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The effect of drug prohibition on drug use, crime, and death: evidence from the U.K. Psychoactive Substances Act 2016

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This study investigates the effect of drug prohibition on drug use, violence, theft, and death from drug use. In 2016 the U.K. introduced a new law that effectively banned all psychoactive substances, including New Psychoactive Substances (NPS), a class of drugs that mimic the effect of established illicit drugs but are itself not prohibited. I used a combination of both conventional difference-in-differences and synthetic difference-in-differences on a panel of 13 European countries to analyze the effect of this new law. I show that while drug use decreases, violence and theft experience a proportionally larger increase, following the introduction of the new law. No conclusions could be drawn regarding deaths from drug use due to a persistent violation of the identifying assumption.

In June 1971, President Nixon of the United States officially declared a “war on drugs” and labeled drug abuse as “public enemy No. 1” (NPR, 2007). The aims of this war included the reduction of drug use and addiction, improving public health and safety, and the reduction of drug-related crime, by strongly enforcing the prohibition of drugs. A stance that many countries around the world adapted. In 2018 alone 1 billion dollars were spent on enforcing drug prohibition globally (Global Commission on Drug Policy, 2021). However, despite this tremendous effort, the war on drugs seems far from over. In fact, by most metrics the situation only seems to have deteriorated since the war has started. The worldwide production of illegal opioids has increased by 950% from 1980 till 2018 (Global Commission on Drug Policy, 2021). Moreover, between 2010 and 2015 drug-related deaths have increased by 60% from around 105,000 to 168,000 (Global Commission on Drug Policy, 2021). By these statistics the current drug policy appears to be largely unsuccessful.

Arguments against prohibition by economists have been around for as long as the war on drugs itself, as evidenced by a 1972 *Newsweek* column by economist and Nobel laureate Milton Friedman. In the column, Friedman labels prohibition as “an attempted cure that makes matters worse—for both the addict and the rest of us,” and argues that while it is unclear that legalizing drugs increases the number of addicts, it would lower crime rates and improve the

quality of law enforcement. The academic literature too predicts that prohibition might have limited success in decreasing drug use and could have the unintended side effects of increasing crime, both violent and economic, and increasing the number of accidental poisonings and overdoses (Miron & Zwiebel, 1995; Cussen & Block, 2000; Boettke et al., 2012). However, casual evidence of such relationships has been severely lacking. Likely because randomized experiments to document the effects of prohibition are virtually impossible and lacking transitions from legal to illegal drug markets have prevented the use of natural experiments.

The question thus remains what the causal effects of drug prohibition are on drug use, crime, and deaths due to drug use. To answer that question, this paper explores a unique transition of a drug market from legal to illegal. In 2016, the U.K. introduced the Psychoactive Substances Act (PSA), officially making the production, supply, and possession of any substance intended for human consumption that can produce a psychoactive effect an offense, with a maximum sentence of 7 years in prison. Exclusions from this law include food, alcohol, tobacco, nicotine, caffeine, and medical products (U.K. government, 2015). The PSA was specifically targeted at New Psychoactive Substances (NPS), a class of drugs characterized by their chemical structure that mimics the effect of illicit drugs but is constantly altered by manufacturers to avoid the law, therefore often referred to as 'legal highs'.

I use a difference-in-differences (DID) approach to analyse the effect of the PSA on drug use disorder rates, homicides rates, theft rates, and death rates due to drug use disorder, using other European countries as a control group for the U.K. I supplement these results with a synthetic difference-in-differences (SDID) approach to account for an apparent lack of parallel trends for drug use disorder rates and to formalize the selection of the control group. The analyses show that while the rate of drug use disorders decreased, homicides rates and theft rates increased to a proportionally larger degree. Moreover, these results were robust to an array of robustness checks. On the other hand, no conclusions with regards to the rate of death due drug use disorders could be drawn from the analyses due to a persistent violation of the parallel trends assumption.

Much of the current literature on drug policy takes a theoretical approach. For example, Miron and Zwiebel (1995), Cussen and Block (2000), and Boettke et al. (2012), predict the effect of prohibition on drug use and its potential negative externalities, while Becker et al. (2004) show that prohibition is likely inefficient using a simple supply and demand model. Most of the empirical literature relevant to the topic focusses on other illegal markets but not on the drug market directly. For example, Miron and Zwiebel (1991) and Owens (2011, 2014) study the U.S. Alcohol Prohibition, while Chimeli and Soares (2017) study the illegal

mahogany market in Brazil. Silverman and Spruill (1977) do study the drug market directly but are limited to studying the effect of price changes. This study fills the gap in the literature by directly documenting the causal effects of drug prohibition.

The remainder of the paper is structured as follows. Section I provides the necessary background information on the PSA. Section II discusses the relevant literature to form the hypotheses for this research. Section III describes the data collection process, the sources used to collect the data, and the sample that is used. Section IV explains the empirical strategy. Section V presents the results of the analyses. Finally, Section VI disuses the results and Section VII concludes the paper.

I. Background

The term 'NPS' was first coined by the U.K. Advisory Council on the Misuse of Drugs (ACMD). In their 2011 report on Novel Psychoactive Substances, they define NPS as: "Psychoactive drugs which are not prohibited by the United Nations Single Convention on Narcotic Drugs or by the Misuse of Drugs Act 1971, and which people in the UK are seeking for intoxicant use." More broadly the Alcohol and Drug Foundation (ADF) describes NPS as a range of drugs designed to mimic the effect of established illicit drugs, of which the chemical structure is constantly altered by manufactures to try to stay ahead of the law (2022). Because of these features, NPS are widely referred to as 'designer drugs' or 'legal highs.' NPS are most popular among a younger audience and are primarily sold online and through so called 'head shops', which are retail outlets specialised in selling legal recreational drugs (Deen et al., 2021). The most common type of NPS are stimulants, followed by synthetic cannabinoids. However, NPS exist for most classes of drugs including hallucinogens and synthetic opioids (UNODC, 2020). As of 2020 more than a thousand different NPS have been recorded (UNODC, 2020).

The highly adaptive nature of this market poses a challenge for law enforcement authorities seeking to keep up with the introduction of these new drugs. In response to these challenges on a global level, the United Nations Office on Drugs and Crime (UNODC) established the Early Warning Advisory (EWA) on NPS in 2013. The goal of the EWA is to monitor, analyse and report global and regional trends on NPS, as an input for evidence-based policy responses (UNODC, 2020). Alongside the EWA, many countries responded with policy changes on a national level. Most notably, the U.K. introduced the PSA in 2016, which made the production, possession with intent of supplying, supplying, offering to supply, and importing or exporting of psychoactive substances an offence. Under the act psychoactive

substances are defined as: “Any substance which is capable of producing a psychoactive effect in a person who consumes it, and is not an exempted substance” (Home Office, 2018). According to a Home Office (2018) review, the PSA led to head shops no longer selling NPS or closing entirely, thereby ending the open sale of NPS. Nevertheless, the report recognizes that the sale of NPS has not ceased entirely and was partly adopted by street dealers.

While no other European country introduced a blanket ban on new psychoactive substances, like the U.K., it is worth mentioning that most countries adopted policies aimed specifically at NPS. Of particular importance to this study are countries that introduced major policy changes coinciding with the introduction of the PSA. Germany and Sweden in 2016, and Belgium in 2017, all changed their drug policy from a substance-by-substance approach to a generic model, which allows for the classification of drugs into groups based on their core chemical structure and ban them as a single group (Neicun et al., 2019; Neicun et al., 2022). Ireland in 2016, and France in 2017 both introduced similar generic models, but also maintained individual listings, allowing for a ban of both families of NPS as well as specific substances (Neicun et al., 2022).

II. Theoretical framework

A. *Supply and demand*

The case for drug prohibition critically rests on its ability to reduce the consumption of drugs. From an economic perspective this argument can be supported by viewing prohibition as imposing additional variable costs on suppliers, so that the costs of supplying drugs rise. These costs include fines and other punishments when apprehended, but also costs incurred for evading apprehension (Miron & Zwiebel, 1995). Similarly, we can view prohibition as shifting demand downwards due to the cost imposed on users. These costs can exist for example of increased inconveniences in acquiring and using drugs or the risk of punishment for possession (Miron & Zwiebel, 1995). Miron and Zwiebel (1995) argue that the costs imposed on consumers are most likely smaller than those imposed on suppliers. Hence, prohibition is likely to cause a substantial upward shift in supply and a smaller downward shift in demand. Therefore, they recognize that, unless demand is far more elastic than supply, under prohibition consumption of drugs will decrease and prices will increase.

The view that prohibition is likely to have a larger effect on the supply side than the demand side is confirmed by the research of Félix and Portugal (2017), who investigate the decriminalization of drug use in Portugal. If prohibition initially imposed substantial costs on

the buyers, we would expect that decriminalization eliminates these costs to a large degree. In turn this would shift the demand curve upwards and, assuming supply stays constant, lead to an increase in prices. Félix and Portugal (2017) find no such increase in prices following decriminalization. Hence, it seems that prohibition influences consumption mainly through the supply side, even when drug use is not decriminalized.

However, the evidence of prohibition increasing costs of suppliers and thereby lowering consumption is not obvious either. For example, Poret (2022) points to the 1980's in the U.S., a period in which the cocaine and heroin trade was becoming increasingly risky, involving severe penalties, while at the same time drug supply increased and prices declined. To explain these seemingly paradoxical events, Poret (2022) builds a theoretical model that investigates the effect of increased law enforcement on drug use. He recognized that traditional analysis of the effect of increased enforcement on the drug market ignores strategic effects which can interfere with the law enforcement policy. Such strategic effects include adapting the number of transactions and the quantity exchanged at the transaction. By including these effects in his model, Poret (2022) concludes that harsher drug law enforcement may in some instances lead to an increased number of consumers and lower prices.

