The effect of alcohol sales restrictions on alcohol poisoning mortality: evidence from Russia

Alexander S. Skorobogatov

National Research University Higher School of Economics

St. Petersburg, Russia

Abstract

This paper examines the tough anti-alcohol legislation recently introduced in Russia, which due to regional variation allows it to be used as a natural experiment. The effect of the restriction of trading hours on alcoholic poisoning mortality is estimated. To establish the causal link, difference-in-differences, synthetic controls, and their generalized version are used. The main conclusion is that the sales restrictions lead to higher alcohol poisoning mortality, which implies that more toxic alcohol surrogates serve as substitutes for commercially available alcohol.

Keywords: Alcohol mortality; Alcohol legislation; Synthetic controls; Informal alcohol market

JEL classification: I12, I18, J48

1. Introduction

Alcohol abuse and acute alcohol poisoning contribute heavily to alcohol mortality in developed and developing countries (Global status report on alcohol and health, 2018, 67). Alcohol poisoning depends on alcohol consumption although there are substantial differences in alcohol poisoning mortality among countries with similar levels of alcohol consumption. For example, while Russia and other former Soviet republics do not differ much in their statistics of alcohol consumption from the other European countries, their alcohol poisoning mortalities are several times higher (42, 13; Yakovlev, 2018).

This suggests the varied efficiency of anti-alcohol policies across countries. Anti-alcohol policies immediately affect alcohol consumption, although their final goal is normally to mitigate its harmful consequences, in particular alcohol mortality. A country facing unusually high alcohol mortality given modest alcohol consumption may be less efficient in dealing with alcohol mortality while imposing restrictions on alcohol consumption. As indicated in Stickley et al. (2007), what is also important for alcohol mortality apart from consumption is widespread drinking habits. The latter include the use of alcohol surrogates such as home-made spirits, contaminated alcohol,

and liquids not meant for ingestion with much higher oral toxicity potential. Given drinking habits and available alcohol surrogates, the effect of alcohol restrictions on alcohol poisoning mortality may be mixed as they impact directly on the consumption of commercially available alcohol and indirectly on its replacement by surrogates.

In this respect, the alcohol trade regulations recently introduced in Russia present a natural experiment which can be used to check the policy's efficiency in specific conditions. Alcohol consumption and its related mortality substantially increased in Russia during the post-Soviet period (Alcohol abuse in the Russian Federation, 2009; Yakovlev, 2013). An important reason for this increase was that alcohol, particularly strong spirits, became available as never before after the previous Soviet regulations for alcohol production and trade were abolished and the anti-alcoholic campaign was stopped (Denisova, 2010; Bhattacharya et al., 2013). To address these catastrophic consequences, the state began to restrict alcohol sales. Beginning in 2005, various regions have been introducing and toughening local regulations on the hours of alcohol sales. Until 2012, the respective restrictions applied only to strong spirits. Russia features widespread 24-hour shops. Moreover, people mostly drink at home rather than in bars (Public Opinion Fund, 2014). Therefore, the newly imposed restrictions actually made strong alcohol less available for 'take-away' and prevented people from beginning or continuing drinking. Some people facing the newly imposed restrictions do not continue drinking using factory-made spirits, while others look for substitutes which in many cases are more toxic. Therefore, even if a small number of people replace factory-made alcohol with surrogates and end up fatally poisoned, the lower recorded consumption may not be consistent with lower alcohol mortality. If the poisoning risk from surrogates replacing commercially available alcohol is comparable with that avoided due to the decreased recorded alcohol consumption, the final policy impact on alcohol mortality is uncertain.

There is rich literature on anti-alcohol policies and their effectiveness at preventing alcohol abuse and its consequences. Alcohol taxes are reported to reduce violent crimes, injury deaths, and traffic accidents (e.g., Cook and Durrance, 2013; Arranz and Gil, 2009). Fertig and Watson (2009) show that strict alcohol regulations are effective in preventing adverse birth outcomes in the US. Plant & Plant (2005) in their review of studies for closing hour restrictions noted that the results had mostly shown the effectiveness of these measures. Likewise, from 15 studies on the effect of restricting the hours of alcohol sales, Popova et al. (2009) concluded that the majority of these studies confirmed the effectiveness of such restrictions. A more recent study shows the effectiveness of time restrictions to prevent such an alcohol-related consequence as traffic accidents (Green et al., 2014).

Although studies of alcohol regulation and time restrictions have mostly demonstrated effectiveness, some studies have concluded the opposite. Son and Topyan (2011) find no evidence of the effectiveness of tax policies in the US. Hahn et al. (2010) in their review of studies on the effect of legal hours of alcohol sales on excessive alcohol consumption and related harms cite a number of papers founding no effects or only insignificant ones. Humphreys and Eisner (2014) did not find any evidence supporting the efficiency of the restrictions on the opening hours of alcohol outlets in preventing alcohol-related violence. They noted that their strict research design aimed at

establishing a causal link may provide evidence contrasting with the previous studies supporting the effectiveness of the restrictions. More recently Lindo et al. (2016) did not support the effect of access to alcohol on traffic accidents.

In addition, the existing evidence mostly concerns developed countries and the effects on alcohol use and alcohol-related traffic accidents and violence. To the best of my knowledge, such an important consequence of alcohol use as alcohol poisoning mortality has not been recently studied in connection with alcohol legislation in either developed, or less-developed countries with traditionally higher levels of alcohol mortality. The literature on alcohol mortality suggests that factors increasing alcohol consumption also lead to higher levels of various alcohol-related mortalities (see, e.g., Carpenter and Dobkin, 2009; Kopp and Ogrodnik, 2017; and Effertz et al., 2017) for evidence for the US, France, and Germany, respectively). In the context of alcohol-related mortality, Law and Marks (2019) examine the prohibition policies conducted in the US between 1900 and 1920 with the results in favor of the effectiveness of the policies. Johansson et al. (2014), in contrast, do not find any mortality or hospitalization effect in Swedish regions near the Finnish border from alcohol-related tax cuts imposed in Finland.

Studies for less developed and former command economies add to the literature exploring relationships between alcohol policies, consumption, and the consequences in specific social and economic environments. In Russia and other former Soviet Republics, these environments feature poor observance of the law, a long tradition of the excessive consumption of strong spirits, and a significant supply of homemade or surrogate alcoholic beverages. The Russian context may help better understand the potential indirect effects of alcohol restrictions in the presence of available alcohol substitutes. There are a number of studies of alcohol use and its effects using Russian data (Nemtsov, 2000; Denisova, 2010; Bhattacharya et al., 2013; Yakovlev, 2013; 2018). Kolosnitsyna et al. (2014, 2017) examined the restrictive Russian policy. Their results supported its effectiveness as a means to decrease alcohol consumption without any evidence of the substitution of beverages not under restriction. However, their research contrasted 2009 with 2010 even though the restriction had been gradually imposed since 2005. Therefore, their research did not distinguish between the immediate and lagged effects of the restriction. In addition, they estimated only the association between the policy and drinking without attempting to address confounding factors such as omitted variable bias. To sum up the existing evidence, there is no consensus for the effectiveness of time restrictions and other alcohol regulations, and the existing results mostly concern developed countries and general consequences.

This research fills the gap in the literature focusing on the policy effect on alcohol poisoning mortality in specific economic and institutional conditions including the informal alcohol market and drinking habits involving the use of surrogates. This study determines the effect of the time restrictive policies of strong alcohol sales on alcohol poisoning mortality. The literature does not contain a relevant evidence to make this effect obvious. The ultimate effect of the restrictions has been questioned by most Russians (VCIOM, 2009) for reasons related to the expected indirect consequences, as well as the aforementioned national features making the policy potentially inefficient. It was unknown, a priori, to what extent people would decrease their alcohol consumption

and replace factory-made alcohol by surrogates in response to the time restrictions with respective implications for alcohol mortality. Unlike most studies, this research examines the indirect effect of possible substitutions for factory made alcohol not falling under the restriction. A number of experts concern that the restrictions could induce the consumption of various alcohol surrogates and thus significantly worsen the alcohol situation (e. g., Nikitina, 2010; BBC, 2013).

The paper adds to the health economics literature in several aspects. First, it sheds light on the general debate on the effects of alcohol regulation in the specific conditions of less developed countries. Second, its conclusions have implications for drinking habits as determinants of overall health. Third, the paper presents a rigorous analysis applying up-to-date methods to establish causal links. To determine the effect of interest, I apply difference-in-differences, synthetic controls and their generalized version. Unlike other studies which examine the same policy (Kolosnitsyna et al., 2014; 2017), this paper examines the effect of the restrictions on longitudinal data from 1996-2011 from the years in which none of the regions were under restrictions to the time in which all the regions were under the restrictions. The results of using the three methods are consistent in that the restrictions increase alcohol poisoning mortality, although differing from each other in the estimated magnitudes of the effect.

