight into the mechanism through which the Texas prison expansion affected drug markets.

We gathered monthly counts of male and female employed sworn officers in local law enforcement agencies from the Law Enforcement Officers Killed and Assaulted Program (LEOKA). We excluded agencies that were not present over the full time period of the panel and aggregate data to the state level on an annual basis for the years 1960 to 2003. We also summed male and female officer employment counts for the year 1971 to 2003 (only total counts are provided for 1960 to 1970). We then transformed the data to be per 100,000 state population using SEER state population estimates. We examined this measure as an indicator of whether Texas law enforcement had altered its behavior in response to the prison expansion.

The Treatment Episode Data Set (TEDS) tracks all monthly admissions into federally funded rehabilitative centers. We collected data on cocaine and heroin admittances for 1992 to 2003 and aggregated the measures to the annual state level.[2] We constructed three different counts of cocaine and heroin admittances: total admittances, self-admittances, and criminal justice referrals. Since TEDS requires patients to list their primary, secondary, and tertiary problem substances, we constructed measures of "total admissions" for both heroin and cocaine (including crack cocaine) by counting all clients who listed heroin or cocaine as any problem substance. We constructed two additional measures by tracing the treatment admission pathways for cocaine and heroin users. TEDS defines "self-admissions" as clients who are referred to treatment by themselves, family members, a friend, or any other individual who does not fall under the umbrellas of criminal justice, school, health care, employer, communal, or religious organizations. "Criminal justice referrals" are defined as clients who are referred to treatment by any police official, judge, prosecutor, probation officer, or other person affiliated with a federal, state, or county judicial system. We aggregated each measure to the state level annually and transformed the measures into rates per 100,000 with the use of SEER state population estimates. We subsequently logged the values so that our estimates present partial elasticities. We

[2]Unfortunately, data on admittances are not available prior to 1992.

examined the response of all six variables to identify how drug usage responded to the natural experiment and further refined our hypothesis according to the mechanism active in the experiment.

We gathered monthly heroin and cocaine price and purity contents from undercover DEA drug purchases from the System to Retrieve Information from Drug Evidence (STRIDE). We collected data for the years 1985 to 2003. We dropped potency values that were greater than 100 and prices that were equal to zero because these values are not possible. Extremely high prices exist in both heroin and cocaine price distributions; therefore we dropped prices that exceeded the 99th percentile from our estimation in order to prevent likely erroneous entries from biasing our results. We averaged both price and purity measures on the annual state level, and then constructed an inflation-adjusted price per pure gram measure by dividing prices by the purity content and then adjusting by the CPI. All three measures were logged so that our estimates present partial elasticities.

Monthly state level covariates were obtained from the Current Population Survey (CPS) for the years 1977 to 2003. Our controls include both household and individual level measures. Our household measures include annual household income and number of children receiving free school lunch. Household income is defined as the sum of all income earned by all household occupants over the course of the previous year, and we included annual state-level averages across all households in the survey in our study. The latter variable was not included in our analysis, but was used to generate annual state proportions of households that have a child receiving free lunch. Our individual level measures are the age, sex, race, and highest educational attainment of the respondent. We separated age into four groups: 0 to 18, 19 to 30, 31 to 64, and 65 or older. We grouped race into white, black, Asian, and other. Educational attainment was subsetted into less than a high school diploma, a high school diploma or equivalent, some college, and a college degree or more. We then constructed measures for the proportion of the state for each age group, gender, race, and educational attainment level. These measures were used as state level controls.

We used estimates of annual state population sizes from the Center for Disease Control's Surveillance, Epidemiology, and End Results Program (SEER) to construct per

capita rates for respective measures, as detailed above. Since SEER presents several differing estimates, we utilized their unadjusted data set.

4 Estimates of the Effect of Prison Expansion on Criminal Justice and Drug Outcomes

4.1 Did Prison Expansion Increase Incarceration?

The validity of our inference on drug markets is reliant on whether the Texas criminal justice system exhibited a real behavioral response to the prison expansion. While it is clear that Texas's prison capacity underwent a significant expansion in 1993, the impact of this event on criminal markets was directly tied to the degree to which Texas utilized the additional prison capacity. If the Texas criminal justice system does not change its behavior in any meaningful manner, then the prison expansion would be expected to have little to no effect on drug markets. However, if Texas did indeed utilize the additional capacity, then long term declines in drug usage may have occurred if either drug prices increased or drug addicts were removed from the population via the expanding prison population.

We begin our analysis by investigating if the Texas prison expansion had any effect on statewide incarceration rates. This relationship may be expressed by the following regression model:

$$y_{st} = \alpha_s + \tau_t + \beta \cdot \mathbf{I}\{s = TX\} \cdot \mathbf{I}\{t \geq T_0\} + \psi X_{st} + \varepsilon_{st} \tag{4}$$

where y is the outcome of interest, α_s is a vector of state dummies, τ_t is a vector of year dummies, T_0 is the year of treatment, X_{st} is a matrix of state level covariates and ε_{st} is the structural disturbance term. The coefficient of interest is β which is the difference-in-difference (DD) estimate of the Texas prison expansion on our set of outcomes. In this stage, we use 1993 as the treatment year because it marks the year that the Ann Richards prison construction began based on both our reading of this period (Perkinson, 2010) and investigation of the changes in prison capacity itself (Figure 2).

DD inference relies on asymptotic properties associated with the assumption that the number of individuals within a state and/or the number of states increases. However, this assumption does not apply to our study because the treatment occurs only in Texas. To address this issue, we employed a variation of Fisher's permutation test (Fisher, 1935; Buchmueller, DiNardo and Valletta, 2011). Our methodology involves estimating Equation (4) for every state in the sample in order to construct a distribution of estimates across all states for β. We determine significance by rank-ordering the list of estimates and dividing them by the number of units in the sample. Achieving statistical significance in a two-tailed test requires Texas to be ranked at most second from the top or bottom end of the distribution. The null hypothesis is that Texas evolved no differently than other states, indicating that the prison expansion carried no effect into the respective outcome.

Table 1 presents the DD results of estimating Equation (4) for each criminal justice outcome using 1993 as the treatment year. We provide the 5th and 95th percentile of the distribution for the placebo estimates. P-values resulting from a two-tailed test for the Texas estimate and observation counts are also presented for each model. All models include state and year fixed effects and time-variant state controls from the CPS, including the percentage of the population that is female, percentage of the population that is male, percentage of the population that is black, percentage of the population that is white, percentage of the population that is Asian, percentage of the population that is of a different race, percentage of the population that has less than a high school degree, percentage of the population that has a high school degree or equivalent, percentage of the population that has some college education, percentage of the population that has a college degree, percentage of the population that is age 18 or younger, percentage of the population age 19 to 30, percentage of the population age 31 to 64, percentage of the population 65 or older, percentage of households with a child receiving free school lunch, and average household income. We also present a histogram for the distribution of the incarceration rate and drug arrests placebo estimates in Figure 4. The Texas estimate is indicated by the solid black line.

For our fixed effects estimator to identify β consistently, the outcomes of interest would have had to evolved similarly across treatment and control groups absent the treatment.

This assumption, called the parallel trends assumption, cannot be directly tested because data on the posttreatment counterfactual for Texas is not available, but we can evaluate its presence in the pretreatment period by comparing outcomes for treatment and control group units using an event study model. The model is:

$$y_{st} = \alpha_s + \tau_t + \beta_t \cdot \mathbf{I}\{s = TX\} \cdot \mathbf{I}\{t = 1981, \cdots, 2003\} + \psi X_{st} + \varepsilon_{st} \tag{5}$$

where β_t is the coefficient values with respect to the omitted year (1993) and all other variables are as in Equation (4). We again utilize randomization inference by estimating Equation (5) for each state in the distribution in order to construct a distribution of estimates for β_t. If the parallel trends assumption holds for Texas in a given year, then Texas's annual estimate of β_t would be expected to lie within the sampling distribution of the other states. Thus, in order to satisfy the parallel trends assumption, Texas's yearly estimates should lie within the sampling distribution during the pretreatment period. Furthermore, movement outside of the distribution of the other states in the posttreatment period would lend evidence that the treatment had a real effect on Texas. Figure 6 presents the event study plot for incarceration rate estimates. The dots represent the annual estimate for Texas relative to 1993. The vertical bars represent confidence intervals for the 5th to 95th percentile of the sampling distribution's annual estimates.

Column 1 of Table 1 presents our estimates for Equation (4) using incarceration per 100,000 state population as the dependent variable. Our results indicate a large and positive effect of the prison expansion on the Texas incarceration rate, estimating an increase of 309.6 incarcerated individuals per 100,000 following 1993. Furthermore, this effect is strongly significant because Texas exhibits the largest estimate of any state.

Careful evaluation of the coefficient estimates for our event study methodology (Equation (5)) on total incarceration rates indicates that the trends are not parallel for the years immediately prior to treatment. This is not necessarily a surprising result. The *Ruiz v. Estelle* overcrowding stipulations created a unique environment in which growth in incarceration rates were largely dependent on prison construction, and it would not be expected that other states's prison systems were subject to similar dependencies. To overcome this

problem, we used synthetic control models which are not dependent on the parallel trends assumption to investigate if the 1993 Richards expansion had a true effect on incarceration rates. The results are presented later and corroborate our DD results.

4.2 Pathways: Probability of Arrest, Prison Releases, Prison Admissions, and Discretionary Paroles

With the understanding that the Richards expansion did allow incarceration rates to grow, we seek to determine the source of this change. To do so, we investigate which parameters of Equation (1) (c, p, or θ) change with respect to drug markets. Inspection of Figure 1 suggests that Texas's manipulation of parole rates might be such a mechanism. Indeed, it appears that paroles were largely used to ensure the 'artificial' steady state observed in the 1980s, and that a tightening in parole policy allowed the newly constructed prisons to fill over the course of the 1990s. This change amounts to a decline in θ. Of course, one could imagine that the increase in incarceration may have been driven by increased arrests (particularly drug arrests) or increased police employment, as opposed to paroles. Either of these changes would reflect increases to p. We investigate this by estimating Equations (4) and (5) for drug arrest and police officer employment rates, but none of those phenomena are statistically different from zero in our models (Table 1, columns 2-3). Our event study plot of Equation (5) for drug arrests is shown in Figure 7, and demonstrates that the prison expansion had virtually no effect on drug arrests.

While we find no change in law enforcement utilization, it is possible that courts changed their sentencing behavior by raising the probability that an offender is sentenced or by extending sentence lengths. The first adjustment equates to a change in the parameter p_t, while the second adjustment equates to a change in the parameter θ (or equivalently, $E(T)$). We examined both cases by examining prison admissions and prison releases per 100,000 state inhabitants. The first case would result in positive estimates for prison admissions, while the second case would result in negative estimates for prison releases. These estimates are presented in column 4-5 of Table 1, and are statistically insignificant in both cases. Thus, our results indicate that it is unlikely that admissions were the primary driver behind the change in incarceration rates.