Even if prohibition is successful in raising prices and lowering demand, it is not necessarily clear that it is also efficient. Becker et al. (2004), study a simple supply and demand model in which they assume consumer prices do increase under prohibition. They find that with inelastic demand, total production costs rise as consumption falls, and enforcement costs rise more rapidly. Based on this they conclude that prohibition is only justifiable in the case that marginal social value of drug use is very negative.

From the theory it seems that prohibition will only decrease the consumption of drugs marginally. It seems that prohibition will not always be able to raise consumer prices extensively, and even if prices rise, the inelastic demand for drugs will prevent large decreases in consumption. This is further supported by empirical evidence from the U.S. alcohol Prohibition, which initially caused a sharp decrease in alcohol consumption to 30% of the pre-Prohibition level, only to be restored to around 60-70% of the pre-Prohibition level in the following years (Miron & Zwiebel, 1991). This decrease in consumption can be considered modest given that alcohol prices rose nearly three-fold. In the case of the PSA in the U.K. we can expect the initially sharp decrease in total drug consumption to be absent as the infrastructure for selling drugs illegally has already been established. Regardless, the theory predicts a negative effect of prohibition on drug use. Hence, my first hypothesis can be stated as follows:

Hypothesis 1. The PSA will lead to a decrease in total drug consumption in the U.K.

I test this hypothesis by estimating the effect of the PSA on the share of the population with a drug use disorder.

B. *Negative externalities*

So far, I have discussed the effects of prohibition on the level of drug consumption, mainly through supply and demand. However, though reducing drug consumption can be considered the main goal of prohibition, it is not the only factor should be considered when evaluating the effectiveness of the policy. There are potential negative externalities that could be a direct result of prohibition, which might outweigh any reductions in consumption it achieves. Miron and Zwiebel (1995) identify three potential externalities: increased levels of violence, increased economic crime, and increased accidental poisonings and overdoses. I will discuss these externalities in detail below.

Prohibition, according to Miron and Zwiebel (1995) is likely to increase the marginal benefits to violence and decrease the marginal costs. The reasoning is as follows, participants of an illegal trade cannot rely on the legal system to resolve disputes, hence the marginal benefits of using violence to resolve disputes rises. Moreover, evading apprehension for trading drugs, is complementary to evading apprehension for committing violent acts, hence the marginal cost of using violence decreases for participants in the drug trade. Another reason why violence might increase, according to Miron and Zwiebel (1995), is that prohibition is likely to promote the formation of cartels. Cartelization introduces the possibility of profits, and hence the marginal benefit of using violence to discourage small competitors from entering increases.

These theoretical predictions are supported by several empirical findings. Chimeli and Soares (2017) find that the shutdown of the legal market for mahogany in Brazil led to establishment of an illegal market and an increase in violence. Similarly, Kronick (2020) finds that an expansion of drug trafficking in Venezuela led to disproportionate increases in violence in municipalities along the trafficking route. On the other hand, Owens (2011) did not find evidence supporting that the U.S. Alcohol Prohibition led to increased violence. However, these results could be driven by unreliable data. Moreover, the effect of illegalizing alcohol on violence is ambiguous if alcohol consumption induces violence, i.e., the increases in market-

based violence could be offset by the reduction in violence following reduced alcohol consumption. Recognizing this fact, Owens (2014) finds evidence that the U.S. Alcohol Prohibition did increase market-based violence. Based on the theoretical predictions and empirical evidence, the second hypothesis can be stated as follows:

Hypothesis 2. The PSA will lead to increased violence in the U.K.

I test this hypothesis by estimating the effect of the PSA on the number of police recorded homicides per hundred thousand inhabitants.

Apart from increasing violence, Miron and Zwiebel (1995) theorize that prohibition might also increase the level of economic crime. More specifically, if drug users have inelastic demand for drugs, face binding liquidity constraints, and cannot obtain a legal income, than an increase in prices due to prohibition is likely to cause an increase in economic crimes. In line with these predictions are the findings of Silverman and Spruill (1977), who estimate that a 50% increase in heroin prices will result in property crime increasing by approximately 14% in Detroit. Moreover, they extrapolated their estimated elasticities to find that around 30% of the property crime in Detroit is committed to help finance the consumption of heroin. Even though the authors recognize that this extrapolation is mathematically and scientifically unjustified, the result is remarkable close to that of Felson and Staff (2017). Using data from the Survey of Inmates of State and Federal Correctional Facilities, they too find that around 30% of property offenders engage in economic crime to help purchase illegal drugs for private use. However, it should be noted that it is not necessarily clear that drug users must resort to economic crime when drug prices increase. Because enforcement on prohibition is not likely to be uniform across all types of drugs, relative prices of drugs change. Therefore, apart from resorting to property crime to maintain current use, users have a myriad of options when prices increase which include lowering use to current income, quitting altogether, or substituting to a now relatively cheaper drug (Benson et al., 2001). On the other hand, Benson et al. (1992) provide an additional reason why economic crime might increase due to prohibition. Namely, they show that as resources are shifted away from controlling economic crimes to enforcing prohibition, the probability of arrest for economic crime falls and its rate increases. Based on the above, the third hypothesis can be stated as follows:

Hypothesis 3. The PSA will increase the level of economic crime in the U.K.

I test this hypothesis by estimating the effect of the PSA on the number of police recorded theft offences per hundred thousand inhabitants.

Finally, the economic literature predicts that prohibition of drugs will increase the number of accidental poisonings and overdoses. The explanation for this increase is twofold. Firstly, the quality of drugs will decrease because the purity of drugs cannot be guaranteed by government regulation under prohibition, less information sharing about products and vendors occurs given the underground nature of the market, and buyers are not able to address quality concerns without incriminating themselves (Miron & Zwiebel, 1995; Cussen & Block, 2000; Boettke et al., 2012). While some of these concerns might be mitigated by the reputation of certain sellers through repeated transactions, this is likely only a partial solution (Miron & Zwiebel, 1995). The second reason why the number of accidental poisonings and overdoses might increase is referred to as the "potency effect". Given the risks of transporting illegal drugs, it is in the best interest of the suppliers to sell more potent drugs because of its greater value per unit (Cussen & Block, 2000). Moreover, since prohibition increases prices, more potent drugs become relatively cheaper compared to less potent drugs, hence demand for them increases (Boettke et al., 2012). Based on these predictions, the final hypothesis can be stated as follows:

Hypothesis 4. The number of deaths from drug use in the U.K. will decrease to a lesser degree, following the PSA, than the number of drug users will decrease.

I test this hypothesis by estimating the effect of the PSA on the estimated number of deaths from drug use disorders per hundred thousand inhabitants.

III. Data

To examine the effect of the PSA in the U.K., a sample of countries relatively like the U.K. is required. Therefore, I limited the sample to Northern, Western, and Southern European countries (according to the EuroVoc division of Europe). In total I was able to collect data on a panel of 18 countries. As discussed in Section I, however, some countries introduced major changes to their drug policy coinciding with the introduction of the PSA and are likely to bias the results. For this reason, I deleted Germany, Sweden, Belgium, Ireland, and France from the sample, leaving me with a final sample of 13 countries. Moreover, the SDID method requires a reasonable number of pre-treatment periods. For the outcome variables on drug use and all

the covariates, I was able to collect data from 1991 onwards, resulting in a total of 25 pre-treatment periods. For the outcome variables on crime the data was scarcer, and I was only able to collect data from 2008 onwards, resulting in a total of 8 pre-treatment periods. For the post-treatment period I was limited to using data until 2019, since 2020 was the first year in which the COVID-19 virus could have had large differentiating effects on the countries in my sample. This resulted in a total of 4 post-treatment periods.

The first source that I used to construct my sample is Our World in Data, an online data portal that is produced by the Oxford Martin Programme on Global Development at the University of Oxford. Our World in Data publishes data analyses and visualizations on a wide range of topics like poverty, disease, hunger, climate change, war, existential risks, and inequality. For this research, I used Our World in Data to collect data on the outcome variables, the death rate from drug use disorders and the share of the population with a drug use disorder, and on the covariate, the share of the population with an alcohol use disorder. The underlying source that Our World in Data used to collect this data, is the Global Burden of Disease Study 2019 (GBD 2019), that was published by the Institute for Health Metrics and Evaluation (IHME).

The second source that I used to collect data is Eurostat, the statistical office of the European Union. Eurostat uses its partnerships with National Statistical Institutes and other national authorities to produce statistics on EU Member States. I used this source to collect data on the outcome variables theft rate and murder rate. However, the data I collected from Eurostat was not complete. The theft rate data of 2018 and 2019 were missing for the U.K. at the time of collection. I had to substitute this data using the database from the U.K.'s Office for National Statistics. Moreover, the murder rate data was missing for some years for certain countries. This data was substituted using data from the United Nations Office on Drugs and Crime (UNODC).

The final two sources I used for this research are the databases from The World Bank and the Organisation for Economic Co-operation and Development (OECD). Both The World Bank and the OECD are international co-operations that collect data on their member countries. From their databases I collected data on the following covariates: GDP per capita, age of the population, unemployment rate, and alcohol consumption per capita.