The rest of the paper is organized as follows. Section 2 presents preliminary observations for the relationship of interest and outlines the data generating process. Section 3 describes the empirical strategy. Section 4 describes the data. Section 5 presents and discusses the results. Section 6 concludes.

2. Alcohol restrictions and mortality in Russia

2.1. Policy and alcohol poisoning mortality

One of the papers dating back to the period the restrictions were just being introduced began by pointing to the 'legendary' alcohol consumption in Russia (Baltagi and Geishecker, 2006, 893). To cope with the problem, several regions began conducting local restriction policies. The restrictions took the form of prohibition of strong alcohol sales between specific times. Table A1 in Appendix contains mean opening and closing hours under the restrictions for regions with earliest and latest years of introducing the restrictions, namely in 2005 and 2011. Average opening and closing hours authorized by the regulation in 2005 were about 8 am and 11 pm, respectively. In subsequent years, other regions were gradually joining those with the restrictions. Fig. 1 presents the timing of the introduction of the restrictions by regions.

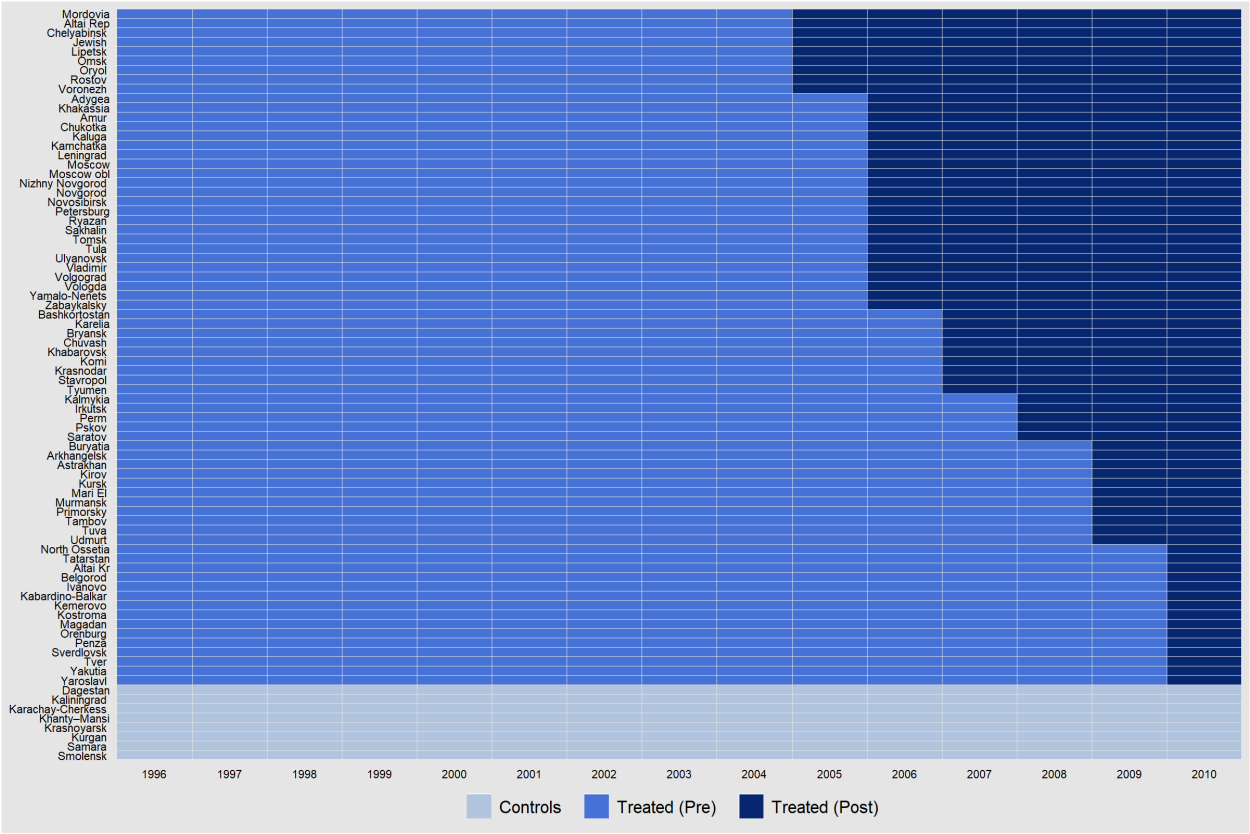


Fig. 1. Timing of introducing the policy by Russian regions

As seen in Fig. 1, the first group of regions introducing the restrictions in 2005 was relatively small, making up about 11% of the regions. The two strongest jumps in introducing the restrictions were in 2006 when the restrictions were first implemented in a number of important regions, including Moscow, St. Petersburg, and their neighboring regions, and in 2010, before the respective federal law was adopted. The first local laws in 2005 and 2006 placed about 40% of the regions under the time restrictions. Between 2006 and 2010, regions that had already introduced restrictions toughened or, sometimes, eased them. Since 2011, all the regions have been under the restrictions, though the actual closing hours vary.

Fig. 2. Vodka sales and alcohol poisoning mortality
Note: Both variables are presented in normalized forms.

Fig. 2 shows the overall dynamics of vodka sales, the most popular strong spirit in Russia, and mortality from alcohol poisoning. During the period of restrictions both alcohol variables were going down though, the downward trends had begun earlier, and should be accounted for when making a causal inference for the impact of the restrictions. To allow for the trend, I regress the dynamics of alcohol poisoning mortality on whether a region was among the first to introduce the restriction. Fig. 3 presents scatterplots of first differences for alcohol poisoning mortality in 2006 and 2007 versus the closing hour restrictions in 2005 and 2006, respectively. Though the mortality dynamics were downward in most of the regions, those under the restrictions tended to show weaker downward trends. The same correlation is also illustrated in Fig. 1A in Appendix which contains maps of regions under the restrictions in 2006 and the first difference of alcohol poisoning mortality in 2007.