Finally, we investigated whether Texas adjusted the discretionary parole rate in response to the prison expansion. However, analyzing pure parole rates does not entirely capture behavioral patterns of Texas parole boards since the feasibility of paroles is largely tied to the offense composition of the contemporaneous inmate cohort. For example, if the cohort of inmates for a given year were particularly violent, then parole boards would be less inclined to approve a high number of paroles, regardless of capacity pressures. However, if paroles are the vehicle through which Texas manipulates Equation (5), then we would expect paroles to constitute a large percentage of prison releases during the entire pre-1993 period. Once overcrowding pressures were alleviated, we would then expect this proportional parole rate to decline, signaling that Texas prisons were no longer relying on paroles to satisfy the litigation requirements. Such an occurrence would reflect a decline in θ in Equations (1) and (2), or equivalently $E(T)$ in Equation (3). We investigated this relationship by estimating Equation (4) for which paroles as a proportion of total releases is used as the dependent variable. In order for paroles to be the root cause of the increase in incarceration, estimates of β would need to be negative. This equates to a decline in the parameter θ. Shown in column 5 of Table 1, we estimate that paroles constitute approximately 16.6 percent less of total annual releases following 1993, but this effect is not statistically significant. The reason for this is largely because of what can be seen in Figure 1. Paroles as a share of releases began falling in 1990, shortly after the first investments in prison capacity, and thus the parallel trends assumption governing the pretreatment period falls apart.

To summarize, our analysis presented in Table 1 indicates that the 1993 Texas prison expansion allowed statewide incarceration rates to balloon. Based on our results in this section, it appears unlikely that the primary driver of incarceration rates is a change in law enforcement utilization, prison admissions, or sentencing practices; however, there is empirical evidence that prison officials manipulated the discretionary parole rate in order to retain a greater number of inmates; our best evidence for this is summarized in Figure 1. In terms of Equation (1), this may be interpretted as a reduction in the parameter θ while c and p remain constant. With this knowledge in mind, we turn to the next step of our analysis devoted to determining the effect on drug markets.

4.3 Impact on Drug Prices and Purity

Having identified paroles as the mechanism most likely responsible for changes in incarceration rates, the remaining portion of our analysis focuses on the effect that the prison expansion had on drug markets. We began by analyzing recorded prices paid by undercover drug purchasers. Furthermore, we construct a purity adjusted real price by dividing price per gram by the purity content. This ratio was then adjusted by the CPI for a given year. This can be considered to be the real price consumers are paying for a pure gram of cocaine or heroin. If a substantial number of potential drug consumers are withheld from the market by the reduction in paroles, then demand for cocaine and heroin would be expected to decline. This in turn would be reflected by reductions in drug prices. On the other hand, if drug suppliers account for the reduced probability of early releases then we may expect prices to rise as this equates to a rise in costs. If a combination of these two effects occurs, then there would likely be an ambiguous effect on prices. Alternatively, suppliers may compensate for the extended sentences by reducing the potency content of their substances in order to counteract the rising cost of longer sentences. We explore these effects by estimating Equation (4) for the logged outcomes of average price per gram, average price per pure gram, and average purity content for both cocaine and heroin. Thus, our estimates reflect price and potency semielasticities. The treatment year for drug markets is ambiguous because incarceration rates do not rise in a one-to-one fashion with prison capacity. Thus, we present estimates corresponding to two distinct models: one using 1993 as the treatment year and one using 1994 as the treatment year. For the sake of brevity, DD placebo distributions and event study plots are not presented for these outcomes.

Estimates for heroin measures are reflected in columns 1 through 3 of Table 2. Panel A presents results for models in which 1993 is used as the treatment date, and Panel B presents results for models in which 1994 is used as the treatment date. Contrary to claims made by other writers (MacCoun and Reuter, 2001), we do not find that higher rates of incarceration raised heroin prices. If anything, our results indicate that heroin prices declined with the increase in incarceration, suggesting that a reduction in demand had

occurred. Our point estimates suggest that the prison expansion resulted in approximately 0.844 to 0.845 and 0.324 percent to 0.354 percent declines in the price per gram and price per pure gram of heroin. Our results also indicate a 0.019 to 0.022 percent increase in average potency content of heroin. However, no estimate is statistically significant. As a result, any changes in price and purity appear to be driven by secular trends as opposed to being unique to Texas, and thus may not be attributable to the Texas prison expansion.

We present estimates for cocaine market measures in the first three columns of Table 3. Coefficients are organized in the same fashion as our heroin estimates from Table 2. In all cases cocaine estimates are inverse to our heroin estimates, suggesting a reduction of supply in cocaine markets. We estimate that the prison expansion resulted in a 0.017 to 0.035 percent and 0.142 to 0.161 percent increases in price per gram and price per pure gram of cocaine. Furthermore, we estimate a 0.139 to 0.160 percent reduction in the average potency content of cocaine following the the prison expansion. However, as in the case of heroin, all specifications yield insignificant results, and thus any changes do not appear to be necessarily attributable to the rise in incarceration rates. This stands in contrast to Kuziemko and Levitt (2004) who find that imprisoning drug offenders led to higher cocaine prices.

4.4 Impact on Drug Treatment Admissions

In the previous section, we did not find any substantial changes in prices or potency within heroin and cocaine markets. However, the three measures we investigated–price per gram, potency, and price per pure gram–are all effectively measures of price. It is possible that demand and supply simultaneously respond to Equation (3) in such a way that price holds constant but drug use changes. Determining if any such adjustments occur in reality is of principal importance, since the negative externalities associated with drug markets are largely a function of the prevalence of substance abuse. As such, any analysis would be incomplete without investigating how illicit drug use responds to exogenous shocks to incarceration. The last stage of our analysis seeks to answer this question. In particular, we investigate changes in total treatment admissions, self-admissions, and criminal justice referrals into publicly funded substance abuse programs for both cocaine and heroin.

Thus, we estimate a total of six different outcomes for Equation (4). We also present event study plots corresponding to Equation (5) estimates for criminal justice referral measures. As in the earlier section, we present two sets of estimates corresponding to models in which the treatment date is 1993 and 1994. Furthermore, we utilize logged values so that our estimates reflect annual percentage changes in admissions in the posttreatment period relative to the pretreatment period.

Before discussing our estimates for treatment admissions, several theoretical points should be noted about the expected effect and expected directional signs of incapacitation and deterrence effects. First, as our estimates in Table 1 and the trends shown in Figure 1 indicate, the observed changes in incarceration were most likely driven by declining paroles. We therefore expect any observed changes in drug treatment admissions to be largely due to subsequent declines in paroles and increases in effective sentencing. Since our criminal justice referral measure includes those who enter treatment as a requirement of parole, any incapacitation effect caused by a reduction in paroles would be expected to have a substantially larger effect on criminal justice referrals relative to self-admissions. On the other hand, deterrent effects may display themselves in various ways. Drug treatment admissions may serve as a method for users to quit using drugs, and therefore it is possible that deterrent effects result in an upward bias on our estimates. However, this bias would be expected to only affect self-admissions (and total admissions by pass through). At the same time, if drug users are motivated to quit without the aid of treatment, then drug treatment admissions would be expected to decline in general. Thus, deterrence will present itself as a negative effect in criminal justice referral measures, whereas the sign for total and self-admissions is ambiguous. In all cases, incapacitation effects would be expected to be negative.

Columns 4-6 in Table 2 presents our results for estimating Equation (4) for logged heroin treatment admission outcomes. Panel A presents estimates for model specifications using 1993 as the treatment year, and Panel B presents estimates for model specifications using 1994 as the treatment year. Our estimates predict a 0.645 to 0.662 percent decline in total annual heroin admissions, a 0.513 to 0.542 decline in annual heroin self-admissions, and a 1.443 to 1.579 decline in annual heroin criminal justice referrals. Criminal justice

referral estimates are significant under both specifications, while total admission estimates are significant only under the 1993 treatment year specification. Heroin self-admission estimates are not significant under either treatment year. Figure 5 presents placebo distributions for both criminal justice referral models. We also present an event study plot for the estimates of Equation (5) in which log criminal justice referrals are the outcome and 1994 is omitted in Figure 10. These estimates provide evidence that the parallel-trends assumption holds for heroin criminal justice referrals, though given the paucity of pretreatment data, we are reluctant to overstate our case here. Our Equation (4) estimates reveal that heroin treatment admissions decline significantly as a result of the increase in incarceration rates; however, this change appears to be primarily driven by reductions in criminal justice referrals. An examination of Figure 10 supports this conclusion, but also indicates that this change appears to have occurred with a two-year lag.

We similarly estimate Equation (4) for logged cocaine treatment admissions outcomes, which we present in Table 3. Panel A presents estimates for model specifications using 1993 as the treatment year, and Panel B presents estimates for model specifications using 1994 as the treatment year. We estimate a 0.423 to 0.563 percent decline in total annual cocaine admissions, a 0.130 to 0.235 percent decline in annual cocaine self-admissions, and 0.770 to 0.993 percent decline in cocaine criminal justice referrals. Our estimates for criminal justice referrals are significant under both treatment year specifications while our general admission estimates are only significant when 1994 is used as the treatment year. Self-admission estimates are not significant under either model. We present placebo distributions for the criminal justice referral specifications in Figure 5. We also estimate Equation (5) for cocaine criminal justice referrals and present the resulting event study plot in Figure 11. It appears that the parallel trends assumption holds for cocaine criminal justice referrals. As in the case of our heroin estimates, Figure 11 indicates that the cocaine criminal justice referrals change in response to the rising incarceration and parole rates with a lag, although the lag for cocaine appears to be three years as opposed to two years. Nonetheless, the results reinforce that criminal justice referrals declined following the Texas prison expansion.

Our estimates for heroin and cocaine treatment admissions indicate that drug use

among the general population declined in tandem with rising incarceration rates. However, this decline appears to have been largely driven by declines in criminal justice referrals for treatment. Furthermore, this decline occurred with a several year lag. While it is not possible to disentangle deterrent and incapacitation effects, this lends evidence to the notion that our results are primarily driven by incapacitation. It seems unlikely that the general population would have responded to changes in incarceration and parole rates two to three years after the fact. A more plausible explanation is that parolees exhibit a transitory deterrent effect when they reenter society. They avoid activities, such as drug use, that would lead to a parole violation. However, this effect diminishes with time, leading parolees to violate parole and be referred to treatment. As a result, changes to the population of parolees would not impact drug markets or treatment admissions for several years. Accordingly, our estimates would be largely reflective of an incapacitation effect associated with higher incarceration rates. This would also provide a potential explanation for our findings in Section 6. Drug market supply and demand curves would be expected to be less sensitive to interventions targeted at parolees. This is because parolees would be expected to use drugs less frequently than drug users who are not on parole. Furthermore, parolees would be more likely to be apprehended for drug use because they are under closer supervision of law enforcement. Thus, it is possible that parolees only reenter drug markets for a brief stint before they are apprehended and referred to treatment by their parole officers. As such, the marginal incapacitation impact on the demand for illicit drugs associated with incarcerating a parolee would likely be far less than that of the typical drug user.