IV. Methodology

The model I use to estimate the effect of the PSA, is a difference-in-differences (DID) model, a popular method used by social scientist for causal effect estimation in non-experimental settings (Roth et al., 2023). The base model I estimate is given by equation (1):

$$Y_{it} = \alpha_i + \gamma_t + \beta \text{Treatment}_{it} + \varepsilon_{it} \qquad (1)$$

Where the subscript $i = 1, \ldots, N$ references each country in the sample and the subscript t denotes each year. Y_{it} refers to one of the four outcome variables: Drug use$_{it}$ indicates the share of the population with a drug use disorder; Theft$_{it}$ indicates the number of police recorded theft offences per hundred thousand inhabitants; Homicide$_{it}$ indicates the number of police recorded homicides per hundred thousand inhabitants; Drug death$_{it}$ indicates the estimated number of deaths from drug use disorders per hundred thousand inhabitants. Treatment$_{it}$ is a binary variable that takes value 1 when the country is the U.K. and when the year is in the post-treatment period. The variable takes value 0 otherwise. β is the main coefficient of interest and captures the treatment effect. The terms α_i and γ_t capture the full set of country dummies and year dummies respectively. Finally, ε_{it} is the error term. The treatment effect is estimated using ordinary least squares (OLS). Formally, the estimator can be defined as equation (2):

$$\widehat{\beta} = \text{argmin}_{\beta}\left\{\sum_{i=1}^{N}\sum_{t=1}^{T}(Y_{it} - \alpha_i - \gamma_t - \beta \text{Treatment}_{it})^2\right\} \quad (2)$$

While estimation of the treatment effect is straight forward using OLS, statistical inference is more complicated. From Figure 1, it seems the outcome variables are non-stationary, leading to potential serial correlation in the residuals. In fact, Bertrand et al. (2004) suggest that in many published papers conventional DID standard errors might be severely understated because of autocorrelation. The literature suggests several solutions including block bootstrapping the standard errors, aggregating the data, and clustering the standard errors at the group level (Bertrand et al., 2004; Cameron et al., 2008; MacKinnon & Webb, 2017). Unfortunately, none of these methods perform well with instances of a single treated unit, as is the case in this study. Roth et al. (2023) suggest that in some cases it is possible to base inference on Fisher Randomization Tests (FTRs), otherwise known as permutation tests. The critical assumption of FTRs, however, is random assignment of treatment, which is

questionable in this study. Instead, I do cluster the standard errors at the country level as this seems to be more conservative than using conventional robust standard errors but note that these standard errors might still be understated. Therefore, the results of the DID should mainly be interpreted for their point estimates and not their statistical significance.

The crucial identification assumption for DID, is the parallel trends assumption. Meaning that the approach will offer unbiased estimates of the effect of the PSA when the U.K. and the counterfactual group have common trends in the outcome variables absent of the PSA. A natural starting point for a control group is to use the rest of the European countries. From Figure 1 we can see graphically that the average trends of this group relatively closely resemble those of the U.K., at least for theft and homicide. However, the trends in the drug use disorder rate and in the number of deaths from drug use disorders seem to be less similar between the two groups. An alternative selection criterion for the control group used by some previous authors is to use a geographically chosen control group (Card, 1990; Card & Krueger, 1993; Sabia et al., 2012; DeAngelo & Hansen, 2014). For this study France and Germany would be great candidates for a geographic control because of their relative proximity and similar size of population and GDP to the U.K. However, as stated before both France and Germany had to be excluded from the sample due their changes in drug policy that could bias the results.

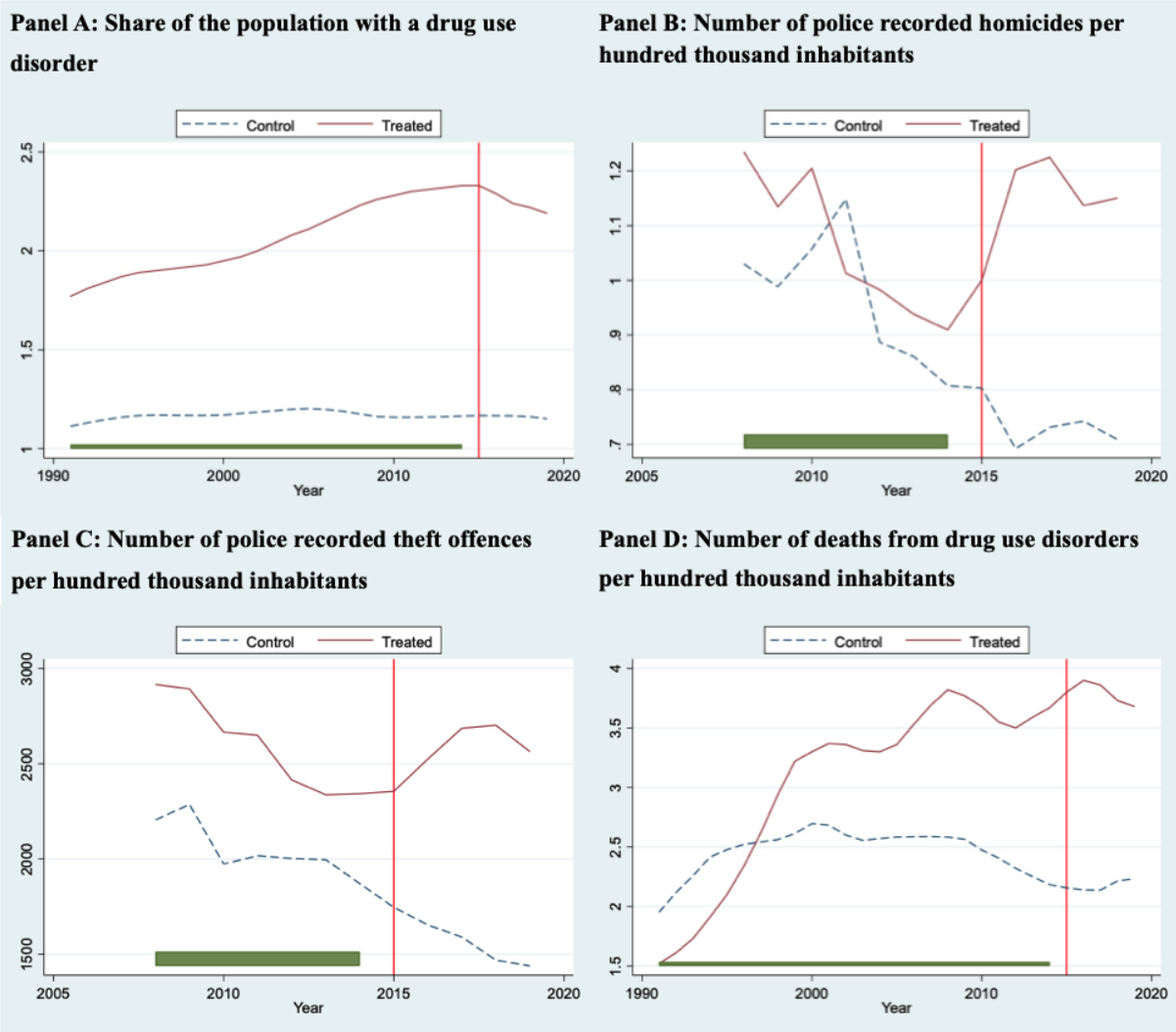

Figure 1. DID trends for U.K. and all other countries. *Notes:* The green shaded area represents the time weights attributed to each period, which are identical for each period when using conventional DID.

A. *Synthetic difference-in-differences*

Another method for choosing a suitable control group that has become popular is to use a data driven approach. The first of such methods, the Synthetic Control (SC) method, was introduced by Abadie and Gardeazabal (2003), and Abadie et al. (2010). This method accounts for a potential lack of parallel trends, by weighing the non-treated units to match the pre-treatment trend of the treated unit(s). However, even this method has its flaws. Specifically, if the treated unit is extreme in the values of the outcome variable in the pre-treatment period, it is not possible to create a weighted average of untreated units that can approximate the pre-treatment trend of the outcome variable for the treated unit (Abadie, 2021). In other words, the pre-treatment trends of the U.K. in the outcome variables should be bounded within the pre-

treatment trends of the donor pool of control countries for this method to be valid. Figure 2, Panel A shows that the trend of the drug use disorder rate for the U.K. is extreme, hence, this method cannot be used.