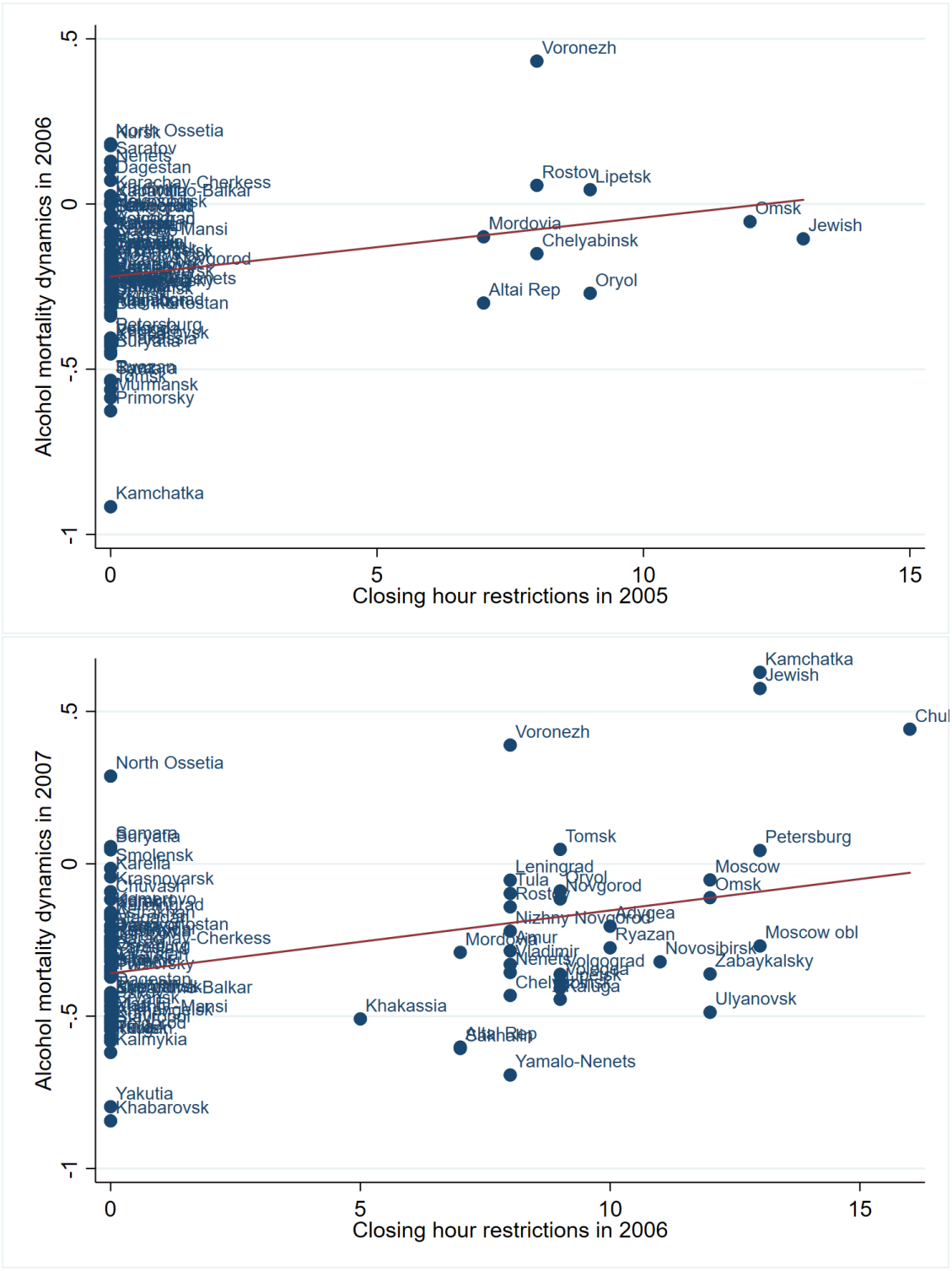


Fig. 3. Closing hour restrictions and the first differences of alcohol poisoning mortality

A comparison of the downward dynamics of alcohol poisoning mortality in regions with earlier and later years of the imposition of the restrictions is also instructive. Fig. 4 presents moving averages of alcohol poisoning mortality for regions which introduced the restrictions in 2005, 2006, and 2011, respectively. In all cases, the mortality measure is going down, but the slope is smaller for regions which introduced the policy earlier. These preliminary observations suggest that alcohol poisoning mortality was decreasing within the period of the policy though, the restriction

slowed down this process rather than induced it. Similar relative dynamics are seen in mean mortality levels in regions under the restriction relative to remaining regions in 2005 and 2011. While at the start of the policy regions first introducing the restriction had lower levels of alcohol poisoning mortality than the remaining regions, at the end of the period considered they had higher mortality levels (for respective means and differences, see Table A1 in Appendix).

Fig. 4. Alcohol poisoning mortality for groups of regions
Note: CH 2005, CH 2006, and CH 2011 denote moving averages of alcohol poisoning mortality for groups of regions introducing the restriction in 2005, 2006, and 2011, respectively.

2.2. Direct and indirect effects of the policy

A preliminary examination of the data suggests that the policy might have an unintended indirect effect offsetting the direct effect on commercially sold strong alcohol. The indirect effect might be related to the use of alcohol surrogates. Alcohol abuse strongly correlates with alcohol use at night (Alcohol abuse in the Russian Federation, 2009, 47). There is also evidence that a significant percentage of the population drink various surrogates as substitutes for commercially available strong spirits, which is typical for developing countries (McKee et al, 2005; Stickley et al., 2007; Rehm et al., 2014). For example, samogon (home-produced spirit) is reported to be substituted for vodka as its price increases (Andrienko and Nemtsov, 2006). Alcohol surrogates are much cheaper for consumers and their distribution is profitable due to the avoidance of alcohol taxation (Stickley et al., 2007; Rehm and Poznyak, 2015). This especially concerns Russia, which has featured widespread use of alcohol surrogates in the post-Soviet period (Stickley et al., 2007), as well as the high prevalence of heavy episodic drinking (Global status report on alcohol and health, 2018; Malisauskaite and Klein, 2018). The consumption of alcohol surrogates is much riskier and contributes

heavily to "extreme alcohol poisoning rates" in former Soviet republics, including Russia (McKee et al, 2005, 447; Gil et al., 2009). The widespread alcohol surrogates in Russia include samogon, medical compounds, and spirits not for consumption such as aftershaves, perfumes, antifreeze, fire-lighting alcohol, window cleaning fluids, and other types of industrial alcohol.[1] In some countries, including Russia, spirits not for consumption are deliberately produced as a potential substitute for vodka to avoid higher taxation (Rehm and Poznyak, 2015). Their much higher toxicity relates to additional toxic alcohols (including methanol), as well as substantially higher concentrations of ethanol (up to 94% for medical compounds) (McKee et al, 2005). Alcohol surrogates are normally not legally sold[2] or not sold as alcoholic beverages thus they are not subject to the policy (McKee et al., 2005). To sum up, alcohol surrogates are widespread, more toxic than beverage alcohol, and not subject to the restrictions, which make them suitable for replacing commercially sold alcohol when the latter is unavailable. Therefore, the policy's effect on alcohol poisoning mortality is ambiguous. While decreasing the use of commercially sold alcohol, the restrictions induce the use of more toxic surrogates, which potentially reverses the ultimate effect on alcohol poisoning mortality.

Starting from the stylized facts on alcohol surrogates, the ultimate effect of the restriction on the outcome of interest can be modelled as follows. Let N be the population of hazardous alcohol consumers involved in binge drinking with the risk of fatal alcohol poisoning, and r is the share of those who would replace commercially sold alcohol with surrogates if the former is unavailable due to the restrictions. Let $P(a)$ and $P(s)$ be the probabilities of fatal poisoning when commercially sold and surrogate spirits are consumed, respectively. For expositional simplicity, assume that the surrogates are consumed only when factory-made alcohol is unavailable, in particular during the restriction hours, and that the surrogates are the only available spirits during the restriction. Then the number of fatal outcomes with and without the policy are $NP(s)r$ and $NP(a)$, respectively, and the net effect of the policy is as follows

$$E = N\left[P(s)r - P(a)\right]. \tag{1}$$

Thus, the net effect depends on the relative magnitude of the terms in brackets. If nobody used surrogates during the restriction hours the net effect would be $-NP(a)$. If fatal poisoning mostly happens to surrogate users, the net effect is close to $NP(s)r$. If the share of potential surrogate users is high enough and the poisoning potential of the surrogates is much higher than that of factory-made alcohol, the effect should be positive, while if the share of potential surrogate users is low and/or poisoning probabilities for factory-made spirits and surrogates are similar, the net effect should be negative. Thus, the net effect depends on relative toxicity of surrogates and the extent to which people replace commercially sold alcohol with surrogates when being under the restrictions.

[1] Acute alcohol poisoning normally results from binge drinking parties in which people get intoxicated by substances entering alcohol consumed. Therefore, what matters is how much toxic substances are consumed per occasion. It depends on the concentration of toxic elements in a spirit: higher concentration of pure alcohol and/or other toxic elements in spirits consumed is normally consistent with higher risk of poisoning. Alcohol surrogates normally contain higher concentrations of toxic elements than commercially sold alcohol.

[2] For example, they can be "exchanged by barter or in payment for informal labour" (Stickley et al., 2007, p. 447).

3. Empirical strategy

3.1. Difference-in-differences

To infer a causal link between the policy effect and the outcome of interest, I use a difference-in-differences analysis (DD) (Angrist and Pischke, 2009). The method distinguishes between the regions exposed to the policy effect in terms of alcohol poisoning mortality and those who are not yet or less exposed to this effect. If the policy has an effect, a systematic difference in the mortality measure should be observed between the groups.

The inference based on a DD specification depends on whether the parallel trend assumption holds.[3] As seen in Fig. 4, the trends in the outcome of interest for regions introducing the policy in different years look similar before 2005, so that a visual inspection of the trends is in favor of the assumption. However, as an additional safeguard against violating the assumption, I control for a number of time-varying regional characteristics, as well as a first lag of the dependent variable as a close covariate of potentially important omitted time-varying confounders. The DD specification is as follows:

$$M_{it} = \alpha C_{it-1} + \beta \mathbf{X}_{it} + \gamma M_{it-1} + \eta Y_t + \epsilon_{it} \tag{2}$$

where M_{it} is log alcohol poisoning mortality in the ith region in the tth year; C_{it-1} is the lagged closing hour restrictions in the ith region in the tth year, which is used to account for many cases of the restrictions imposed toward the end of a year, which might lead to effects no earlier than the following year;[4] $\mathbf{X}_{it}$ is a vector of control variables grouped in three categories as economic, demographic, and geographic regional characteristics (see Table A1 in Appendix);[5] Y_t is the tth year fixed effect; ϵ_{it} is a two-component error term. To consistently estimate the regression, I use the Arellano-Bond estimator.