Another possible explanation for these results may be that prison construction took several years to complete. If this is the case, then drug consumption would not exhibit a significant response until prison construction is completed and additional capacity is made available. Under this interpretation, our results may be driven by both incapacitation and deterrence without any indication as to which effect is dominant. Nonetheless, this explanation does not provide an obvious argument as to why declines in heroin occur one year before that of cocaine, and thus we favor the former explanation.

5 Robustness: Synthetic Control Analysis

Given our concern that the common trends assumption may not hold for any of our outcomes, we estimate several synthetic control models for robustness (Abadie and Gardeazabal, 2003; Abadie, Diamond and Hainmueller, 2010). The synthetic control approach is a generalization of the DD framework, but unlike DD models, the synthetic control model does not rely on the common trends assumption for identification. Synthetic control also uses a subset of units for controls for comparison (as opposed to all states). This method selects control states that exhibit the same pretreatment dynamics as Texas. If there is some concern that our main DD results are biased, then the synthetic control method is an alternative means of relaxing that assumption for estimation.

Let Y_{st} be the outcome of interest for unit s of $S+1$ state units at time t, and treatment group be $s = 1$. The synthetic control estimator models the effect of prison capacity expansion at time T_0 on the treatment group using a linear combination of optimally chosen states as a synthetic control. For the posttreatment period, the synthetic control estimator measures the causal effect as $Y_{1t} - \sum_{s=2}^{S+1} w_s^* Y_{st}$ where w_s^* is a vector of optimally chosen weights. Matching variables, X_1 and X_0, are chosen as predictors of post-intervention outcomes and must be unaffected by prison capacity expansion. We describe the covariates used in these models in Tables 4-6.

Similar to our earlier inference, we follow the recommendation by Abadie, Diamond and Hainmueller (2010) and use a Fisherian inferential technique based on several placebo exercises. We apply the treatment year to every state in our sample of units (50 states plus District of Columbia), placing Texas back into the set of states in the donor pool. We estimate a set of optimal weights that minimizes the root mean squared prediction error (RMSPE) pretreatment, and then apply those weights to the outcomes for our synthetic control *ex post*. We then calculate the RMSPE for the posttreatment period and the pretreatment period separately. We generate a ratio of the post/pretreatment RMSPE for each state. This ratio should be high for Texas, suggesting that the model fit the pretreatment trends well (represented by a small pretreatment RMSPE) but has failed to replicate the posttreatment series (represented by a large posttreatment RMSPE). We

rank the ratio of post-/pretreatment RMSPE for all units in our sample from highest to lowest. The probability that chance could have produced our Texas results is the rank order of Texas in that distribution divided by the number of units. This exercise allowed us to examine whether the effect of the prison expansion was large relative to the distribution of the effects that we estimated for states not exposed to such expansion.

It should be noted that this placebo-based inference method ultimately requires two separate conditions be satisfied in order to find statistically significant results. Note that the test statistic equals $\frac{RMSPE_{-t}}{RMSPE_{+t}}$, where the numerator is the posttreatment RMSPE and the denominator is the pretreatment RMSPE. In order to have a high rank in the distribution, the Texas effect must simultaneously have a relatively small RMSPE_{-t} and a large RMSPE_{+t}. That is, our estimator must identify a set of states that look nearly identical to Texas pretreatment while also finding a relatively large posttreatment effect. This procedure seems mainly appropriate for relatively large treatment effects and treatment units that are similar to the donor pool units, in other words.

Figures 12-15 display the gap between observed and synthetic estimates for the total incarceration rate, male drug arrest rate, log heroin criminal justice referrals, and logged cocaine criminal justice referrals. We focused on these outcomes because earlier analysis showed no effect on other outcomes of interest. We used 1993 as the treatment date for the first three results, while 1994 was used as the treatment date for the latter two because of observed lag effects. We present the rank of the Texas ratios and p-values calculated according to the methodology above on each corresponding figure. Synthetic control estimates improve in accuracy as the pretreatment tail extends. Conceptually, this is due to the fact that posttreatment estimates may be based off of a more complete pretreatment trend. Thus, we utilized the full-time period for each particular outcome measure.

The synthetic control model for total incarceration rates is presented in Figure 12. Texas's ratio of post- to pretreatment RMSPE is ranked second in the distribution, giving it a value of 0.04. The pretreatment fit is very good, and as we saw in Figure 3, there is a large increase in the total incarceration rate following 1993. This, combined with graphical evidence in Figure 3 and our marginal effects from Table 1 suggest that the

immediate impact of the prison expansion was a large increase in the total incarceration rate.

The synthetic control model for male drug arrests is presented in Figure 13. Texas' ratio is ranked 3rd most extreme and the corresponding p-value is 0.06. This indicates that male drug arrests did indeed respond to the Texas prison expansion, contradictory to our DD results. However, a closer inspection of Figure 13 does not substantiate this result. The gap in both the pretreatment and posttreatment periods is small. Furthermore, the posttreatment gap is substantially smaller than those of many other states. The estimated causal effect represented is very small and switches between a positive and negative effect over time. Thus, we believe that this effect is merely a precisely estimated zero to small effect.

Figure 14 displays the prediction error gap between Texas and its synthetic estimate for logged heroin criminal justice referrals per 100,000 state inhabitants. The Texas test statistic is the second largest and carries a p-value of 0.05 indicating a significant change in heroin use as a result of the prison expansion. Furthermore, the pretreatment fit is very good and the trend visually supports that there was a significant decline in heroin use. This corroborates with our previous DD findings.

The gap in prediction error for the model where cocaine criminal justice referrals is the outcome is shown in Figure 15. The corresponding Texas rank and p-value is 11 and 0.25 indicating no statistically significant change. This contradicts our DD estimates. However, the pretreatment fit for Texas looks substantially better than many other states, indicating that there might be a problem of generalizability as in the case of the drug arrest synthetic control model. The fact that the trend for cocaine in Figure 15 strongly matches that of heroin in Figure 14 indicates that the prison expansion did affect cocaine and heroin use in similar ways.

6 Discussion and Conclusion

In conclusion, we discuss what we have learned from this exercise. Starting in the late 1980s, and particularly in the early 1990s, Texas made large investments in its prison

infrastructure leading to a new incarceration steady state in Texas. The immediate effect of the expansion was a voluntary decrease in using paroles as a mechanism for managing prison flows. With increased capacity, the state not only reduced its use of paroles, but also began sending more people to prison, presumably for longer periods, roughly doubling incarceration rates in only two years. Texas, we argued, rapidly moved into an age of mass incarceration as a result of these prison investments.

It stands to reason, based on earlier evidence (MacCoun and Reuter, 2001; Kuziemko and Levitt, 2004) that this shift may have reduced drug use in the population through a type of deterrence mechanism; for example, in the form of higher drug prices due to the higher expected costs of drug trafficking. Kuziemko and Levitt (2004) found that imprisoning drug offenders resulted in cocaine prices increasing on average 5-15%, ceteris paribus. We did not find evidence of such deterrence effects. We did not find any effect on drug prices whatsoever following the expansion, nor did we find any change in voluntary treatment admissions. Instead, our results appear to indicate that the majority of declines in drug consumption were driven by incapacitation effects. We reach this conclusion as reductions in drug consumption are largest several years after prison construction. It seems unlikely that drug users would be deterred three years after prison construction begins, though we note that we cannot explicitly disentangle deterrence and incapacitation effects. This type of intervention may not be a socially optimal solution to containing drug markets; however, a complete analysis comparing the costs of treating addiction via drug treatment programs versus the costs of incarceration itself is needed to make a determination. Mueller-Smith (2015) finds that these costs can be large and include negative effects on violence and labor market outcomes. More work needs to be done to better understand the costs and benefits of such events.

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Table 1 Estimated effect of prison expansion on prison measures

Prison Measures:	**Incarceration**	**Sworn Officer Employment**	**Drug Arrests**	**Prison Admissions**	**Prison Releases**	**Paroles/Total Releases**
			1993 Treatment Year			
Prison expansion	269.452	-6.227	-6.865	26.635	48.458	-0.285
5th percentile	-79.898	-21.341	-61.048	-71.607	-79.187	-0.165
95th percentile	162.797	23.679	129.653	74.428	65.998	0.177
Two-tailed test p-value	0.04	0.39	0.76	0.69	0.24	0.05
N	612	612	584	608	608	581
State and year FE	Yes	Yes	Yes	Yes	Yes	Yes
Time variant controls	Yes	Yes	Yes	Yes	Yes	Yes

State population are used as analytical weights. Time-variant controls include state-level values of total food stamp expenditures, mean of households receiving free lunch, household income, percent aged 15-20 and percent aged 21-30, percent white, percent black, percent of population below poverty line and AIDS mortality rates per 100,000. Table presents 5th and 95th percentile confidence intervals from placebo-based inferential calculations, and p-values from a two-tailed test. * $p<0.10$, ** $p<0.05$, *** $p<0.01$

Table 2 Estimated effect of prison expansion on heroin

Heroin:	**Ln(Price/Gram)**	**Ln(Price/Pure Gram)**	**Ln(Purity)**	**Ln(Admiss)**	**Ln(Self)**	**Ln(CJ)**
			Panel A: 1993 Treatment Year			
Prison expansion	-0.845	-0.324	0.022	-0.645*	-0.542	-1.443**
5th percentile	-1.047	-0.999	-0.776	-0.595	-0.736	-1.139
95th percentile	1.711	0.698	0.689	0.922	1.386	1.151
Two-tailed test p-value	0.22	0.36	0.96	0.09	0.39	0.05
N	858	858	912	584	580	576
			Panel B: 1994 Treatment Year			
Prison expansion	-0.844	-0.354	0.019	-0.662	-0.513	-1.579**
5th percentile	-1.025	-0.846	-0.701	-0.682	-0.793	-1.114
95th percentile	1.868	0.800	0.709	0.952	1.527	1.047
Two-tailed test p-value	0.27	0.36	1.04	0.19	0.39	0.05
N	858	858	912	584	580	576
State and year FE	Yes	Yes	Yes	Yes	Yes	Yes
Time variant controls	Yes	Yes	Yes	Yes	Yes	Yes

Time-variant controls include state-level values of percent White, Black, Asian, Other race, less than a high school degree, with a high school degree, with some college, with college degree or higher, aged 0-18, 19-30, 31-65, 65 and older, male, female, as well as average household income and percent who receive free lunch. Table presents 5th and 95th percentile confidence intervals from placebo-based inferential calculations, and p-values from a two-tailed test. * p<0.10, ** p<0.05, *** p<0.01