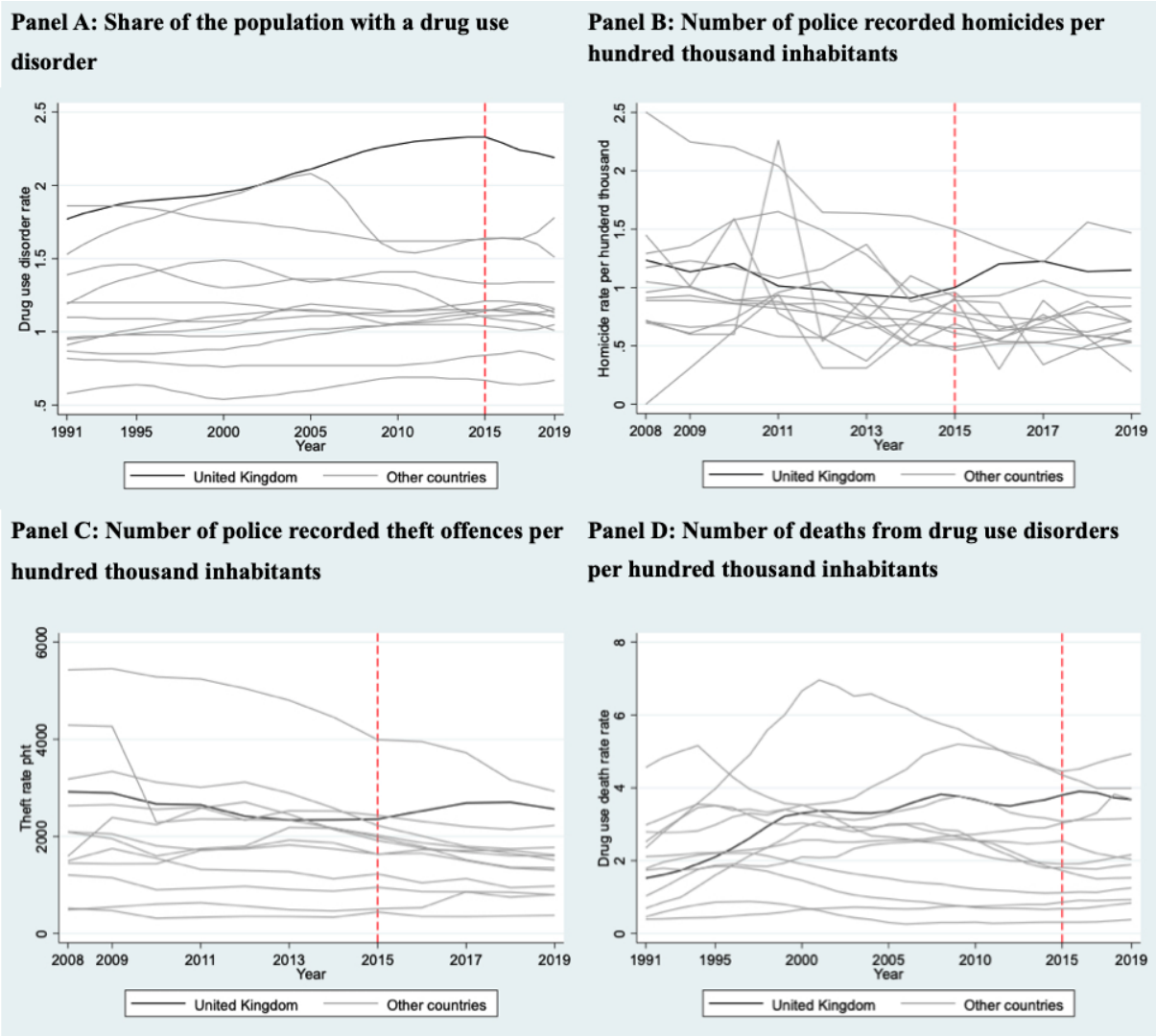

Figure 2. Time trends for the U.K. and all other countries

Fortunately, a new data driven method, Synthetic Difference-in-Differences (SDID), was introduced by Arkhangelsky et al. in 2021. This new method allows for an intercept, which means that the unit weights no longer need to be constructed in such a way that the untreated pre-trends perfectly match the treated ones (Arkhangelsky et al., 2021). Moreover, the authors claim that their proposed method is competitive with (or dominates) both traditional DID and SC. Therefore, I use this method next the traditional DID to formalize the selection of the control group and to account for the apparent lack of parallel trends for the drug use disorder

rate and the number of deaths from drug use disorders when using all European countries as the control group.

SDID estimates the effect of treatment exposure by solving the same two-way fixed effects regression problem as defined in equation (2) for the DID but adds time and unit weights, as seen in equation (3):

$$\hat{\beta} = \text{argmin}_{\beta}\{\sum_{i=1}^{N}\sum_{t=1}^{T}(Y_{it} - \alpha_i - \gamma_t - \beta \text{Treatment}_{it})^2 \hat{\omega}_i \hat{\lambda}_t\} \quad (3)$$

The unit weights $\hat{\omega}_i$ are found so that they align pre-treatment trends in the outcome of control units with those for the U.K., while the time weights $\hat{\lambda}_t$ are found so that they balance pre-treatment time periods with post-treatment ones. The precise algorithm for finding the unit and time weights is formally defined in Arkhangelsky et al. (2021, algorithm 1). In their paper, Arkhangelsky et al. (2021) also show that the estimator is asymptotically normal, suggesting that confidence intervals on β can be constructed as:

$$\hat{\beta} \pm z_{\alpha/2}\sqrt{\hat{\mathbb{V}}_{\beta}} \quad (4)$$

Where $z_{\alpha/2}$ indicates the inverse normal density function at percentile α/2, and $\hat{\mathbb{V}}_{\beta}$ refers to the estimate of the variance of β. To compute this estimate of the variance, Arkhangelsky et al. (2021) propose three different methods. Of these, the placebo-based inference method is the only one suitable for instances of a single treated unit and hence will be used in for this study. The method estimates the variance based on a large set of placebo tests performed on the non-treated units, as laid out in Arkhangelsky et al. (2021, algorithm 4). Importantly, the standard errors derived from this method do not suffer from the same concerns of serial correlation as with the DID method.

Examining the parallel trends assumption, we can see in Figure 3, Panel A that the SDID method improves the parallel trends between the U.K. and the control group for the drug use disorder rate considerably compared to the DID method. Moreover, we can see in Panel B and Panel C that SDID, like DID, can produce relatively parallel trends for homicide and theft. However, Panel D shows that also like DID, SDID fails to produce parallel trends for the number of deaths from drug use disorders.

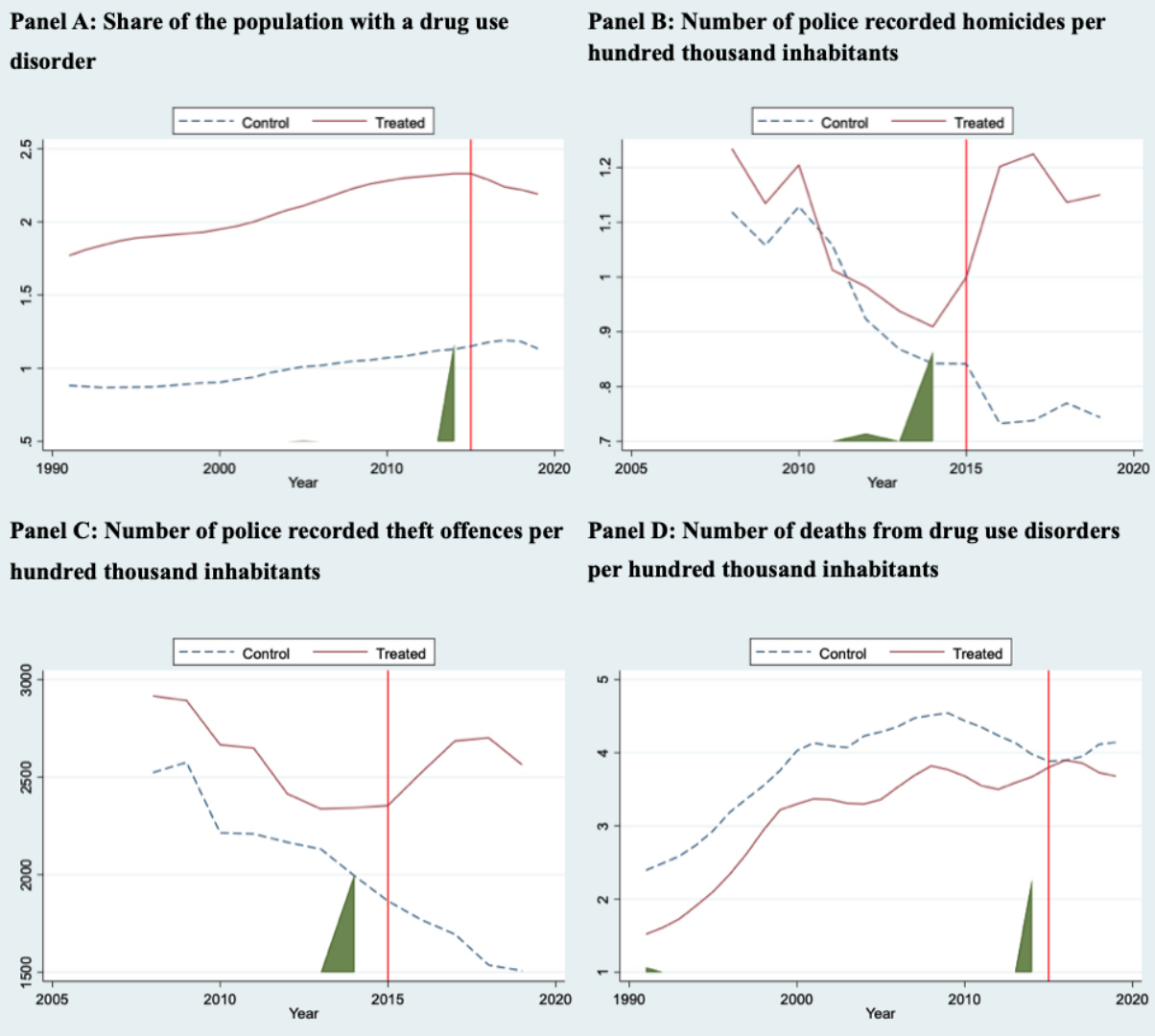

Figure 3. SDID trends for the U.K. and the synthetic control. *Notes:* The green shaded areas represent the time weights attributed to each period.

B. *Robustness checks*

Despite the graphical evidence from Figure 1 and Figure 3 that show relatively parallel trends for homicide and theft using both DID and SDID, and for the drug use disorder rate using SDID, one might be concerned that the treatment variables could be capturing differential pre-existing dynamics of the outcome variables between the U.K. and the control countries. Note that this would have to be the case conditional on both country fixed effects and time fixed effects. To address this concern, it is good practice to show the estimates are robust to the inclusion of covariates. Therefore, as a robustness check I include a set of covariates that consists of GDP per capita, the unemployment rate, the share of the population aged between

15 to 24 years, the share of population with an alcohol use disorder, and alcohol consumption per capita. For this robustness check the estimated model is given by the following equation:

$$Y_{it} = \alpha_i + \gamma_t + \beta \text{Treatment}_{it} + X'_{it}\delta + \varepsilon_{it} \qquad (5)$$

Where the vector X_{it} captures the full set of time-varying country specific characteristics and δ is a vector of coefficients. The model will be estimated using both DID and SDID. Note that the SDID method takes the covariates into account when constructing the synthetic control group. Hence, the trends of synthetic control produced by this specification differ slightly from those of the baseline model. The SDID trends of this specification are shown in Figure A1 in the appendix. Most notably, the trends for the drug use disorder rate seem less parallel to the U.K. than in the baseline model.