The variable of interest allows me to check whether there was a change in the mortality measure after beginning the policy, which would be specific to the regions under the restrictions. If harm from switching to alcohol surrogates is more than the harm prevented due to the direct effect of the policy one obtains $\alpha > 0$. In this case, the regions under the restrictions would systematically differ from the rest of the regions by their alcohol poisoning mortality dynamics after the restrictions were imposed.

[3] This requires that treatment and control groups show similar trends before a treatment. In this case different trends after the treatment are considered as evidence for the treatment effect.

[4] Using the lagged variable is also consistent with the theory of rational addiction which suggests a stronger negative price elasticity in the long run compared with the short term (Becker and Murphy, 1988; Baltagi and Geishecker, 2006). The same should be true for any restriction of addictive behaviors.

[5] As follows from the literature, all the control variables are potential covariates for alsohol use, drinking patterns, and alcohol mortality. This is true for economic conditions including income, prices, and their determinants (Kolosnitsyna et al., 2014; Johansson et al., 2006; Treisman, 2010; Skorobogatov, 2012; Cook and Peters, 2005; Dee, 2001), geographic (Skorobogatov, 2018; Yakovlev, 2018), and demographic characteristics (Radaev and Roshchina, 2019; Yakovlev, 2012; Case and Deaton, 2017; Isabel and Molina, 2007).

3.2. Synthetic controls

The main concern when using DD is that it only accounts for time-constant region-specific heterogeneity. In the DD specification, even though I allow for time-varying heterogeneity including a number of time-varying regional characteristics and the dependent variable time lag, potentially important omitted time-varying characteristics may bias the results. To more fully allow for the time-varying heterogeneity, I use the synthetic control method (SC) of Abadie and Gardeazabal (2003), which is applied widely for determining causal links in health economics literature (e.g., Barofsky et al., 2015; Bilgel and Galle, 2015; Green et al., 2014; Kreif et al., 2015; Hernæs, 2018; Lépine et al., 2018).

The task is to estimate the policy effect defined in (1) which when applied to the data is given by

$$\bar{E}_t = \bar{M}_t^P - \bar{M}_t^N \tag{3}$$

where $\bar{E}_t$ is the average policy effect in regions under the policy in the tth year; $\bar{M}_t^P$ is the alcohol poisoning mortality in regions under the policy in the tth year; and $\bar{M}_t^N$ is the alcohol poisoning mortality in the same observational units which would have been observed in the absence of the policy. Of the two terms on the right-hand side of (3) only the former is observed, so that the estimation of the policy effect reduces to the construction of the counterfactual $\bar{M}_t^N$. The outcome of interest in region $j = 1, ..., J$ not exposed to the policy in the tth year is modelled as

$$M_{jt} = \delta_t + \theta_t \mathbf{X}_j + \lambda_t \mu_j + \varepsilon_{jt} \tag{4}$$

where λ_t time-varying unobserved common factors, δ_t and μ_j are unknown factor loadings, and $\mathbf{X}_j$ is a vector of observed covariates including the same regional characteristics as in (2), and ε_{jt} is the error term. Define a vector $\mathbf{w} = (w_1, ..., w_J)$, $w_j \geq 0$, $\sum_{j=1}^{I} w_j = 1$. As follows from Abadie et al. (2010), a vector $\mathbf{w}^*$ satisfying the conditions $\sum_{j=1}^{J} w_j^* M_{jt} = \bar{M}_t^P$ and $\sum_{j=1}^{J} w_j^* \mathbf{X}_j = \bar{\mathbf{X}}^P$ provides an unbiased estimator of $\bar{M}_t^N$.

The data I use differs from those used by Abadie et al. (2010; 2015) for illustrating the performance of SC in two respects. First, in the dataset I use, there are numerous units subject to the treatment in any year of the treatment period. That is why I take the mean values of interest and control variables for the treatment group, rather than values for a particular unit. Second, in the data used by Abadie et al. (2010; 2015), units from the control groups are never exposed to the effect of the treatment over the period considered, which makes the breakdown of observational units into treatment and control groups unambiguous. In the dataset I use, the number of units exposed to the treatment changes over the treatment period. As mentioned (see Fig. 1), different Russian regions had been imposing the restrictions in different years until all of them were under the restrictions. Therefore, I divide the units into treatment and control groups depending on whether they are under the restriction in a particular year. Specifically, the treatment group is made up of the regions which imposed the restrictions not later than in 2006, while the control group consists of the rest of the regions. In doing so, I assume that over a relatively short period earlier exposure

to the treatment suggests a stronger effect, which should translate into a systematic difference in the outcome of interest between the groups.[6] The choice of the year of exposure as a criterion for dividing the regions into the groups is motivated by the numbers of units in both groups. As seen in Fig. 1, by 2006 the number of regions having introduced the policy is large enough to make the results representative for the whole population of regions, while the number of control regions is also large enough to make the respective linear combinations good approximations of covariates for the treated regions. In addition, to check whether the results depend on this division of regions, I perform the same analysis using different divisions of regions into the groups.[7]

Following Abadie et al. (2015), I perform several robustness checks. The leave-one-out placebo checks if the results depend on particular units from the control group, obtaining estimates without each of the units with a positive weight. The in-place-placebo estimates the counterfactuals for regions from the control group to check whether the result for the treatment group systematically differs from those for control regions. Another check is an estimate of the distribution of post-MSPE (mean squared prediction error) to pre-MSPE ratios, which gives an exact picture of the relative counterfactual dynamics in both groups.

3.3. Generalized synthetic controls

As an additional safeguard against potential bias caused by the assumed relationship between timing and the effect of restriction, I also employ a method independent of such assumptions. The generalized synthetic control method (GSC) proposed by Xu (2017) enables one to deal with observational units experiencing the treatment at different times. The method lets units move from the control group to the treatment group once their actual treatment period starts. In this respect this method fits the data I use well.

Like in (3), the task is to estimate the policy effect as follows

$$E_{it} = M_{it}^{P} - M_{it}^{N}. \tag{5}$$

However, now the effect is estimated for every unit entering the treatment group. The factor model has following functional form:

$$M_{it} = \delta_{it} D_{it} + \theta \mathbf{X}_{it} + \lambda_t \mu_i + \varepsilon_{it} \tag{6}$$

which again is similar to (4), but now the treatment dummy indicator D_{it} lets different regions experience the treatment in different years. The estimation of the counterfactual M_{it}^{N} involves three steps. The first step is the estimation of an interactive fixed effects model for the control units only $M_i = \theta \mathbf{X}_i + \lambda_t \mu + \varepsilon_i$. At the next step, one estimates factor loadings μ_i for the treated units based on the predicted outcome of interest in the pretreatment period. The final step

[6] A similar assumption is made in Abadie et al. (2003) breaking down observational units based on the extent to which they are exposed to an intervention.

[7] In particular, I divide the regions into those which introduced the restrictions in 2005 and 2010 to see the dynamics in the interim period. As the control group in this case contains a small number of units this gives a poor fit in the pre-treatment period which makes it less reliable for a post-treatment comparison. Nevertheless, I report the results in Appendix.

computes the counterfactual for the treated units using the estimates $\hat{\theta}$, $\hat{\lambda}_i$, and $\hat{\mu}_i$. In addition, to select the number of factors in the vector λ_i, which would be optimal with respect to MSPE in the pretreatment period, a cross-validating procedure is used (for more details, see Xu (2017)).

As mentioned, GSC solves the problem of the proper breakdown of the units into the treatment and control groups when they move from one group to another over the period considered, although this requires having a group of units never entering the treatment group. When using GSC, for such a group I use regions with the latest date of introducing the restrictions of 2011, and therefore the estimates are obtained for the period ending in 2010.

4. Data

All the variables used are region-level ones. Mortality measures and other regional characteristics come from the official statistical agency Rosstat (2014). Data about the regional laws restricting the time of alcohol sales are from a commercially sold collection of local acts Consultant plus (2014).

As mentioned, relative dynamics of alcohol poisoning mortality (see Fig. 4) are in favor of the common trend assumption. In addition, a comparison of the trends also gives an initial result for the effect of interest. The curves substantially change their relative slope only after the beginning of the treatment period. The direction of the change of trends relative to each other is in favor of the positive effect. The groups show similar upward and downward dynamics before the treatment, but after the treatment is in action, regions which introduced the restrictions earlier show a substantially less evident fall in alcohol poisoning mortality.

Summary statistics are presented in Table A1 in Appendix for all the variables used in the analysis. To make them informative for the effect of interest, I divided them into those for regions which introduced the restrictions in 2005 and the rest of the regions to compare their relative statistics in 2005 and 2011. Again, at the start of the period, the first group showed lower levels of alcohol poisoning mortality and at the end these levels surpassed those for the second group.