Table 3 Estimated effect of prison expansion on cocaine

Cocaine:	**Ln(Price/Gram)**	**Ln(Price/Pure Gram)**	**Ln(Purity)**	**Ln(Admiss)**	**Ln(Self)**	**Ln(CJ)**
			Panel A: 1993 Treatment Year			
Prison expansion	0.017	0.142	-0.160	-0.423	-0.130	-0.770*
5th percentile	-0.483	-0.423	-0.149	-0.455	-0.667	-0.684
95th percentile	0.581	0.420	0.183	0.632	0.685	0.900
Two-tailed test p-value	0.84	0.60	0.12	0.18	0.62	0.10
N	950	950	950	587	584	583
			Panel B: 1994 Treatment Year			
Prison expansion	0.035	0.161	-0.139	-0.563**	-0.235	-0.993**
5th percentile	-0.530	-0.507	-0.176	-0.455	-0.671	-0.703
95th percentile	0.534	0.424	0.153	0.595	0.785	0.923
Two-tailed test p-value	0.84	0.52	0.20	0.05	0.48	0.05
N	950	950	950	587	584	583
State and year FE	Yes	Yes	Yes	Yes	Yes	Yes
Time variant controls	Yes	Yes	Yes	Yes	Yes	Yes

Time-variant controls include state-level values of percent White, Black, Asian, Other race, less than a high school degree, with a high school degree, with some college, with college degree or higher, aged 0-18, 19-30, 31-65, 65 and older, male, female, as well as average household income and percent who receive free lunch. Table presents 5th and 95th percentile confidence intervals from placebo-based inferential calculations, and p-values from a two-tailed test. * p<0.10, ** p<0.05, *** p<0.01

Table 4 Actual vs Synthetic Texas Characteristics for Male Drug Arrest Rate Model

Inputs	**Treated**	**Synthetic**
Drug Arrests (1985)	317.967	317.935
Drug Arrests (1986)	294.557	294.510
Drug Arrests (1987)	311.365	311.343
Drug Arrests (1988)	307.413	307.180
Drug Arrests (1989)	357.194	357.039
Drug Arrests (1990)	306.893	306.868
Drug Arrests (1992)	329.385	329.340
Drug Arrests (1993)	335.080	334.801

Table 5 Actual vs Synthetic Texas Characteristics for Incarceration Rate Model

Inputs	**Treated**	**Synthetic**
Total Incarceration Rate (1978)	5.204	5.183
Total Incarceration Rate (1982)	5.463	5.438
Total Incarceration Rate (1985)	5.441	5.428
Total Incarceration Rate (1986)	5.450	5.496
Total Incarceration Rate (1990)	5.679	5.702
Total Incarceration Rate (1993)	5.956	5.864

Table 6 Actual vs Synthetic Texas Characteristics for Log Heroin Criminal Justice Referrals Model

Inputs	**Treated**	**Synthetic**
Ln Heroin Criminal Justice Referrals (1993)	2.541	2.538
Ln Heroin Criminal Justice Referrals (1994)	2.900	2.897
Proportion of Population Female (1992-2003)	0.518	0.517
Average Household Income (1994)	35,628.086	41,763.011
Age 0-18 (1993)	0.310	0.278

Table 7 Actual vs Synthetic Texas Characteristics for Log Cocaine Criminal Justice Referrals Model

Inputs	**Treated**	**Synthetic**
Ln Cocaine Criminal Justice Referrals (1992)	3.997	4.029
Ln Cocaine Criminal Justice Referrals (1993)	4.232	4.291
Ln Cocaine Criminal Justice Referrals (1994)	4.591	4.447
Ln Cocaine Criminal Justice Referrals (1992-2003)	4.115	4.160
Average Household Income (1993)	34,031.371	41,124.126
Age 0-18 (1994)	0.314	0.332
Age 19-30 (1993)	0.186	0.173
Age 65 or Older (1992)	0.102	0.102
Age 65 or Older (1992-2003)	0.102	0.101
Less than Highschool (1993)	0.118	0.041
College (1994)	0.118	0.151

Table 8 Weights for Drug Arrests Synthetic Control

State	Weight
Alabama	0.005
Alaska	0.006
Arizona	0.351
Arkansas	0.014
California	0.140
Colorado	0.003
Connecticut	0.002
Delaware	0.002
Georgia	0.006
Hawaii	0.046
Idaho	0.002
Illinois	0.006
Indiana	0.002
Iowa	0.002
Kansas	0.003
Kentucky	0.087
Louisiana	0.004
Maine	0.003
Maryland	0.010
Massachusetts	0.033
Michigan	0.002
Minnesota	0.004
Mississippi	0.003
Missouri	0.003
Montana	0.002
Nebraska	0.002
Nevada	0.003
New Hampshire	0.007
New Jersey	0.004
New Mexico	0.004
New York	0.002
North Carolina	0.003
North Dakota	0.004
Ohio	0.003
Oklahoma	0.004
Oregon	0.002
Pennsylvania	0.002
Rhode Island	0.004
South Carolina	0.002
South Dakota	0.027
Tennessee	0.003
Utah	0.126
Vermont	0.002

Table 9 Weights for Total Incarceration Rate Synthetic Control

State	**Weight**
Connecticut	0.230
Florida	0.245
Georgia	0.174
North Carolina	0.222

Table 10 Weights for Log Heroin Criminal Justice Referrals Synthetic Control

State	Weight
Alabama	0.000
Maryland	0.484
Missouri	0.332
Oregon	0.117
Utah	0.066

Table 11 Weights for Log Cocaine Criminal Justice Referrals Synthetic Control

State	Weight
Maryland	0.480
Utah	0.520

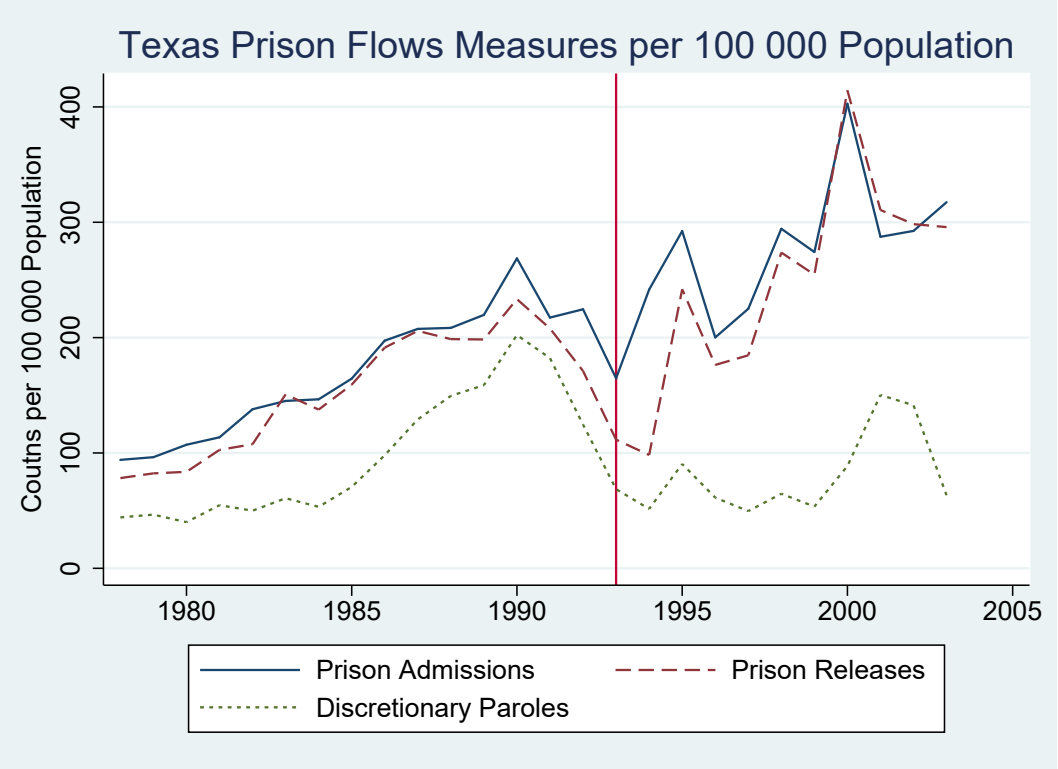

Figure 1 Texas paroles, admissions and releases before and after the year of the major expansion.

Figure 2 Changes in operational capacity for the state of Texas before and after the 1993 prison expansion.

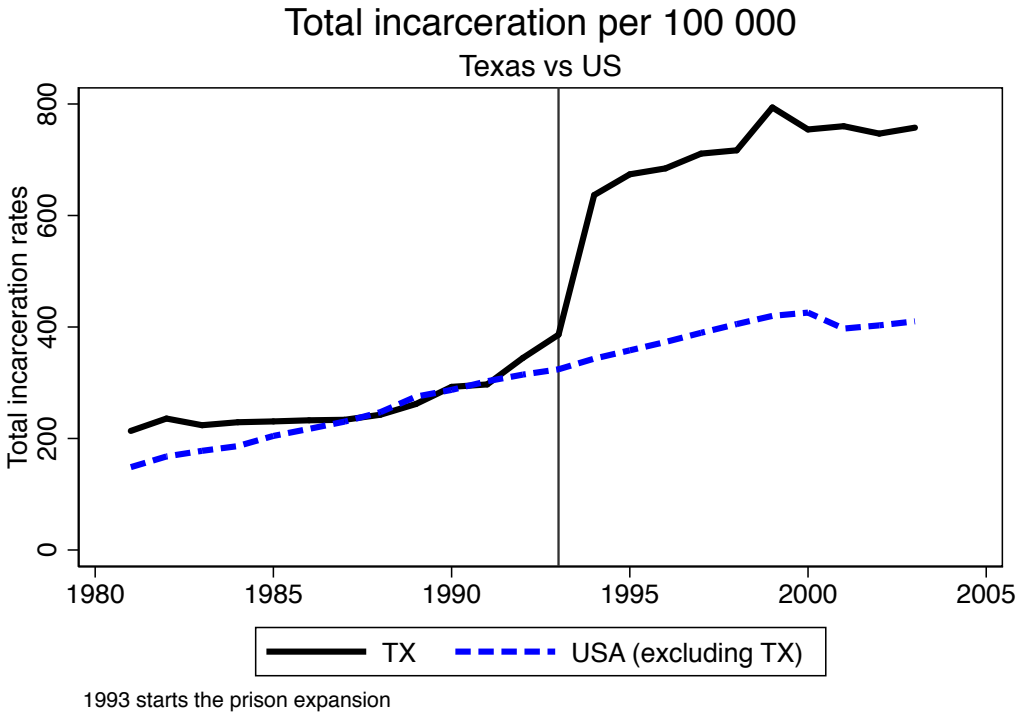

Figure 3 Total incarceration rates for Texas vs. US.