Apart from using a set of covariates, one might also consider adding lagged values of the dependent variable to the model. Considering this question, Roth et al. (2023) suggest including lagged outcomes could be sensible when the decision to take up treatment is based on lagged outcomes, which might be the case for the drug use disorder rate and the number of deaths from drug use disorders. However, this seems less likely for homicide and theft as both displayed downward trends pre-treatment. In fact, Daw and Hatfield (2018) advise against using lagged outcomes when there is no or a weak relationship between lagged outcomes and the take up of treatment. They show that doing so can induce bias due to a mean-reversion effect. Therefore, I include the first lag of the outcome for drug use disorder rate and the number of deaths from drug use disorders but not for homicide and theft. The relevant model is given by the following equation:

$$Y_{it} = \alpha_i + \gamma_t + \beta \text{Treatment}_{it} + X'_{it}\delta + \theta Y_{i(t\text{-}1)} + \varepsilon_{it} \qquad (6)$$

Where $Y_{i(t\text{-}1)}$ refers to the first lag of the dependent variable and θ is a coefficient. The model is estimated using both DID and SDID. The SDID trends for this specification are shown in Figure A2 in the appendix. The trends for the drug use disorder rate seem like those of the previous specification and are again less parallel to the U.K. than the baseline model.

Since the SDID method is relatively new, not a lot of literature has been written on the best practices in terms of robustness checks specifically for this method. However, given its similarities with the SC method, which has a much richer literature on robustness checks, I

argue that implementing the proposed checks for SC is a relevant exercise for SDID as well. I perform these robustness checks for the SDID on the baseline model, as including covariates considerably increases computational costs.

The first of these robustness checks that I will implement is called "backdating" in Abadie (2021) and refers to the procedure of artificially assigning an earlier treatment period. If successful, backdating can improve credibility in two critical ways. Firstly, one should expect an absence of estimated effects prior to the actual treatment period. If this is indeed the case, it demonstrates that the synthetic control group generated by the SDID is successful in producing a parallel trend in the outcome variable for the treated unit before the treatment period. Secondly, if the SDID generated a credible control group we should expect to see the trends of the treated unit and the control group only start to diverge after the actual treatment period, even when the treatment period is artificially backdated. When this can be observed graphically, it supports the validity of the SDID estimates. For this study I will backdate the treatment period by four years to 2012. Given that the data on theft and homicide only extends to 2008, I believe further backdating will lead to too little pre-treatment periods for the SDID to generate an accurate synthetic control group.

The second robustness check I will implement for the SDID method is referred to as the "leave-one-out" analysis (Abadie, 2021). Typically, in this type of analysis all the countries that make up the synthetic control group are removed from the donor pool one at a time. This ensures that the estimated effect is not driven in large part by one country. If there does exist a large change in the estimated effects by leaving out one country, without the exclusion rendering the pre-treatment trend no longer parallel, it reduces the credibility of the results. Particularly, it could mean the measured effect was driven by policy changes or by particularly large idiosyncratic shocks on the outcome of the excluded country (Abadie, 2021). It should be noted that the SDID method favours giving weight to a lot of countries, compared to the SC method. Therefore, I will restrict the leave-one-out analysis to the five countries that received the highest weight in the synthetic control group. The overview of the weights attributed to each country is provided in Table B1 in the appendix. An extension of the leave-one-out analysis would be to exclude all countries that make up the synthetic control group from the donor pool (Gilchrist et al., 2023), or in this case all five highest weighted countries. However, as my sample of countries is limited, this would likely underpower my research design.

Finally, it should be noted that the treatment variable is defined as the interaction between an indicator variable for the treatment group, the U.K., and a dummy variable that takes value one in the post-treatment period. Therefore, it might not exclusively capture the

effect of the PSA, but could potentially capture any policy change in the U.K. that might have influenced the outcome variables and coincided with the PSA. I therefore provide a review of the important policy changes in the U.K. that happened in 2016.

V. Results

In this section I estimate the effect of the PSA on the different outcome variables. First, I will examine the four hypotheses in order by estimating the model specified in the previous section using both DID and SDID. I then turn to examining the robustness of these estimates by performing both a backdating analysis and a leave-one-out analysis.

A. *Drug use disorder*

I start by evaluating Hypothesis 1, which states that the PSA will decrease drug use in the U.K. As mentioned before, the fraction of the population with a drug use disorder is used as a proxy for drug use. Whether this is a valid proxy will be further discussed in Section VI. I estimate equations (1), (5) and (6) defined in the previous section using both DID and SDID. Columns 1 and 2 of Table 1 present the results of the DID method using all countries in the sample expect the U.K. as a control group. Column 1 shows a positive and statistically significant effect of the PSA on drug use disorders, $\beta = 0.175$, $p = 0.001$, while using no controls. However, this result is likely driven by a violation of the parallel trends assumption as seen in Figure 1. In line with this explanation is the fact that with the inclusion of control variables the effect size decreases and is no longer significant, $\beta = 0.018$, $p = 0.804$, as shown in column 2. This indicates that most of the measured effect was likely driven by differing country specific time trends. Interestingly, Column 3 shows a negative and statistically significant result, $\beta = -0.061$, $p < 0.001$. This can possibly be explained by the inclusion of both controls and the lagged value relaxing the parallel trends assumption, making this result more representative of the true effect despite pre-trends not being perfectly parallel. Column 4 shows that the result of the SDID without covariates also indicates a negative and statistically significant effect of the PSA on drug use disorder, $\beta = -0.112$, $p < 0.001$. Moreover, As seen in Figure 3 the parallel trends assumption holds for this specification. When covariates are included to the specification, shown in Column 5, the effect size decreases and is no longer significant at the 10 percent level, $\beta = -0.066$, $p = 0.111$. However, column 6 shows a very

similar result that is significant at the 10 percent level, β = -0.063, p = 0.092, when the lagged outcome is added to the model. Moreover, Figure A1 and Figure A2 in the appendix show that both these specifications generated trends that were less parallel to the U.K. than the one generated by the specification without covariates. These results can be interpreted as the PSA decreasing the share of the U.K. population with a drug use disorder by 0.061 to 0.112. Given the pre-treatment share of the U.K. population with a drug use disorder, this constitutes a decrease of 3 to 5 percent. Therefore, the hypothesis that the PSA decreases drug use in the U.K. cannot be rejected.

Table 1. Effect of the PSA on drug use disorder

	Drug use disorder					
	All countries (w/o the U.K.)			Synthetic control group		
	(1)	(2)	(3)	(4)	(5)	(6)
Treatment	0.175***	0.018	-0.061***	-0.112***	-0.066	-0.063*
	(0.039)	(0.069)	(0.007)	(0.031)	(0.041)	(0.037)
Controls	No	Yes	Yes	No	Yes	Yes
Lagged outcome	No	No	Yes	No	No	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes	Yes	Yes
Observations	377	377	364	377	377	364
R-squared	0.949	0.963	0.982	N/A	N/A	N/A

Notes: The dependent variable is the fraction of the population with a drug use disorder. The sampling period goes from 1991 to 2019. The control variables include GDP per capita, the unemployment rate, the share of the population aged between 15 to 24 years, the share of population with an alcohol use disorder, and alcohol consumption per capita. Standard errors for column (1), (2), and (3) are clustered at the country level and are between brackets. Standard errors for column (4), (5), and (6) are based on placebo tests as explained in section IV and are between brackets. *** p<0.01, ** p<0.05, * p<0.1.

B. *Homicide*

Next, I turn to evaluating Hypothesis 2, which states the PSA will increase the level of violence in the U.K. As a proxy for violence, the number of police recorded homicides per hundred thousand inhabitants will be used. The validity of this proxy will be discussed in section VI, as with the proxy for drug use. I will evaluate Hypothesis 2 by estimating equations (1) and (5) using both DID and SDID. Column 1 of Table 2 shows a positive and significant effect of the PSA on homicide, β = 0.355, p < 0.001, while using all countries in the sample expect the U.K. as a control group. This result seems robust to the inclusion of controls, as seen in Column 2, β = 0.344, p = 0.002. The claim that these results are reflective of the true causal

effect is further supported by the relative parallel pre-treatment trends as seen in Figure 1. Columns 3 and 4 also show similar effect sizes when using a synthetic control group, $\beta = 0.311$, $p = 0.010$, without covariates, and $\beta = 0.274$, $p = 0.078$, with covariates. Considering that both specifications of the SDID generate a synthetic control with relatively parallel trends to the U.K., as seen in Figures 3 and Figure A1, it strengthens their claim of a causal effect. Overall, the four different specifications estimate similar effect sizes and are all statistically significant at the 1 and 10 percent level. We can interpret these results as the PSA increasing the number of police recorded homicides in the U.K. per hundred thousand inhabitants by 0.274 to 0.355. Given the pre-treatment level of homicides in the U.K., this constitutes an increase of 27-36 percent. Based on these estimates the hypothesis that the PSA increases the level of violence in the U.K. cannot be rejected.

Table 2. Effect of the PSA on homicide

	Homicide			
	All countries (w/o the U.K.)		Synthetic control group	
	(1)	(2)	(3)	(4)
Treatment	0.355***	0.344***	0.311***	0.274*
	(0.064)	(0.085)	(0.121)	(0.155)
Controls	No	Yes	No	Yes
Year FE	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes
Observations	156	156	156	156
R-squared	0.701	0.733	N/A	N/A

Notes: The dependent variable is the number of police recorded homicides per hundred thousand inhabitants. The sampling period goes from 2008 to 2019. The control variables include GDP per capita, the unemployment rate, the share of the population aged between 15 to 24 years, the share of population with an alcohol use disorder, and alcohol consumption per capita. Standard errors for column (1) and (2) are clustered at the country level and are between brackets. Standard errors for column (3) and (4) are based on placebo tests as explained in section IV and are between brackets. *** p<0.01, ** p<0.05, * p<0.1.