Other variables mostly display substantial differences between the groups, which motivates their use as controls. Regional conditions, in addition to the time restrictions, include local prices and sales for beverages, the share of urban population, the workforce in the region's population, log population size, log land area, climate conditions measured by average temperature in winter and summer, and general mortality.

Given the timing of the local policies, I use a time frame of 2005-2011 for estimating the policy's indirect effect on alcohol poisoning mortality.

5. Results

5.1. Main results

Table 1
Closing hour restrictions and alcohol poisoning mortality, 2005-2011

	(1)	(2)	(3)	(4)
Closing hour restrictions	0.0109**	0.0138***	0.0136***	0.0135***
	[0.0048]	[0.0048]	[0.0049]	[0.0049]
Region level characteristics				
economic	No	Yes	Yes	Yes
demographic	No	No	Yes	Yes
geographic	No	No	No	Yes
Observations	485	469	469	469

Note: GMM stardard errors are in parentheses. *** p<0.01, ** p<0.05, * p<0.1. All the specifications include year fixed effects. Economic characteristics include log regional income per capita, log prices of consumer basket and vodka, the share of urban population, and the share of laborforce. Demographic characteristics include log population size, log mortality, and deaths in traffic accidents per 100,000 population. Geographic characteristics include average winter and summer temperatures.

The results from estimating DD specifications for the log alcohol poisoning mortality are presented in Table 1. The main variable of interest, closing time restrictions, is significantly positive in all specifications. Adding controls tends to increase the absolute estimate. In particular, adding economic, and then demographic and geographic, controls increases the estimate from 0.0109 to 0.0138, 0.0136 and 0.0135, respectively. The latter estimate from the longest specification suggests that an additional hour of restrictions predicts a 1.36% higher alcohol poisoning mortality. Given the mean restriction of 9 hours for the regions which introduced the restrictions not later than 2006 (see Table A1) their predicted effect is a 12.2% higher alcohol poisoning mortality. For all the regions at the end of the period the mean restriction was 10 hours, so that the predicted effect is 13.6%.[8] To get a better sense of this effect, I estimate the same specification replacing the closing hours variable with log vodka sales to obtain an elasticity coefficient of alcohol poisoning mortality with respect to vodka sales of 0.147. This number suggests that by the predicted effect on alcohol poisoning mortality the hour restriction policy is comparable with a more than doubling of vodka sales.[9]

[8] I calculate the effect using the conventional formula for the log-linear specifications $[\exp(\alpha) - 1] \times 100\%$.

[9] I calculate the comparative effect using the formula: $\exp[\log(a\bar{C} + 1)/coef_{vodka}]$, where $a = [\exp(\alpha) - 1]$, $\bar{C}$ is the mean hour restrictions, and $coef_{vodka}$ is the vodka elasticity coefficient.

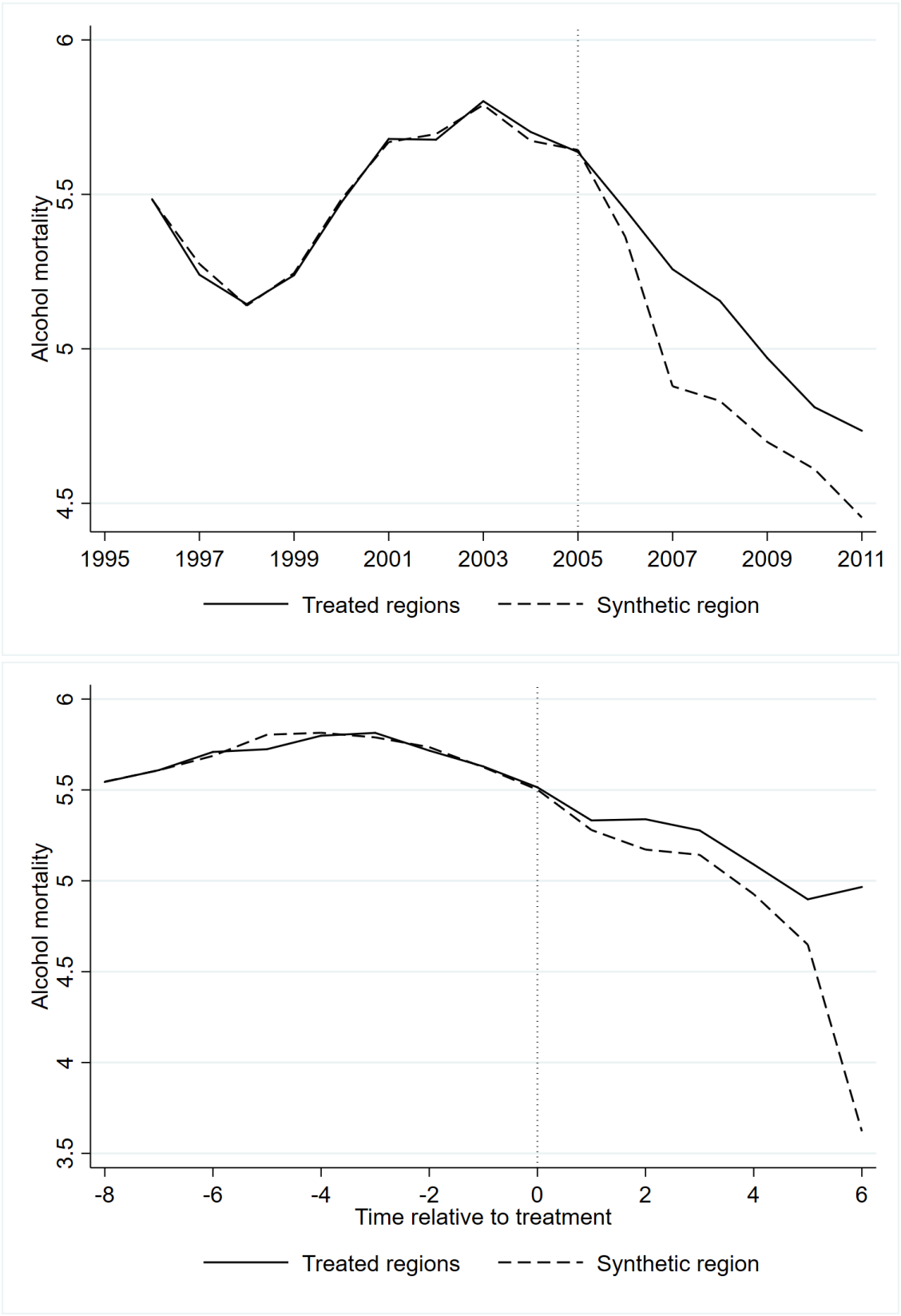


Fig. 5. Real and counterfactual dynamics of alcohol poisoning mortality in the treated regions
Note: The plots above and below present SC and GSC estimates, respectively.

Fig. 5 presents a visualization of the estimates obtained by SC and GSC. Both estimates fit the data well in the pre-treatment period. For the SC estimate, the average absolute difference between

the synthetic and actual values in the pre-treatment period is 0.013 and for the post-treatment period, 0.257, while for the GSC estimate, the absolute differences in the pre- and post-treatment periods are 0.02 and 0.352, respectively. In addition, I obtain the SC estimate with the validation period from 2002 with the results presented in Fig. A5 in Appendix. Now the absolute gaps for the pre-treatment, validation, and post-treatment periods are 0.018, 0.017, and 0.218, respectively. Thus, the out-of-sample prediction for the validation period is as good as the in-sample prediction for the matching period contrasting sharply with that for the post-treatment period.

When using conventional SC, a potential interpolation bias is related to variables for the treated regions which should not go beyond any linear combination of the values for the control group (McClelland and Gault, 2017). Table A2 in Appendix reports the means of the predictor variables for the synthetic control, the treatment group, and 13 regions receiving positive weights when solving for the optimal weight vector (for their mutual location see also the maps in Fig. A in Appendix). The table makes it clear that the values of the synthetic variables are close enough to those for the treatment group, which corresponds to the respective gaps for the outcome of interest. Differences between the means for the treated and control regions are mostly small or within a standard deviation of the respective average for the treatment group (see summary statistics in Table A1), making the groups similar in their means for the predictor variables. Several cases of more pronounced differences concern the regions with very little weights used for constructing the synthetic control, and they do not impact the final result (see below). In addition, I run the same procedure omitting a number of variables with pronounced differences to obtain very similar results.