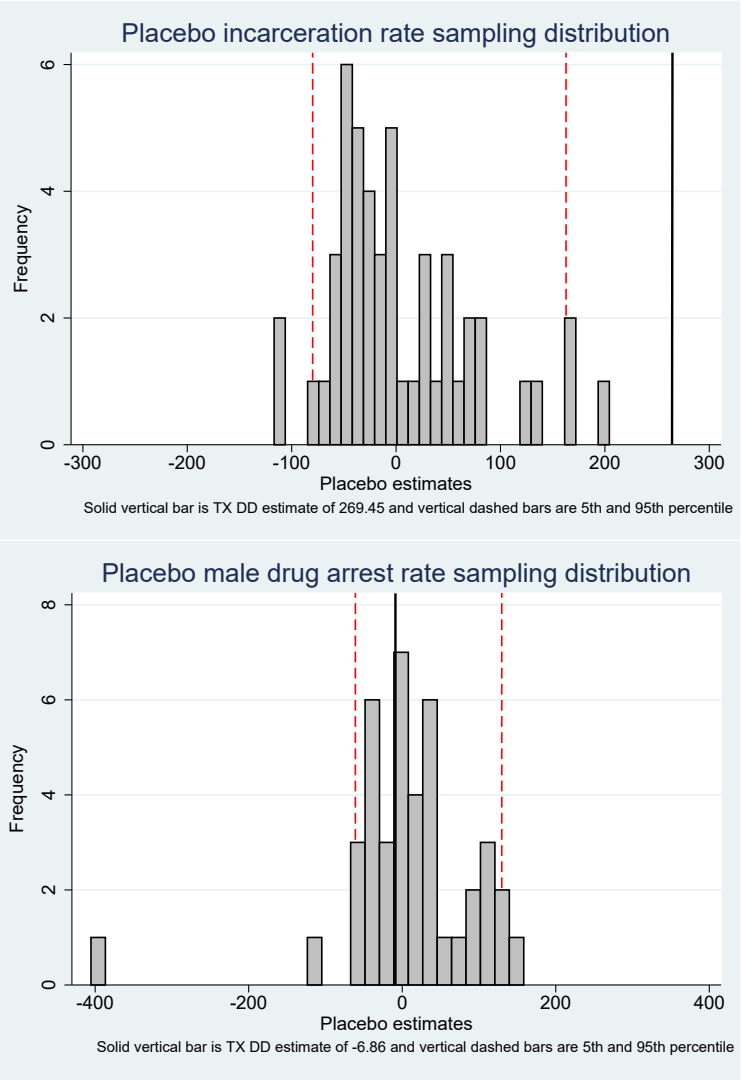

Figure 4 Sampling distribution for total incarceration and arrests from randomization inference for Equation (4).

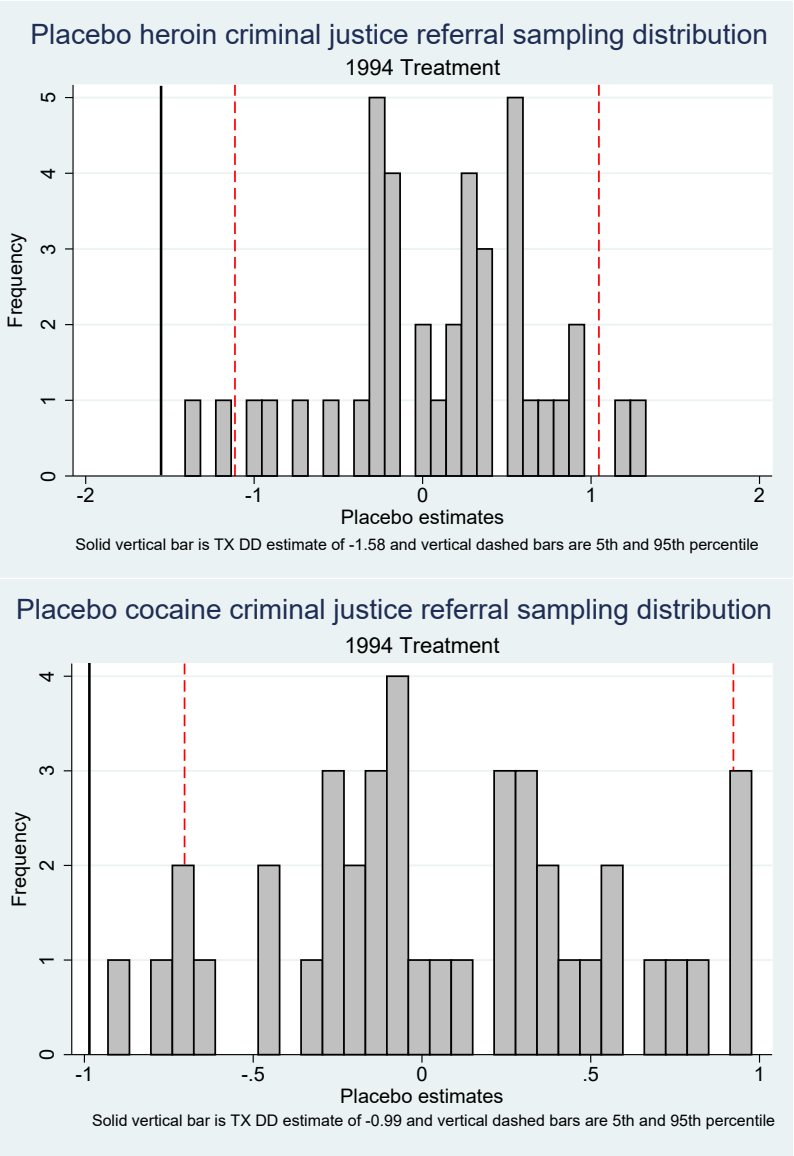

Figure 5 Sampling distribution for TEDS outcomes from randomization inference for Equation (4).

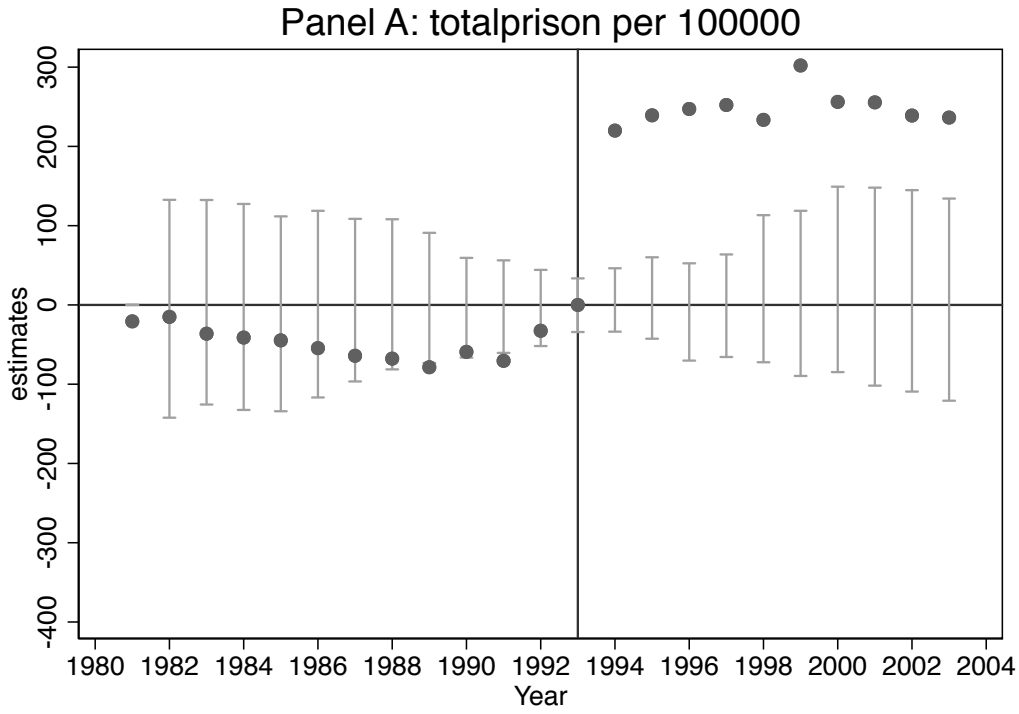

Figure 6 Event study plots for total incarceration per 100,000.

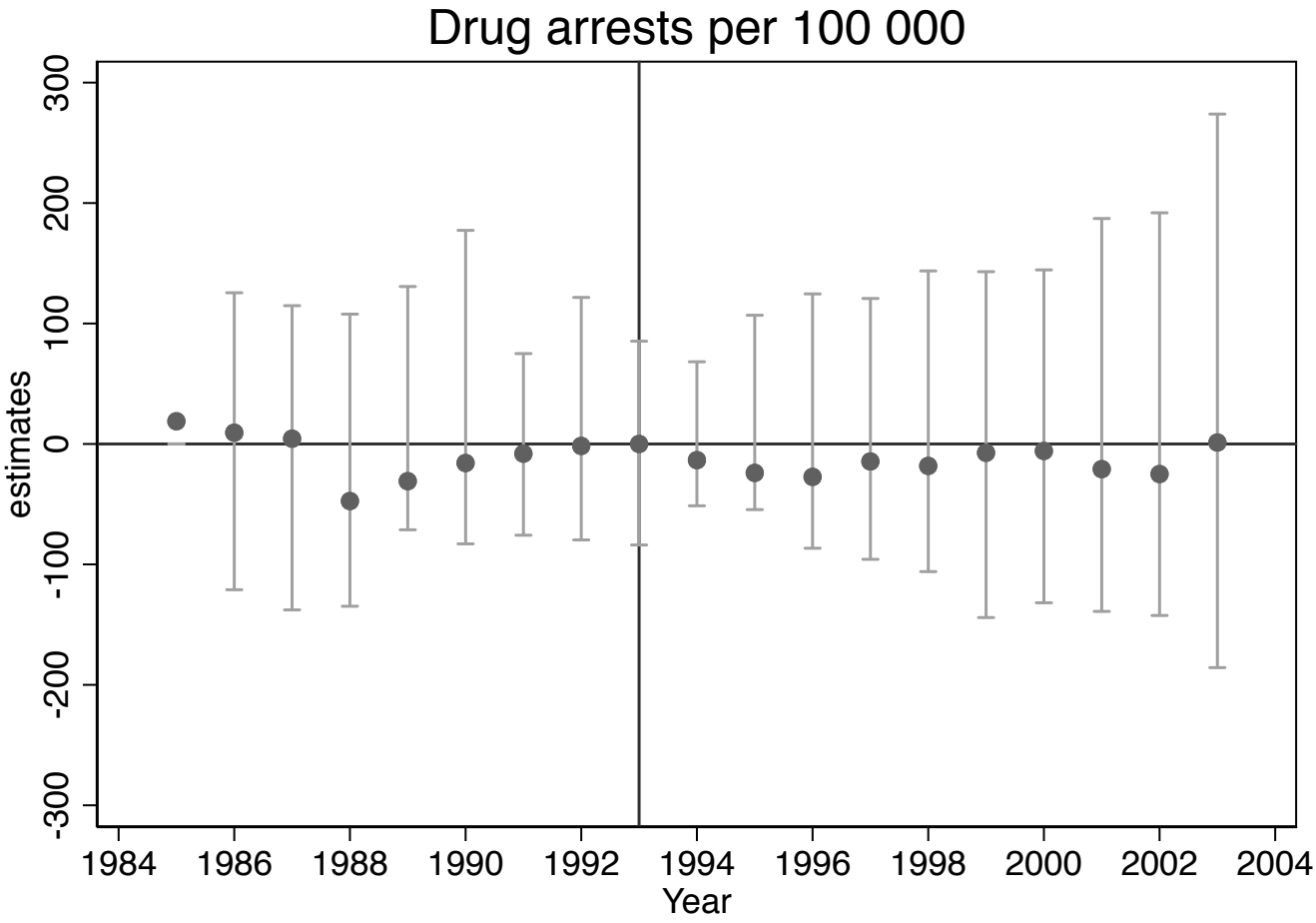

Figure 7 Event study plots for drug arrests per 100,000.