C. *Economic crime*

Next, I evaluate Hypothesis 3, which states that the PSA will increase the level of economic crime in the U.K. To evaluate this hypothesis, I will again estimate equation (1) and (5) using both DID and SDID, this time with the number of police recorded theft offences per hundred thousand inhabitants as the outcome variable. Columns 1 and 2 of Table 3 show the DID results

using all countries except the U.K. as the control group. The specification in Column 1 uses no controls and presents a positive and statistically significant effect, $\beta = 521.50$, $p = 0.010$. The effect size increases somewhat when control variables are added and remains statistically significant, $\beta = 731.25$, $p = 0.031$, as presented in Column 2. As seen in Figure 1 the trend of the control group is relatively parallel to that of the U.K., supporting the estimate as being reflective of the causal effect. The estimated effect size of the SDID specification without covariates is very similar to that of the DID specification without covariates, $\beta = 543.24$, $p = 0.060$, as shown in Column 3. Again, when covariates are added to the specification in Column 4, the effect size increases somewhat and remains statistically significant, $\beta = 597.13$, $p = 0.019$. The trends of both synthetic controls are parallel to that of the U.K. to a similar degree, as shown in Figure 3 and Figure A1. Overall, the estimated effect size remains reasonably stable over the four specifications and the effect is statistically significant in all four specifications. We can interpret these results as the PSA increasing the number of police recorded theft offences per hundred thousand inhabitants by 521.50 to 731.25. In respect to the pre-treatment level of theft offences in the U.K., this constitutes a 22-31 percent increase. These results provide evidence in favour of the hypothesis that the PSA increases the level of economic crime in the U.K.

Table 3. Effect of the PSA on theft

	Theft			
	All countries (w/o the U.K.)		Synthetic control group	
	(1)	(2)	(3)	(4)
Treatment	521.496***	731.248**	543.237*	597.129**
	(169.642)	(300.084)	(289.222)	(253.699)
Controls	No	Yes	No	Yes
Year FE	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes
Observations	156	156	156	156
R-squared	0.903	0.923	N/A	N/A

Notes: The dependent variable is he number of police recorded theft offences per hundred thousand inhabitants. The sampling period goes from 2008 to 2019. The control variables include GDP per capita, the unemployment rate, the share of the population aged between 15 to 24 years, the share of population with an alcohol use disorder, and alcohol consumption per capita. Standard errors for column (1) and (2) are clustered at the country level and are between brackets. Standard errors for column (3) and (4) are based on placebo tests as explained in section IV and are between brackets. *** p<0.01, ** p<0.05, * p<0.1.

D. *Death from drug use*

Finally, I evaluate Hypothesis 4, which states that the PSA will decrease death from drug use less than the PSA will decrease drug use. As with Hypothesis 1, Hypothesis 4 is evaluated by using both DID and SDID to estimate equation (1), (5) and (6). The estimated number of deaths from drug use disorders per hundred thousand inhabitants is used as the outcome variable. Column 1 of Table 4 presents the results of the DID specification using all countries except the U.K. as a control and without control variables, and shows a positive and statistically significant effect, $\beta = 0.988$, $p < 0.001$. However, like the DID analysis of Hypothesis 1, this result is most likely driven by a violation of the parallel trends assumption, as we can see in Figure 1. When controls variables are added to the specification in Column 2, the effect size decreases considerably and is no longer statistically significant at the 10 percent level, $\beta = 0.532$, $p = 0.439$. Again, like the DID analysis of Hypothesis 1, when the lagged outcome is included in the Column 3, the effect becomes negative and statistically significant, $\beta = -0.237$, $p = 0.005$. However, while the inclusion of the lagged value relaxes the parallel trends assumption, it should be noted that the DID trends are wildly different between the U.K. and the other countries, as seen in figure 1. Moreover, including the lagged value in the SDID specification gives a positive and statistically insignificant result, $\beta = 0.048$, $p = 0.241$, as shown in Column 6. Figure A2 shows that the control group produced by this specification has a different trend than that of the DID but again is not parallel to the U.K. Columns 4 and 5 do show similar results for the SDID specifications with and without covariates and no lagged outcome. Both estimates show a small positive effect that is insignificant at the 10 percent level, $\beta = 0.135$, $p = 0.584$, and $\beta = 0.114$, $p = 0.724$, respectively. However, as seen in Figure 3 and Figure A1 neither of these specifications manages to produce a control group with a credible parallel trend to that of the U.K. Ultimately, the parallel trends assumption is violated in all six specification and therefore no conclusion can be drawn from these results with regards to the hypothesis that the PSA will decrease death from drug use less than the PSA will decrease drug use.

Table 4. Effect of the PSA on death from drug use disorder

	Death from drug use disorder					
	All countries (w/o the U.K.)			Synthetic control group		
	(1)	(2)	(3)	(4)	(5)	(6)
Treatment	0.998***	0.523	-0.237***	0.135	0.114	0.048
	(0.203)	(0.666)	(0.067)	(0.247)	(0.326)	(0.241)
Controls	No	Yes	Yes	No	Yes	Yes
Lagged outcome	No	No	Yes	No	No	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes	Yes	Yes
Observations	377	377	364	377	377	364
R-squared	0.949	0.963	0.993	N/A	N/A	N/A

Notes: The dependent variable is the estimated number of deaths from drug use disorders per hundred thousand inhabitants. The sampling period goes from 1991 to 2019. The control variables include GDP per capita, the unemployment rate, the share of the population aged between 15 to 24 years, the share of population with an alcohol use disorder, and alcohol consumption per capita. Standard errors for column (1), (2), and (3) are clustered at the country level and are between brackets. Standard errors for column (4), (5), and (6) are based on placebo tests as explained in section IV and are between brackets. *** p<0.01, ** p<0.05, * p<0.1.

E. *Robustness checks*

Although the initial analysis showed a significant negative effect of the PSA on drug use disorder, and positive significant effects on homicide and theft, it is important to evaluate the robustness of these results. I start by performing a backdating analysis, where I artificially backdate the treatment year to 2012. Table 5 presents the results of the robustness check, with each column presenting the estimated effect for one of the four outcome variables, using the SDID specification without covariates. Column 1 presents the estimated effect of the PSA in the artificial treatment year 2012 on drug use disorder. As expected, the effect is close to zero and statistically insignificant, β = -0.039, p = 0.532, lending credibility to the parallel trends assumption. Moreover, Figure 4 shows that trends after 2011 remain relatively parallel and only start to diverge after 2015, when the actual PSA was implemented. Column 2 similarly shows a statistically insignificant effect of the artificially backdated PSA on homicide, β = 0.114, p = 0.761. Additionally, Figure 4 shows that for homicide too the trend of the synthetic control after 2011 stays parallel to the U.K. until 2015, and starts to diverge from 2016 onwards, when the PSA was implemented. This lends credibility to the capability of the SDID to create a suitable control group. The estimated effect of the backdated PSA on theft is presented in Column 3, and again shows that the effect is not statistically significant, β = 55.105, p = 0.906. Moreover, Figure 4 shows a similar downward trend between the U.K. and the synthetic control

after the backdated treatment period and a divergence in trends after the actual treatment period. Finally, Column 4 shows a statistically insignificant effect of the backdated PSA on death from drug use disorder, $\beta = 0.558$, $p = 0.128$. However, from Figure 4 we can see that the trend of synthetic control starts to diverge from that of the U.K. before the actual PSA was implemented in 2016. This confirms that the SDID is indeed not able to construct a suitable control group for the outcome variable death from drug disorder, as stated in the previous section.

Table 4. Backdating analysis

	(1) Drug use disorder	(2) Homicide	(3) Theft	(4) Death from drug use disorder
U.K.*After 2011	-0.039	0.114	55.105	0.558
	(0.062)	(0.373)	(468.086)	(0.366)
Controls	No	No	No	No
Year FE	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes

Notes: Each column presents the result for one of the four outcome variables. In column (1) the dependent variable dependent variable is the fraction of the population with a drug use disorder. In column (2) the dependent variable is the number of police recorded homicides per hundred thousand inhabitants. In column (3) the dependent variable is he number of police recorded theft offences per hundred thousand inhabitants. In column (4) the dependent variable is the estimated number of deaths from drug use disorders per hundred thousand inhabitants. Standard errors are based on placebo tests as explained in section IV and are between brackets. *** p<0.01, ** p<0.05, * p<0.1.