Both results are consistent with each other and the DD estimates in that the policy effect on alcohol poisoning mortality is positive. SC estimates, presented in the first graph, suggest that the regions introducing the restrictions in 2005 and 2006 would have lower levels of alcohol poisoning mortality if they had not introduced them. This is seen in the dynamics of alcohol poisoning mortality for the synthetic region constructed from the data for regions having introduced the restriction later. Both the treated and synthetic regions show downward dynamics of alcohol poisoning mortality but the slope is steeper for the synthetic region.

The second graph in Fig. 5 visualizes the GSC estimates according to which treated regions show a weaker downward trend in alcohol poisoning mortality than the synthetic region, which is now constructed from all the regions currently not under the restrictions.

To exactly calculate the policy effect based on the SC and GSC estimates, I rely on the gaps between the treated and synthetic outcome over the treatment period presented in Fig. 6. The average gap based on the SC estimate is equal to 0.26. The average gap for the GSC estimate is 0.35. Given the average log alcohol poisoning mortality for the treatment group in 2005 (Table A1 in Appendix) of 5.36, in absolute terms the SC estimate suggests an effect of 30%, while the GSC estimate is consistent with an absolute effect of 42%.

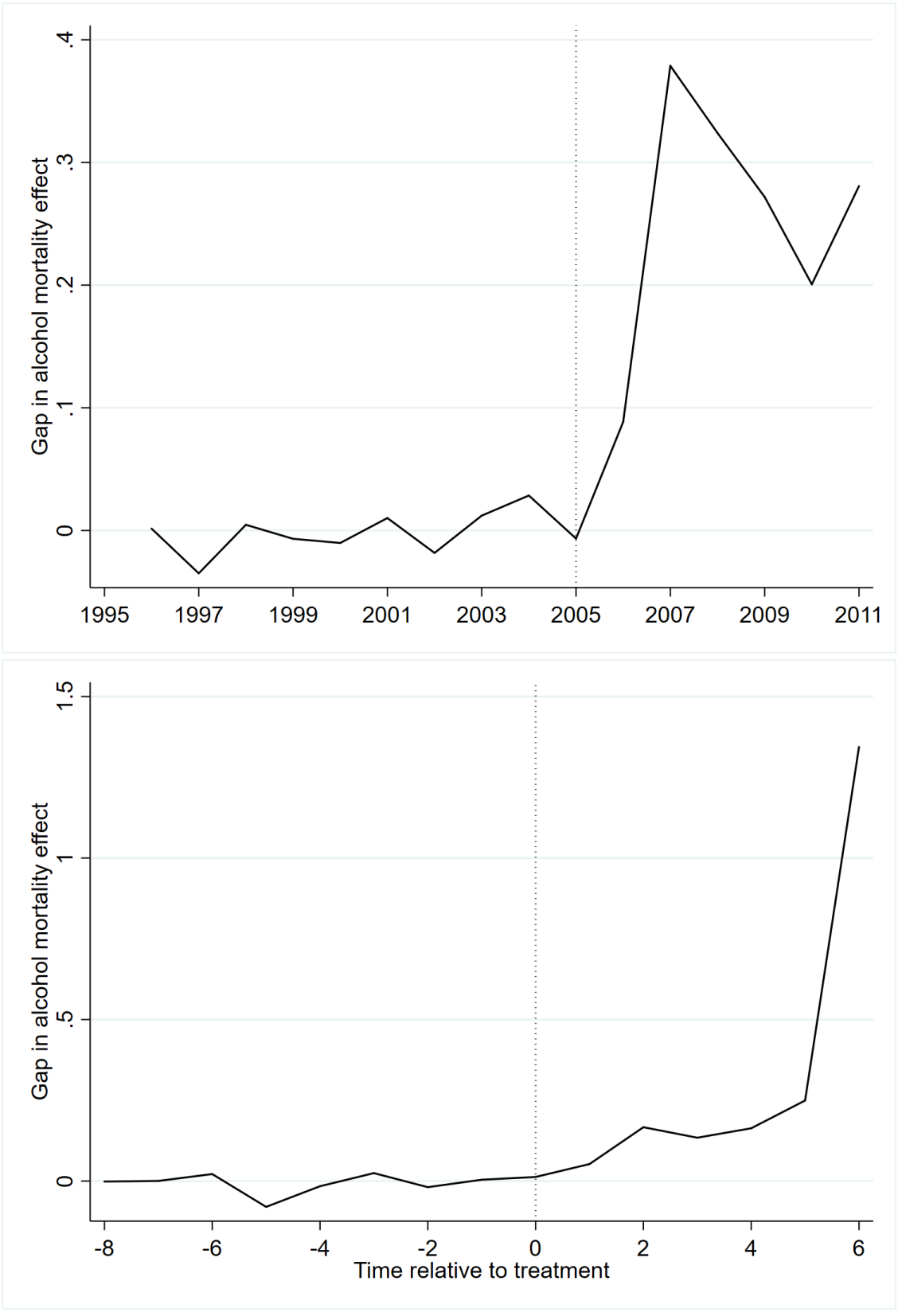


Fig. 6. Alcohol poisoning mortality gap between real and synthetic treated regions
Note: The plots above and below present SC and GSC estimates, respectively.

The three methods used are consistent in that the policy effect is positive in terms of alcohol poisoning mortality. However, they give different levels of the effect. Despite the difference in the

way treatment and control groups are formed, the SC and GSC approaches give similar results, which are substantially higher than the DD result. Given that the DD method is based on stricter assumptions than the SC and GSC methods and the latter best fits the data used, one can conclude that a relatively more relevant method is conducive to a higher positive policy effect.

Table 2
Time-varying effect of closing hour restrictions on alcohol poisoning mortality, 2005-2011

	(1)	(2)	(3)	(4)
Closing hour restrictions	-0.0102	-0.0066	-0.0069	-0.0081
	[0.0124]	[0.0125]	[0.0125]	[0.0125]
Interactions of closing hours and year dummies:				
2007	0.0290**	0.0300**	0.0309**	0.0315**
	[0.0126]	[0.0127]	[0.0127]	[0.0127]
2008	0.0151	0.0137	0.0137	0.0150
	[0.0133]	[0.0135]	[0.0136]	[0.0135]
2009	0.0089	0.0072	0.0071	0.0085
	[0.0136]	[0.0138]	[0.0138]	[0.0138]
2010	0.0234*	0.0212	0.0210	0.0225
	[0.0139]	[0.0141]	[0.0141]	[0.0141]
2011	0.0324**	0.0342**	0.0346**	0.0357**
	[0.0162]	[0.0167]	[0.0168]	[0.0168]
Region level characteristics				
economic	No	Yes	Yes	Yes
demographic	No	No	Yes	Yes
geographic	No	No	No	Yes
Observations	485	469	469	469

Note: GMM stardard errors are in parentheses. *** p<0.01, ** p<0.05, * p<0.1. All the specifications include year fixed effects. For controls see Note to Table 1.

As seen in Fig. 5 and Fig. 6, the policy effect predicted by the SC and GSC estimates varies over the period. In particular, the treatment group, made up of regions which were under the restriction since 2005/2006, show the strongest effects in 2007 and 2011 (the first graph in Fig. 6). This is in line with the DD regression estimates of the year-specific effect of interest presented in Table 2. Significantly positive coefficients in all the specifications are obtained for 2007 and 2011, but, unlike the SC estimate, the DD effect for 2011 is higher than that for 2007. For the remaining years, the coefficients are positive, though insignificant, which may follow from the fact that DD tends to understate the policy effect compared to the SC and GSC estimates. The DD estimates suggest that for all the regions under the restriction the strongest effect is seen in the last year of the treatment period. The GSC gaps plotted in the second graph in Fig. 6 show similar relative effects by year, in that considering all the regions under the restriction the strongest effect is observed in the last year of the treatment period.

The exact levels of the effect for 2007 and 2011 based on the DD estimates are 3.2% and 3.6% higher alcohol poisoning mortality per additional hour of restriction, which given the mean values of alcohol poisoning mortality (see Table A1 in Appendix) is consistent with 29% and 36% higher level of alcohol poisoning mortality in 2007 and 2011. The SC estimates suggest that the regions which were the first to introduce the restriction made their alcohol poisoning mortality levels 46% and 32% higher in 2007 and 2011, respectively. Finally, the GSC estimate suggest a slightly higher effect in the second year of the treatment and monotonously growing positive effect in the three last years of the treatment period. As a whole, the three approaches are consistent in that the effect tends to become stronger with time, which can be explained by the fact that the closing time restrictions tended to extend over the period.