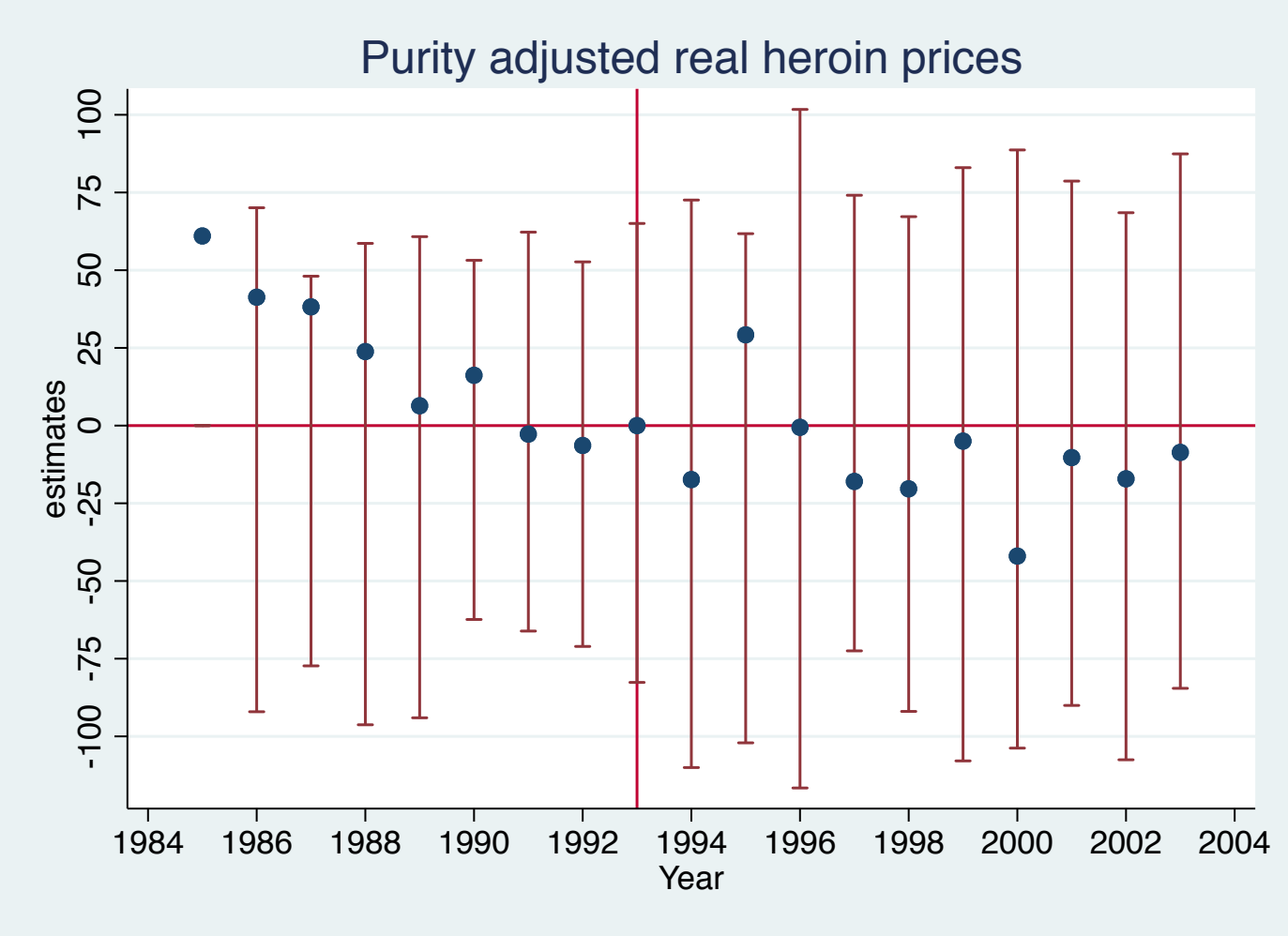

Figure 8 Event study plots of purity adjusted real heroin prices.

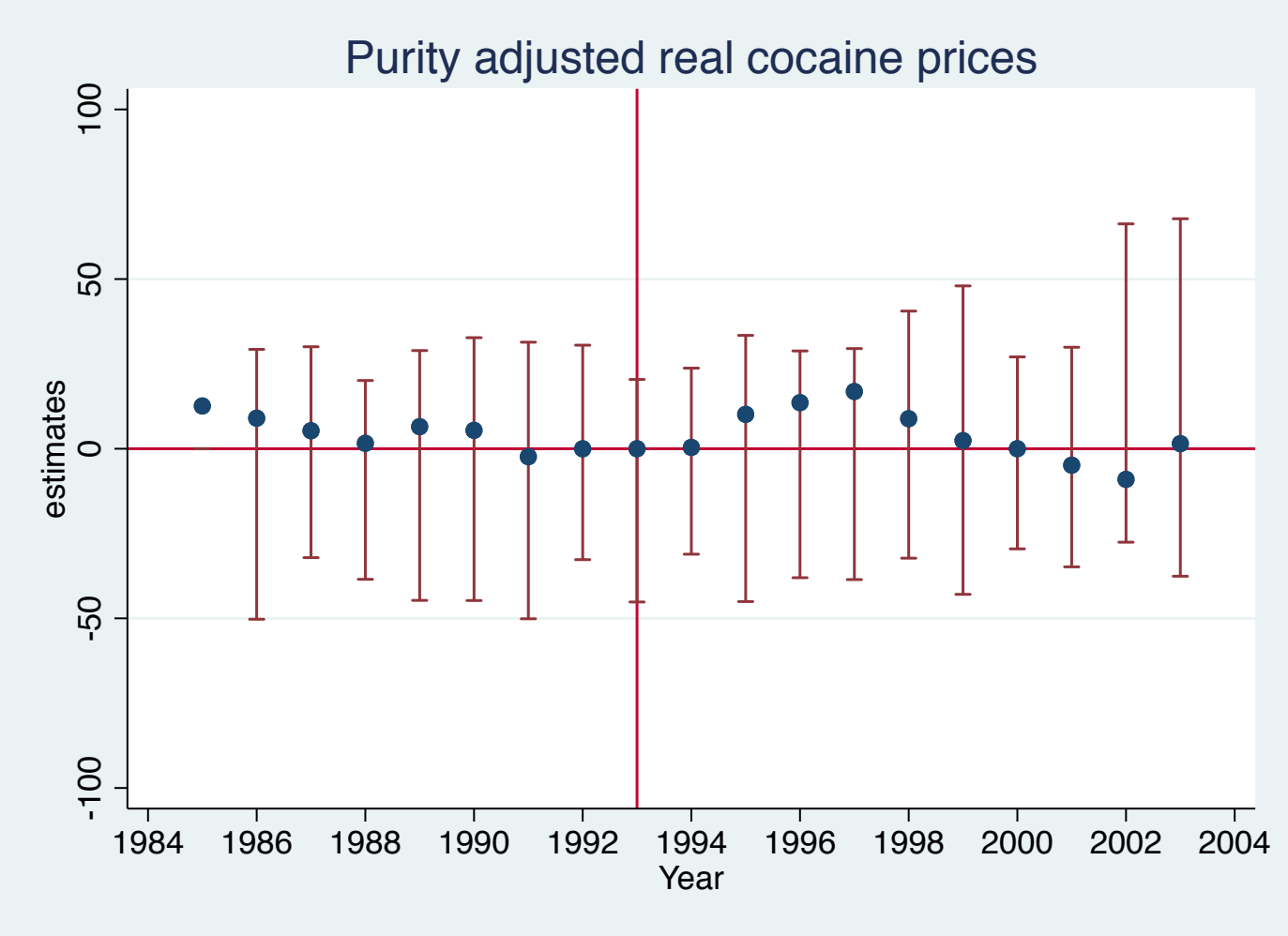

Figure 9 Event study plots of purity adjusted real cocaine prices.