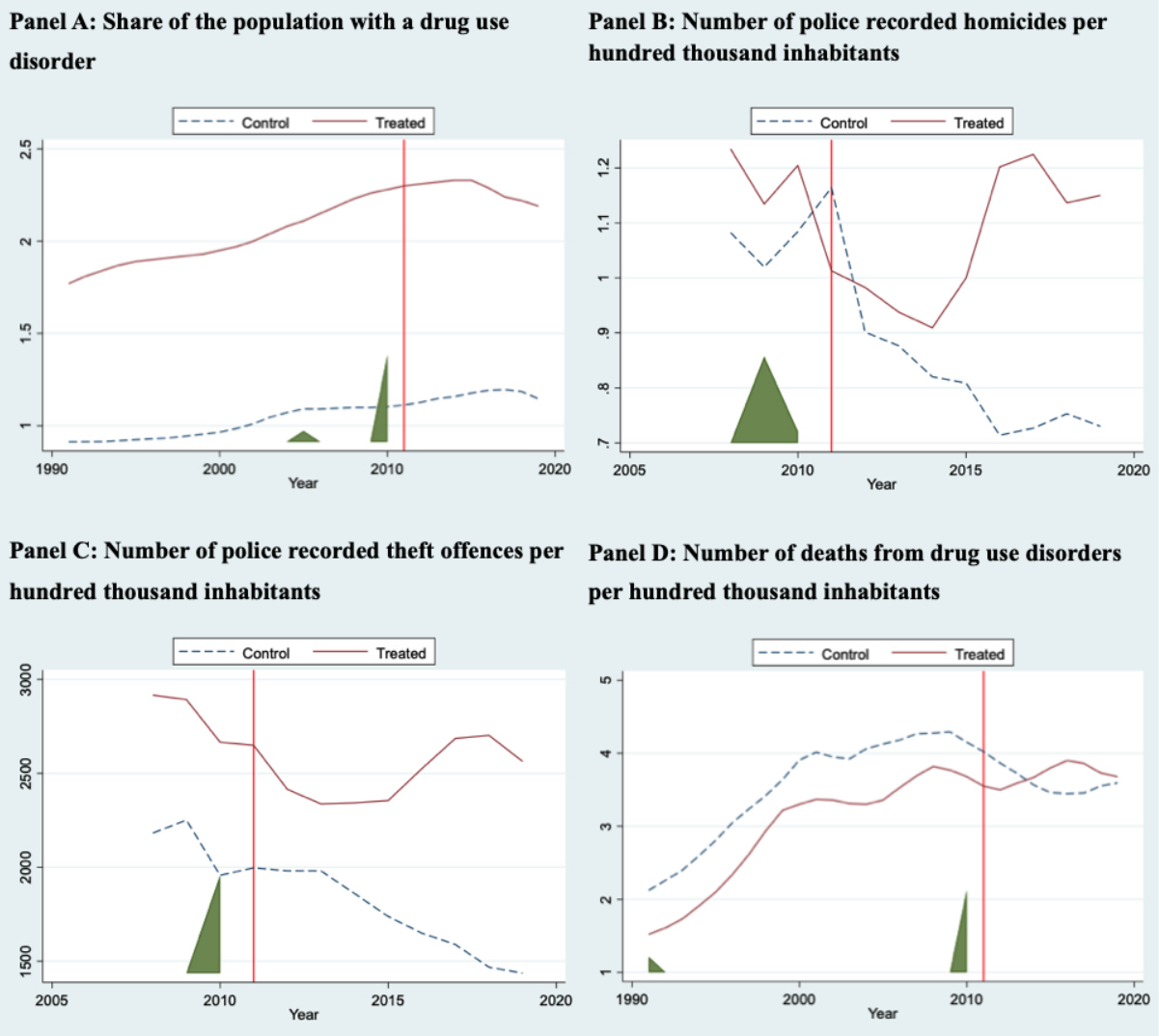

Figure 4. Trends of the U.K. and synthetic control with backdated treatment. *Notes:* The green shaded areas represent the time weights attributed to each period.

Next, I perform the second robustness check where I leave out the five highest weighted countries in synthetic control from the donor pool, one at a time. The weights each country received is presented in Table B1 in the appendix. Table 6 presents the results of the leave-one-out analysis, with every column presenting the estimated treatment effects on a different outcome variable and each panel dropping another country, descending in the amount of weight they received. Column 1 presents the estimated effects of the PSA on drug use disorder. Panels A shows a similar treatment effect and standard error to the main analysis, β = -0.118, p = 0.001. In Panel B the effect size is somewhat lower but ultimately remains statistically significant, β = -0.072, p = 0.024. Overall, the estimated effect of the PSA on drug use disorder seems robust to the leave-one-out analysis. Column 2 tells a similar story for the estimated effects of the PSA on homicide. Both the estimated effect and the standard error remain

consisted among all panels. In none of the panels the effect becomes statistically insignificant. Therefore, the estimated effect of the PSA on homicide also seems robust to the leave-one-out analysis. Column 3 on the contrary shows the estimated effect of the PSA on theft should be interpreted more cautiously. Panel A and C both show an estimated effect that is no longer significant at the 10 percent level, $\beta = 451.81$, $p = 0.130$, and $\beta = 540.88$, $p = 0.118$, respectively. This is a potential indication that the estimated effect could be influenced by policy changes or idiosyncratic shocks that occurred in the excluded countries and coincided with the introduction of the PSA. Therefore, the causality of the estimated effect of the PSA on theft loses some of its credibility. However, it should be noted that the results remain centred around the result produced using the entire donor pool. Overall, these results still support main conclusion of a positive effect of the PSA on theft. Finally, while Column 4 shows reasonably consistent effect sizes and standard errors across Panels A through E for the effect of the PSA on death from drug disorder, the earlier noted parallel trends violation renders this analysis invalid regardless.

Table 5. Leave-one-out analysis

	(1) Drug use disorder	(2) Homicide	(3) Theft	(4) Death from drug use disorder
Panel A: without 1st highest weighted country				
Treatment	-0.118***	0.318**	451.815	0.194
	(0.035)	(0.136)	(298.654)	(0.261)
Panel B: without 2nd highest weighted country				
Treatment	-0.072**	0.299**	582.586*	0.030
	(0.032)	(0.140)	(320.288)	(0.188)
Panel C: without 3rd highest weighted country				
Treatment		0.303**	540.876	0.250
		(0.140)	(345.783)	(0.249)
Panel D: without 4th highest weighted country				
Treatment		0.328**	573.176*	0.143
		(0.125)	(316.444)	(0.185)
Panel E: without 5th highest weighted country				
Treatment		0.304**	593.486**	0.121
		(0.138)	(286.711)	(0.247)
Controls	No	No	No	No
Year FE	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes

Notes: Each column presents the result for one of the four outcome variables. In column (1) the dependent variable dependent variable is the fraction of the population with a drug use disorder. In column (2) the dependent variable is the number of police recorded homicides per hundred thousand inhabitants. In column (3) the dependent variable is he number of police recorded theft offences per hundred thousand inhabitants. In column (4) the dependent variable is the estimated number of deaths from drug use disorders per hundred thousand inhabitants. Each panel removes a different country from the control group donor pool in descending order based on the weight they received in the original synthetic control. Standard errors are based on placebo test explained further explained in section IV and are between brackets. *** p<0.01, ** p<0.05, * p<0.1.

F. *Concurrent policy changes*

Although the results were mostly robust to the leave-one-out analysis, indicating that the results are most likely not driven by idiosyncratic shocks or concurring policy changes in countries in the control group, it is still possible that the estimates capture the effect of other policy changes in the U.K. that coincided with the PSA. It is therefore important to investigate if such changes occurred and evaluate what their potential implications for the results are. One Important area to explore is the strategy of the U.K. on prevention of drug use and on treatment of drug misuse. For example, if the U.K. increased their efforts on drug use prevention or treatment of drug misuse in 2016, this could very well be an explanation for the measured decrease in drug use. However, a 2016 report by The UK Focal Point on Drugs on the current drug situation shows no major updates to the prevention or treatment strategies were made in

2016. In fact, both were still covered by the UK *Drug Strategy 2010, 'Reducing demand, restricting supply, building recovery: supporting people to live a drug free life'* (UK Government, 2010). Another important area to look at is the amount of funding the police received in 2016. Specifically, if significantly less resources were made available to the police in that year compared to previous years, it could be an alternative explanation for the measured increase in violent and economic crime. However, this does not seem to be the case either. According to a Home Office (2022) report, police funding has increased every year from 2016 onwards, while funding between 2011 and 2015 fell each year. The report does note that the structure of funding for policing did change after a 2015 spending review, making a direct comparison between the two periods difficult. Non the less, it does not seem that the measured effect on violent and economic crime can be explained by a reduction in policy funding.

VI. Discussion

The results of this paper provide a deeper understanding of the effect of banning all psychoactive substances in the U.K. While the initial DID analysis suffered from a violation of the parallel trends assumption, the SDID analysis indicated a decrease in drug use disorder by 3 to 5 percent post-PSA implementation, highlighting a positive outcome. In terms of homicides, both DID and SDID methods exhibited a significant increase by 27 to 36 percent, indicating a rise in violence following the PSA. Economic crime, measured by theft offences, also showed an increase in both DID and SDID analysis, by 22 to 31 percent. Regarding deaths from drug use, constructing a suitable control group proved a challenge for both methods due to persistent parallel trends violation, making the results unreliable. The robustness checks I performed largely supported the main findings, reinforcing the credibility of the results for drug use disorder and homicides. However, the impact on theft became somewhat less certain due to fluctuations in results, possibly influenced by external policy changes or idiosyncratic shocks in the control countries.

In line with the first hypothesis, the results provide evidence for the PSA resulting in a decrease in drug use. This outcome is consistent with the theoretical prediction of Miron and Zwiebel (1995), that making drugs illegal shifts supply curve upwards and to a lesser degree shifts demand downwards, resulting in an increase in prices and a decrease in consumption. However, without information on exact price increases it is not possible to draw any conclusions about the elasticity of drug demand. Moreover, since the NPS market only resembles a fraction of the total drug market, while the decrease is measured over the total drug

use, it is difficult to draw conclusions about the magnitude of the effect. In line with the second hypothesis, the results show an increase in violence following the PSA. This supports the theoretical predictions of the academic literature and is consistent with earlier empirical results (Soares, 2017; Kronick, 2020; Owens, 2014). The results regarding the third hypothesis, while less conclusive, do provide evidence supporting that economic crime increased following the PSA. The results do not, however, reveal the mechanism behind this increase and could either be driven by users having to resort to theft to sustain their current level of use following an increase in prices (Silverman & Spruill, 1997; Felson & Staff, 2017) or by a shift in police resources away from controlling economic crime to enforcing prohibition (Benson et al., 1992). Finally, the results regarding the fourth hypothesis remain inconclusive, so this study was not able to contribute to the understanding of the effect of drug prohibition on death from use.