Particular examples of regions which in the treatment period show a positive effect in terms of alcohol poisoning mortality are presented in Fig. A2 in Appendix. The first two graphs present the policy effects for Republic of Mordovia and Penza Region exemplifying the first and last regions to introduce the restrictions. In both cases we see slightly downward dynamics of alcohol poisoning mortality with substantially weaker slope than that for their synthetic counterparts. The last two graphs present the effects for Irkutsk Region and Primoski Region which introduced the restrictions in 2008 and 2009, respectively. These show upward dynamics of alcohol poisoning mortality versus downward ones for their synthetic counterparts.

To sum up the results, the effect of sales restrictions on alcohol poisoning mortality is unambiguously positive and strong.

5.2. Robustness checks

To check the robustness of the results, I perform several analyses using several procedures. The first one is the leave-one-out analysis which consecutively excludes each of the control regions with a positive weight to check whether the estimates substantially depend on a particular control region as a source of the synthetic region. To check whether the SC estimates are sensitive to particular regional weights, I obtained 13 SC estimates excluding each of the control regions with a positive weight. The results are presented in Fig. 7. All the 13 estimates are similar to the main SC estimate in that all of them show a positive gap in the outcome of interest throughout the period. The most substantial differences in the results are seen in estimates excluding the regions with relatively high weights. Nonetheless, the main results are unchanged.

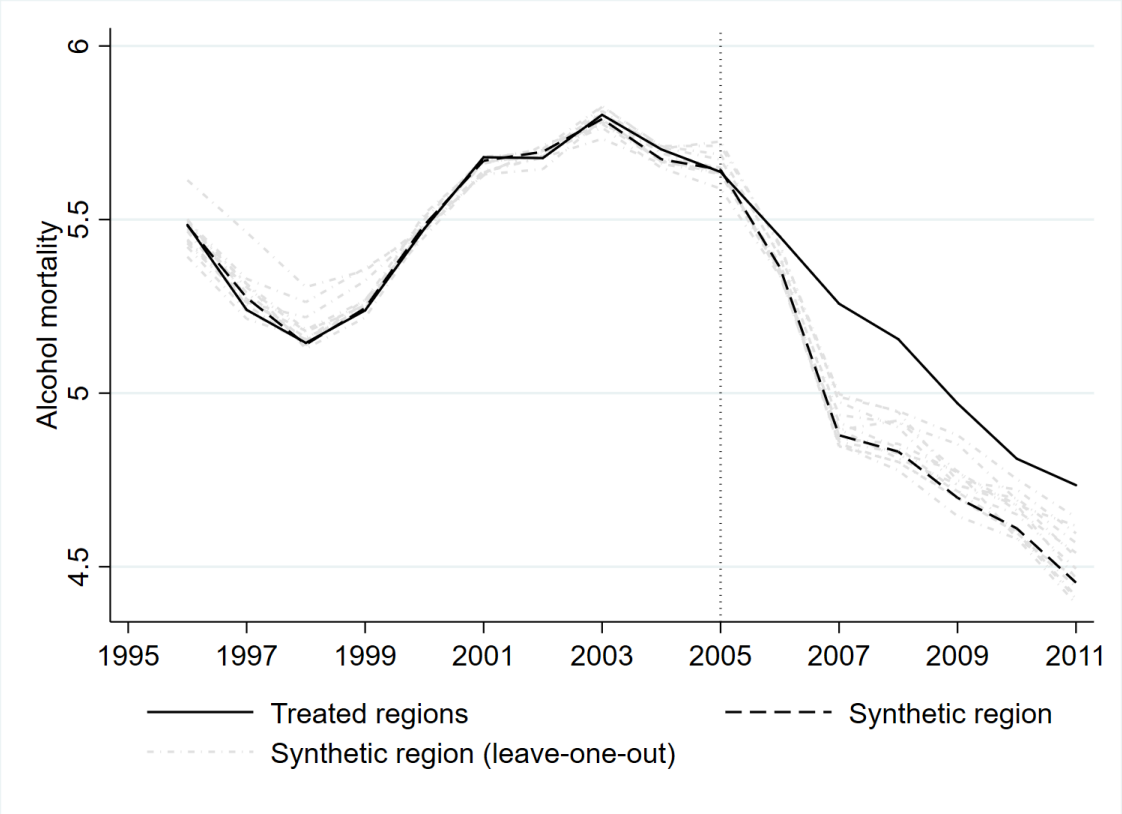
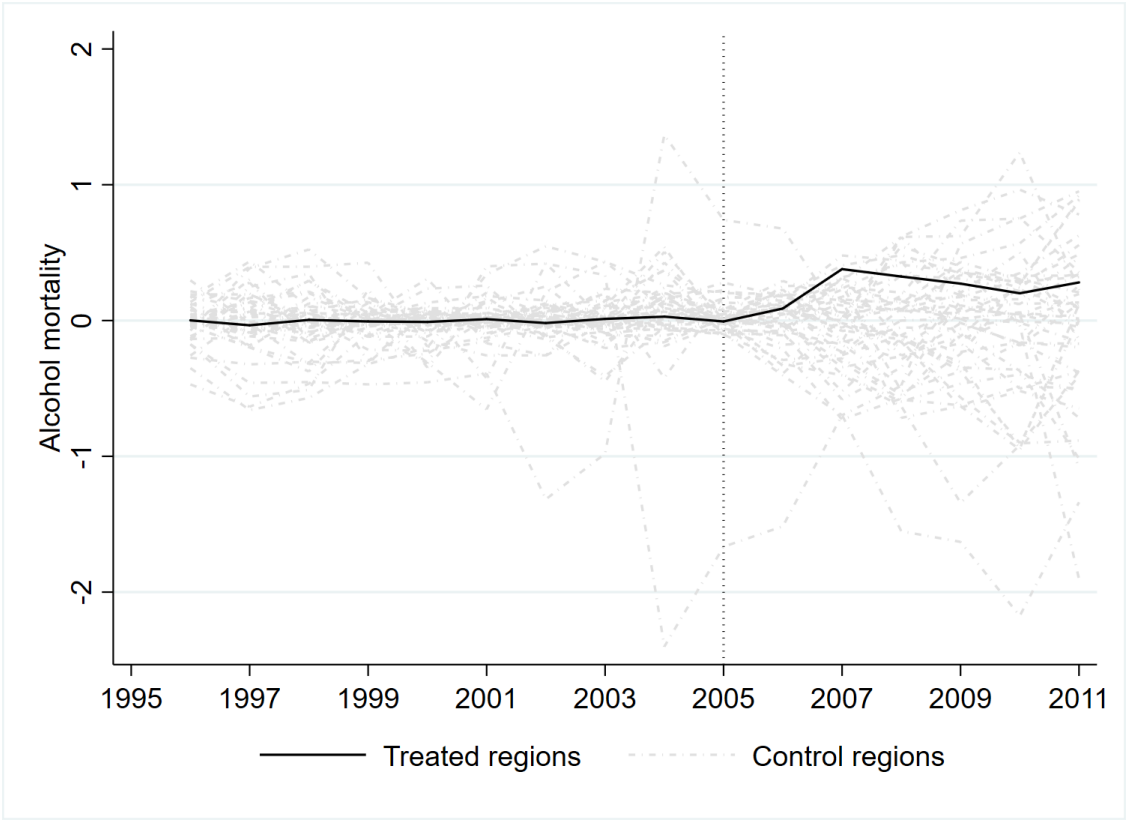


Fig. 7. Leave-one-out robustness check
Note: The plot presents counterfactuals excluding each of the regions with positive weight in the main SC analysis.