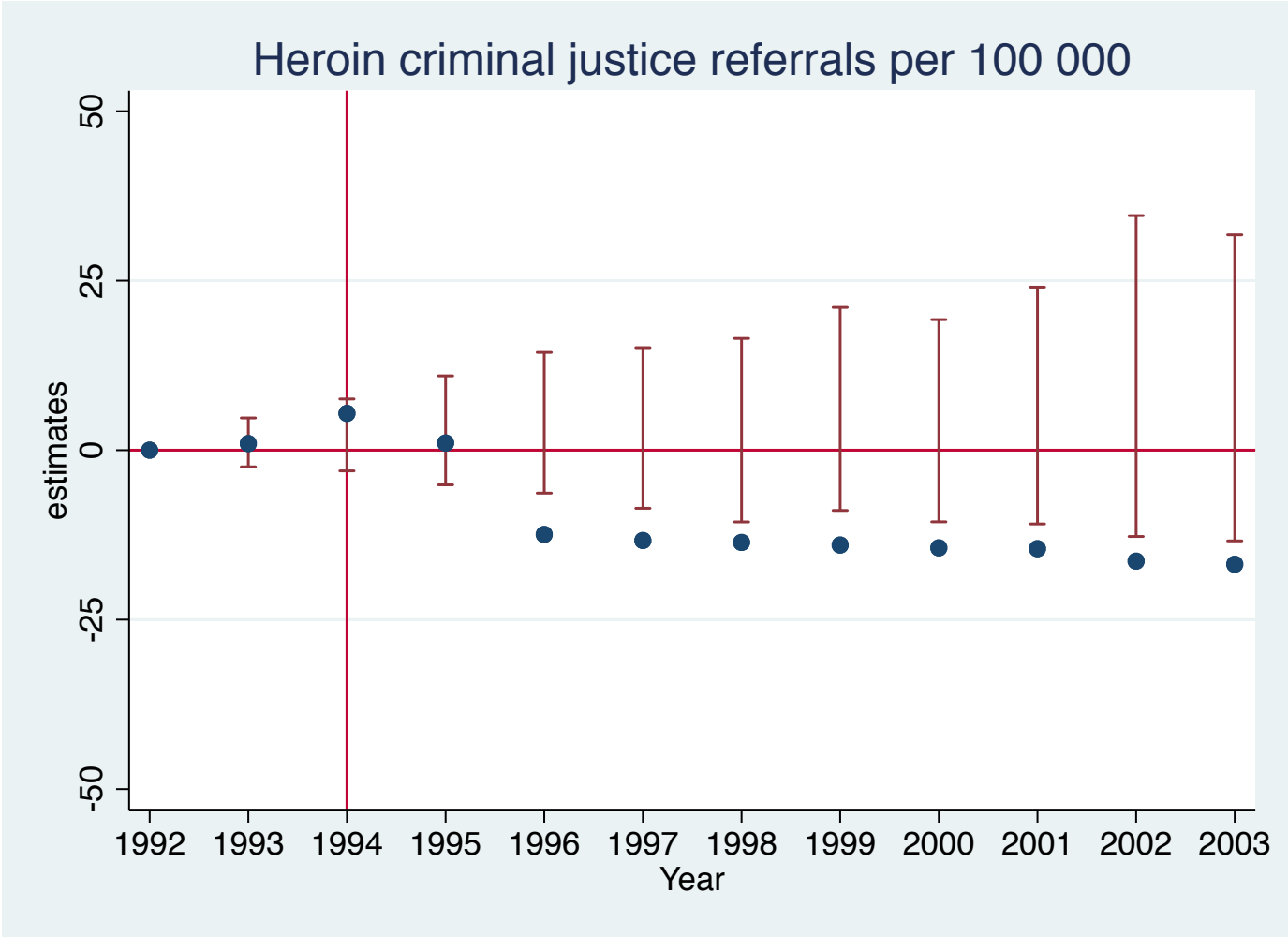

Figure 10 Event study plots of heroin criminal justice treatment referrals.

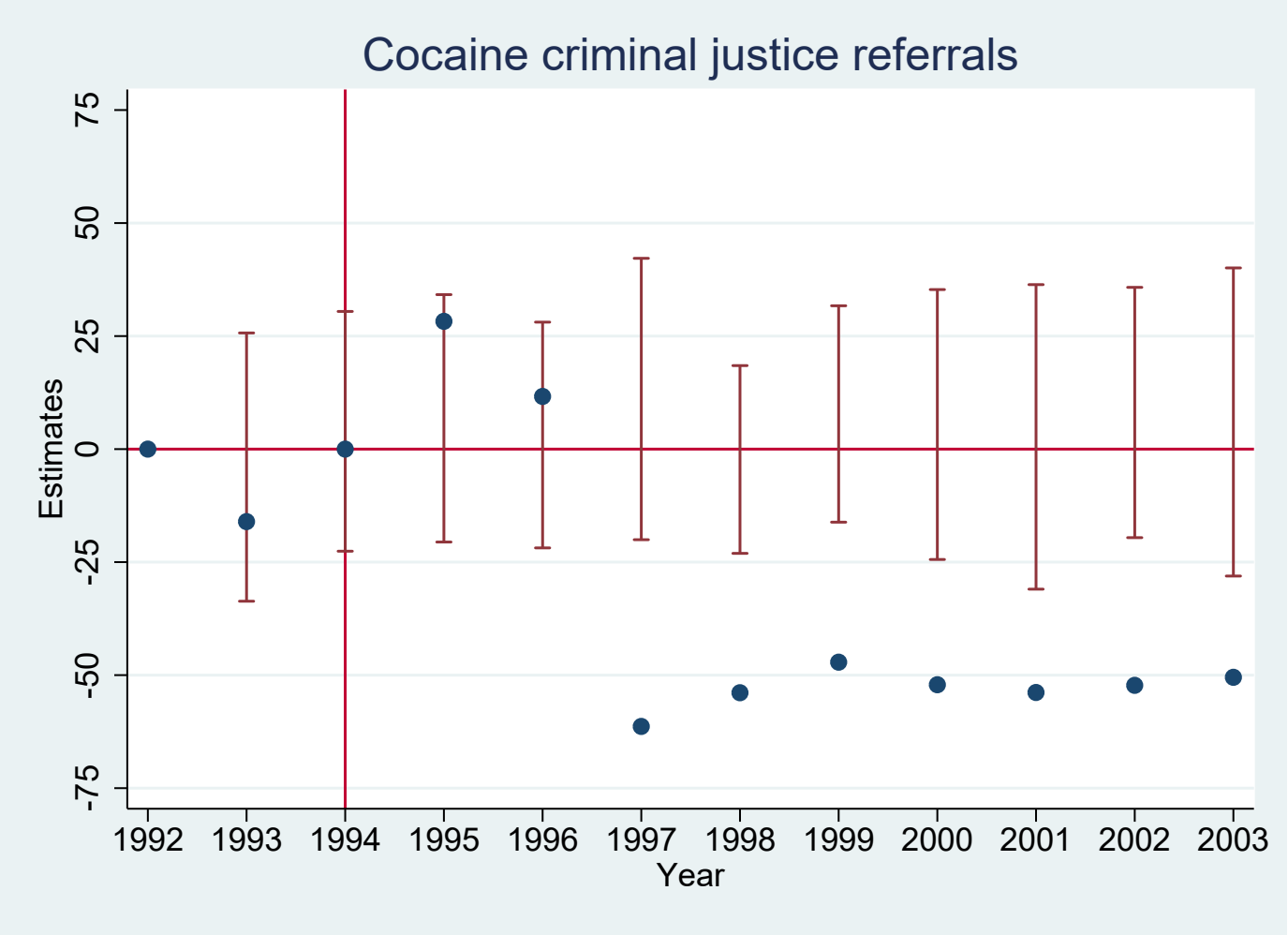

Figure 11 Event study plots of cocaine/crack criminal justice treatment referrals.

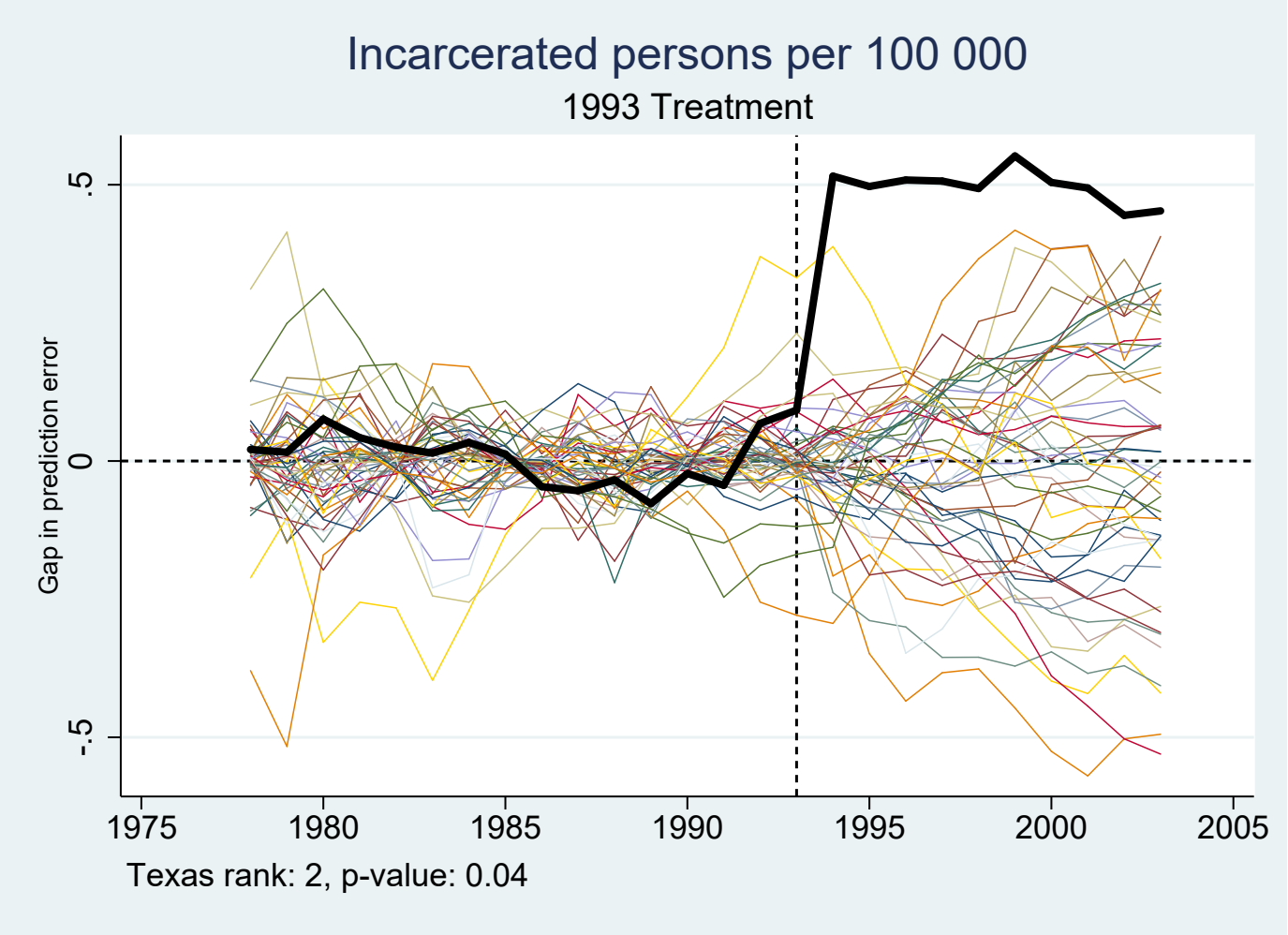

Figure 12 Synthetic control estimate of total incarceration per 100,000.