While providing valuable insights, the study design is subject to several limitations that warrant consideration. Firstly, the use of the share of the population with a drug use disorder as a proxy for drug use has inherent limitations. This proxy represents only a subset of individuals with a drug use disorder, excluding individuals who do not seek help or are not officially registered. Moreover, while individuals with a drug use disorder are a crucial demographic, it does not encompass the entire spectrum of drug users, potentially limiting the comprehensiveness of the findings. Similarly, employing homicide as a proxy for violence has its constraints. This measure fails to capture acts of violence such as shootings and stabbings that are non-lethal, which are potentially significant negative externalities associated with the PSA. Consequently, the impact of the PSA on overall violence in the U.K. might be underestimated.

Secondly, in an ideal study design, the countries in the control group should remain unaffected by any policy changes related to NPS to prevent bias in the results. Although I attempted to mitigate this bias by excluding countries that implemented major NPS-related policy changes in the same year and the year following the implementation of the PSA, it's important to acknowledge the potential influence of policy changes in other countries. Many countries adjusted their drug policies during the observation period to some degree, possibly introducing bias into the results. Notably, these policy changes aimed to reduce drug use by banning more NPS and intensifying enforcement efforts, aligning with the objectives of the PSA. Therefore, our findings likely represent a lower bound of the actual effects, indicating a conservative estimate of the impact of the PSA. Moreover, the potentially confounding policies might be an explanation for the sensitivity of the statistical significance of the results on theft to the exclusion of specific countries.

Despite the limitations this study exploited a unique opportunity to estimate the causal effects of drug prohibition. Building upon these insights, future research could attempt to estimate the costs and benefits of this policy in monetary terms. To improve upon these results future research might investigate different proxies for the different outcome variables and confirm replicability. Moreover, future research could enlarge the scope of outcome variables in general or investigate the underlying mechanisms at play. Finally, while this study clearly showed negative externalities to drug prohibition, it did not generate insights into the feasibility of alternative policies, another potential avenue for future research.

VI. Conclusion

As drug use is becoming ever more prevalent around the globe despite considerable efforts of law enforcement, it is important to reflect upon the effectiveness of current drug policy and its potentially unintended side effects. By exploiting a unique policy change in the U.K. that banned all psychoactive substances, including a wide range of New Psychoactive Substances (NPS) often referred to as “legal highs”, this study aimed to document the causal effect of drug prohibition. While establishing a decrease in drug use, this decrease was accompanied by proportionally larger increases in the level violence and economic crime. These results should be considered by policymakers and prompt an investigation into alternative drug policies that are able to achieve a similar level of restraint on drug use without being subject to negative externalities that bear significant social costs such as violence and economic crime.

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Appendix A. Additional synthetic difference-in-differences graphs

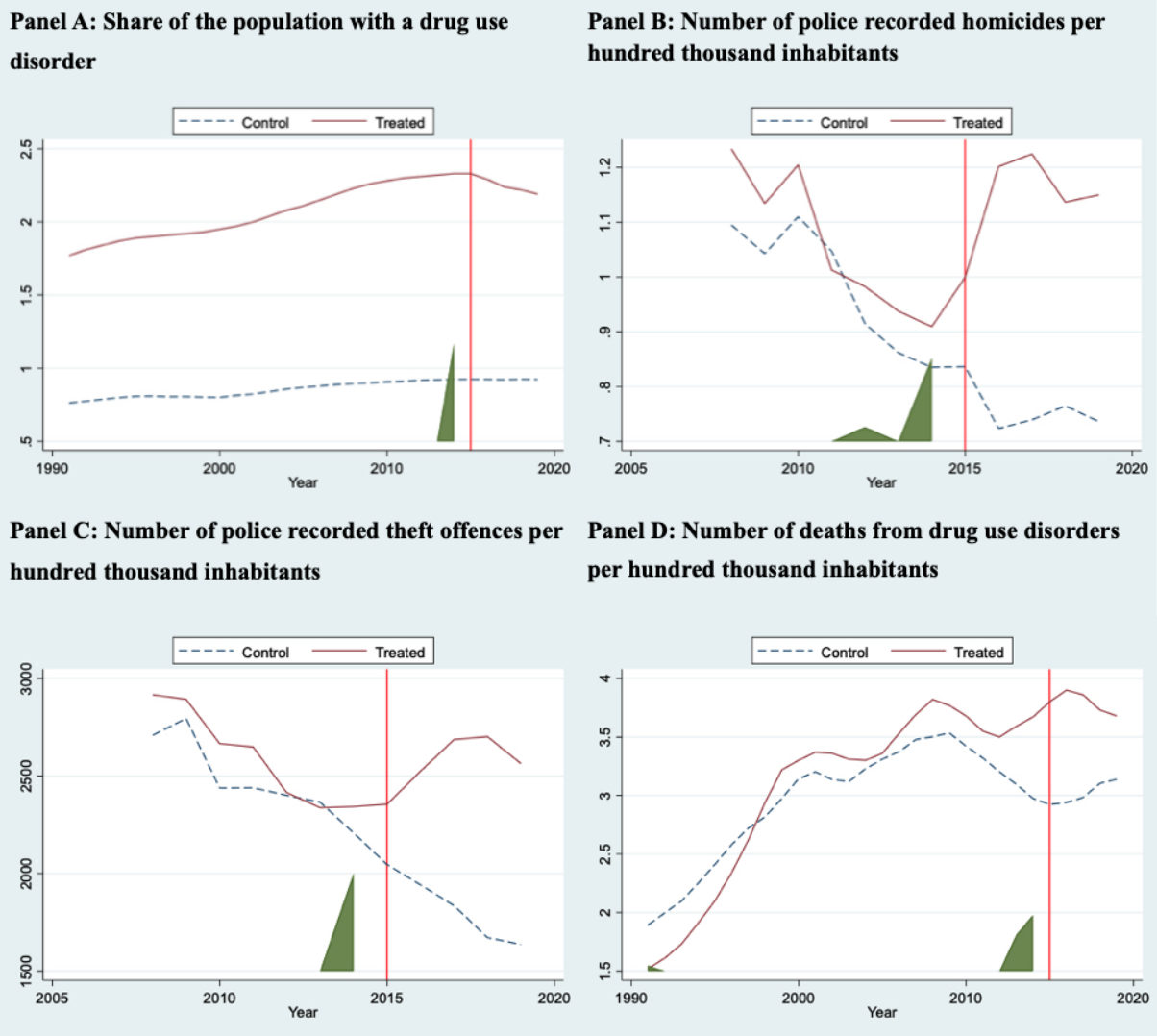

Figure A1. Trends of the U.K. and synthetic control with covariates. *Notes:* The green shaded areas represent the time weights attributed to each period.

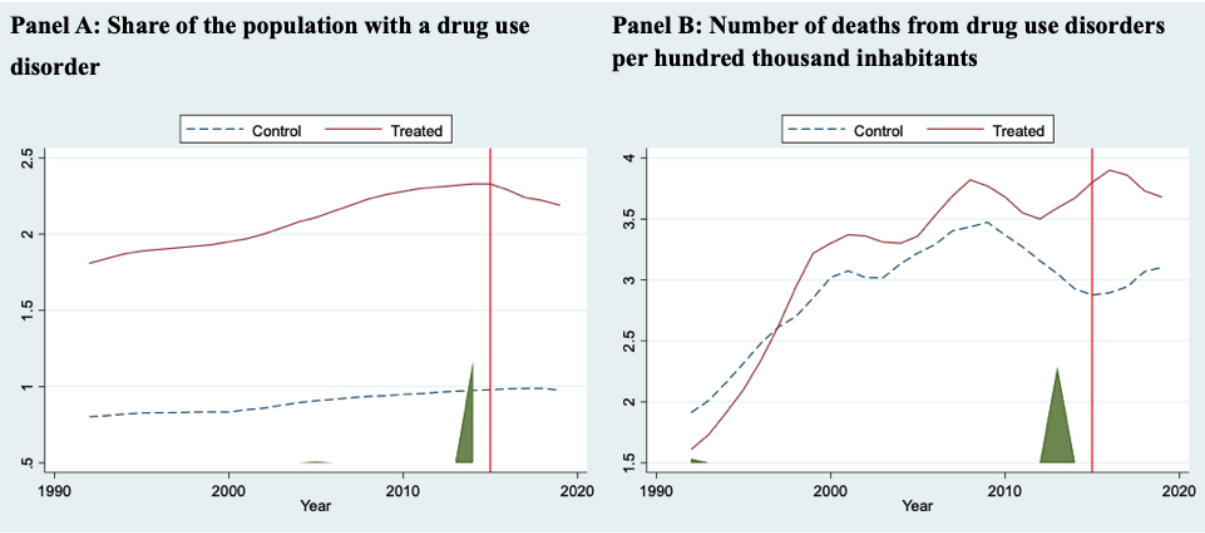

Figure A2. Trends of the U.K. and synthetic control with covariates and a lagged outcome. *Notes:* The green shaded areas represent the time weights attributed to each period.

Appendix B. Country weights for the synthetic control group

Table B1. Country weights in the donor pool for the synthetic control group of the baseline model

	Drug use disorder	Homicide	Theft	Death from drug use disorder
Austria	0.146	0.077	0.085	0
Denmark	0	0.094	0.144	0
Finland	0.854	0.103	0.105	0.457
Greece	0	0.074	0.096	0.047
Iceland	0	0.054	0.105	0.192
Italy	0	0.094	0.032	0
Luxembourg	0	0.115	0.033	0
Netherlands	0	0.092	0.112	0.037
Norway	0	0.012	0.094	0.265
Portugal	0	0.096	0.086	0.002
Spain	0	0.092	0.082	0
Switzerland	0	0.096	0.026	0