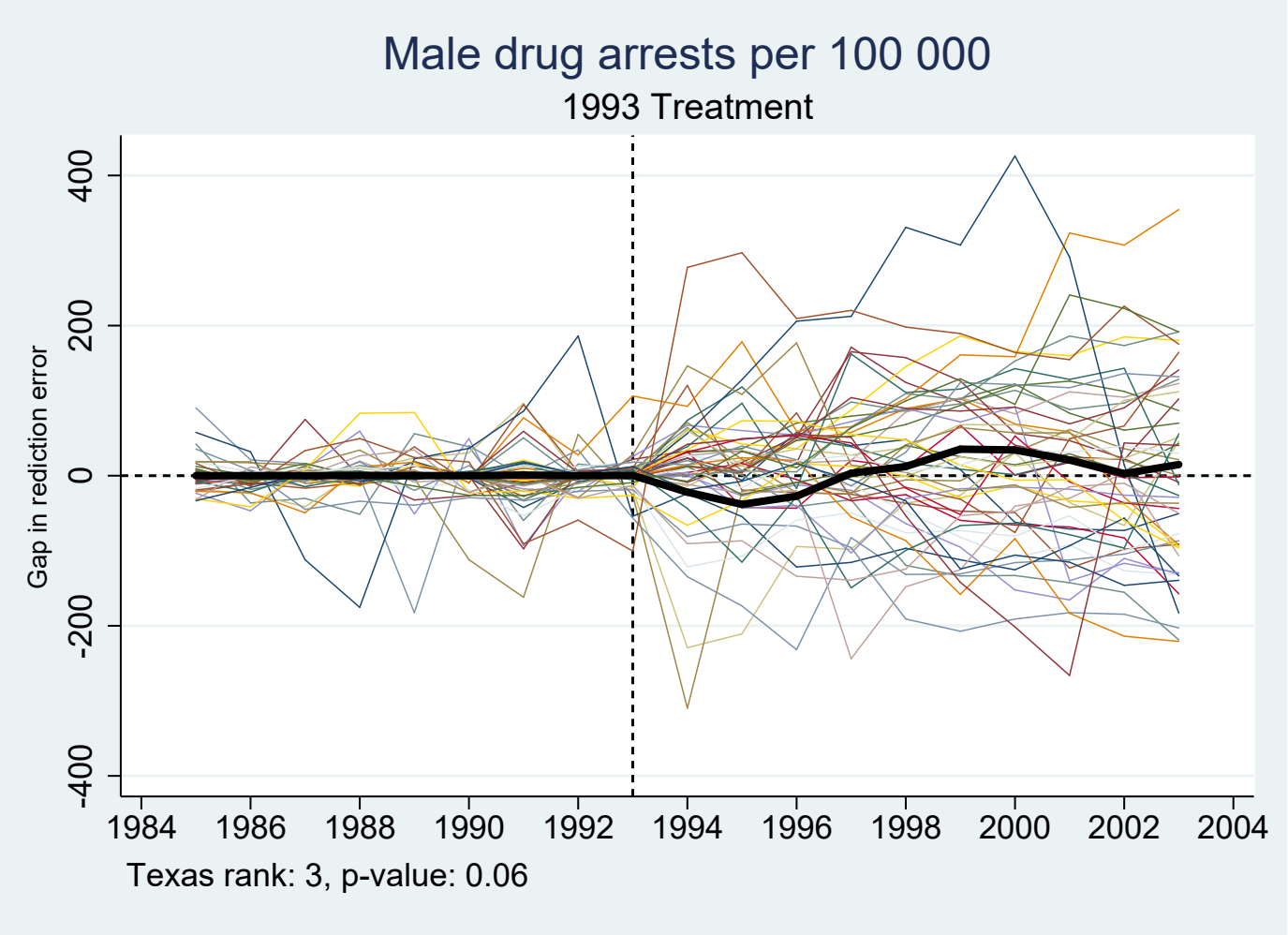

Figure 13 Synthetic control estimate of drug arrests per 100,000.

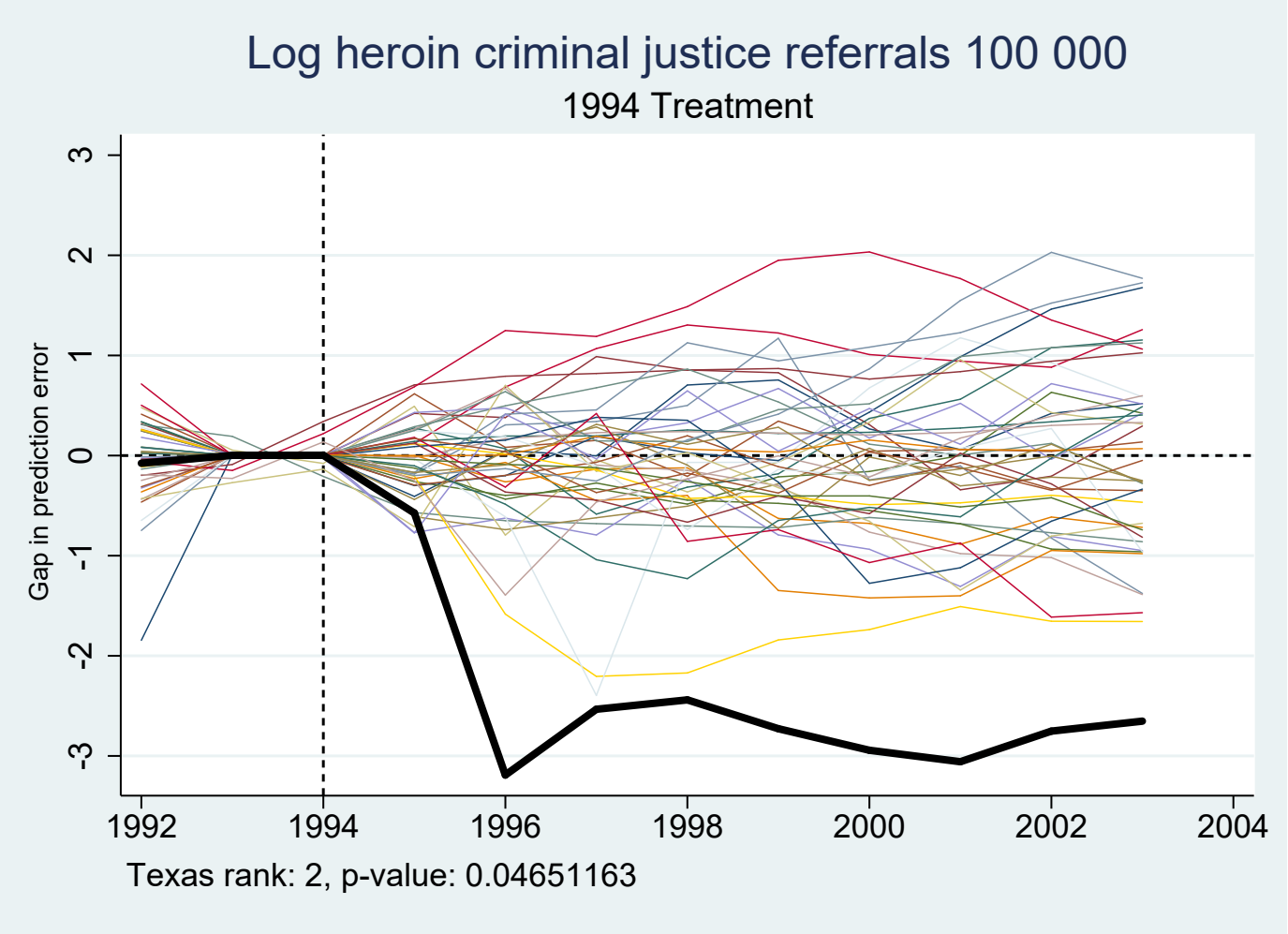

Figure 14 Synthetic control estimate of log heroin criminal justice referrals per 100,000.

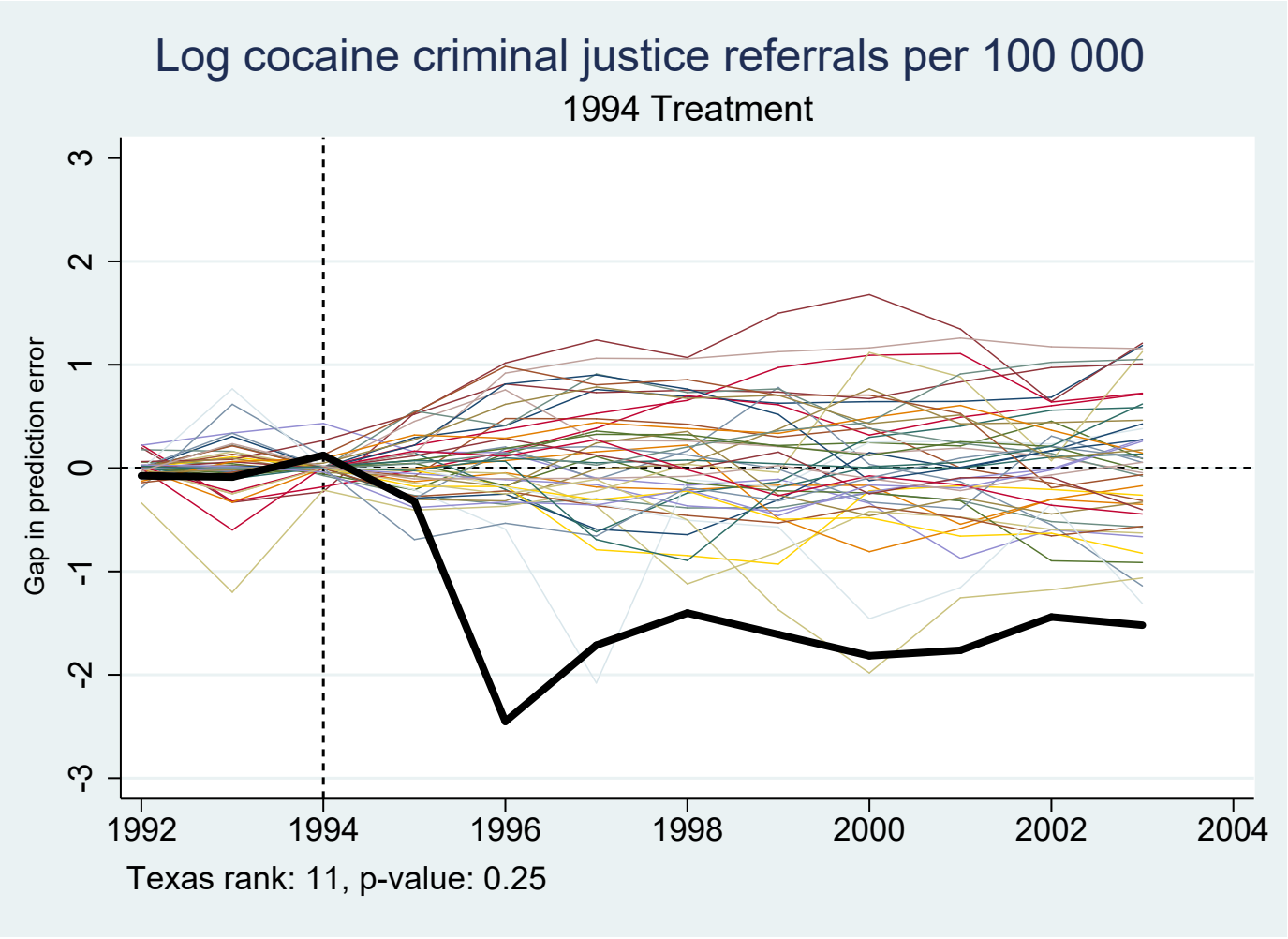

Figure 15 Synthetic control estimate of log cocaine criminal justice referrals per 100,000.