Another robustness check is the in-time-placebo performed with the SC estimate. The result is presented in Fig. 8, panel a. The gray lines display the gaps in alcohol poisoning mortality between regions from the control group and their synthetic counterparts. The solid black line superimposed on the gray ones shows the gap estimated for the treated regions. The gap for the treated regions after 2005 is large enough relative to the gaps estimated for the control regions. Nevertheless, there are several gaps for the control regions larger than the gap for the treated regions in some years. This is mostly explained by the poor fit of alcohol poisoning mortality by the SC prior to the treatment period for the respective regions from the control group. If one excludes the regions with the pretreatment MSPE of more than twenty times the MSPE for the treated regions (for the results, see Fig. A6 in Appendix), as is done in Abadie and Heinmueller (2003), the majority of the large gaps for the control regions disappear making the gap for the treated regions more extreme relative to the remaining gaps. In addition, the majority of regions from the control group introduced the policy later in the treatment period. This explains a number of relatively large gaps from the control group in later years. Panel a of Fig. 8 also shows a good fit by the SC for the treated regions before the restrictions were imposed. The same is true for the majority of regions from the control group. However, there are several regions with a relatively poor fit which is explained by their unusual levels of alcohol poisoning mortality which cannot be reproduced by respective time series for the other regions.

a)



b)

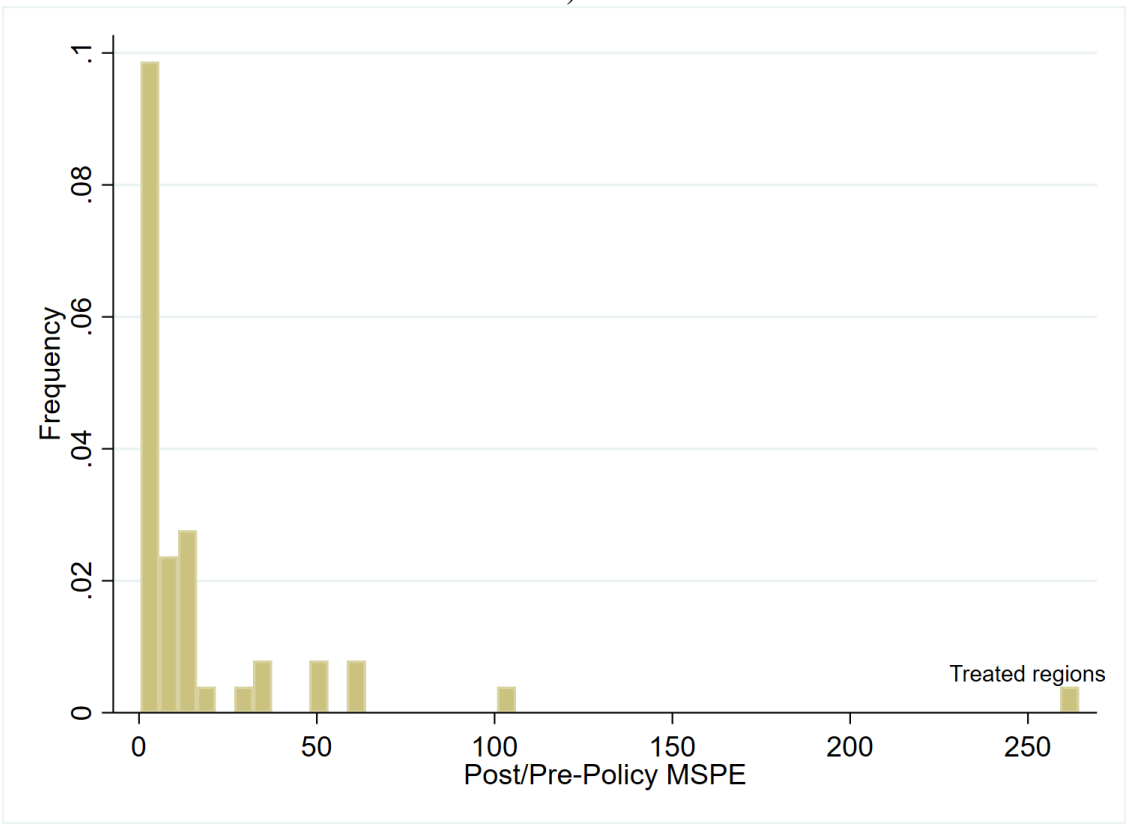


Fig. 8. In-place-placebo robustness check and post/pre-treatment MSPE histogram
Note: The plots above and below present SC gaps in terms of alcohol poisoning mortality for all regions, and distribution of post- to pre-treatment MSPE ratio, respectively.

An additional robustness check is to estimate the ratio of post-treatment MSPE to pre-treatment

MSPE. Pre-treatment MSPE is a measure of the goodness of fit of an SC estimate, while post-treatment MSPE measures the discrepancy between the observed outcome of interest and its synthetic estimate in the post-treatment period, so that the ratio shows the relative difference between the observed and synthetic time series in the post-treatment period. If this difference is small this means that the post-treatment period does not differ much from the pre-treatment period in terms of the outcome of interest. Since control regions differ from treated regions in the intensity of treatment in the post-treatment period, they should show systematically lower ratios than the treated regions. Fig. 8 panel b presents a histogram of the ratios. The ratio for the treated regions is an evident outlier among ratios for the control regions. This is more than two and a half times higher than the second highest ratio for the control region. For 47 control regions the median value of the ratio is 5.1, so that the ratio for the treated region is 52 times higher than this median value. Thus, in this measure the treated regions show unusually high values in comparison to the control regions.[10]

6. Conclusion

The research presented in this paper evaluates the effect of local restrictions on the closing hours for strong alcohol sales on alcohol poisoning mortality the reduction of which should have been one of the principal goals of the policy. To establish a causal link from the policy to the outcome of interest, the analysis uses difference-in-differences and synthetic control methods. The effect of the policy turns out an increase in mortality caused by alcohol poisoning. This result suggests that those inclined to begin or continue their drinking at night replace vodka with alcohol substitutes which are not subject to the time restrictions and are more dangerous for health.

The main implication of these results is that although the closing hours are effective at preventing people from using factory-made strong alcohol, in a country with poor observance of the law and substantial informal alcohol production, such restrictions induce people to substitute alcohol which does not fall under the restrictions and is more harmful. These indirect consequences of the restrictions should be taken into account by decision makers.

The strengths of the research are related to the high reliability and robustness of the results. The design allows systematic differences between observational units exposed to the restrictions and those not to be controlled for, and thereby coping with a major confounding factor. The main limitation of this research relates to the way it uses the conventional synthetic control method. In particular, the choice of the treatment group is based on the year the restriction was introduced rather than whether it was introduced at all. Therefore, the analysis is based on the assumption that regions which introduced the restrictions earlier were more subject to its effect, so that the conclusion is based on whether the policy effects in the distinguished groups actually fit this assumption. However, the alternative methods independent of this assumption, difference-in-differences

[10] In addition, to make sure that the policy has expected immediate effects, I obtained the GSC estimates for the effects for vodka and wine sales as two commercially sold beverages of which the former is directly under the restriction and the latter is a possible substitute. The results are presented in Fig. A3 in Appendix. Expectedly, the estimate for vodka sales suggests a negative effects, which is consistent with Kolosnitsyna et al. (2014), and the estimate for wine sales, on the contraty, indicates a positive effect.

and generalized synthetic controls, support the results obtained with the use of synthetic control method.

Acknowledgments

The author is grateful to Andrey Aistov, Kristian Behrens, Christopher Gerry, Alexander Nemtsov, and Ke-Li Xu for valuable comments. All the views and errors are of the author alone.

Appendix A. Summary statistics, weights, and means of predictor variables

Table A1
Summary statistics

	Regions under restriction in 2005			Remaining regions			difference
	obs	mean	sd	obs	mean	sd	
Panel A: 2005							
Log alcohol poisoning mortality	9	5.36	1.398	73	5.725	1.370	-0.365
Closing hour interval	9	9	2.121	73	0	0	9
Close hours	9	22.89	0.601	73	24	0	-1.11
Open hours	9	7.889	1.616	73	0	0	7.889
Log vodka sales	9	7.03	1.282	73	7.348	1.123	-0.318
Log vodka price	9	4.954	0.0526	73	5.006	0.217	-0.052
Log wine sales	9	6.168	1.176	73	6.46	1.153	-0.292
Log wine price	9	4.819	0.157	73	4.872	0.310	-0.053
Log consumer basket price	9	8.361	0.104	73	8.462	0.211	-0.101
Log GRP per capita	9	11.18	0.358	70	11.34	0.591	-0.16
Share of urban population in %	9	62.28	15.03	73	70.31	12.29	-8.03
Share of laborforce in %	9	62.37	1.461	73	63.47	2.934	-1.1
Log population size	9	6.999	1.142	73	7.102	0.950	-0.103
Log mortality	9	9.839	1.127	73	9.844	1.050	-0.005
Deaths in traf. ac. per 100 th.	9	25.94	7.953	73	24.77	6.611	1.17
Average summer temperature	8	19.46	1.633	72	18.83	2.939	0.63
Average winter temperature	8	-8.425	8.133	72	-9.242	8.576	0.817
Panel B: 2011							
Log alcohol poisoning mortality	9	4.923	0.843	72	4.711	1.402	0.212
Closing hour interval	9	9.333	1	72	10.14	1.586	-0.807
Close hours	9	22.89	0.333	72	22.51	0.872	0.38
Open hours	9	8.222	0.667	72	8.653	0.937	-0.431
Log vodka sales	9	6.856	1.008	71	7.218	0.941	-0.362
Log vodka price	9	5.48	0.0919	71	5.549	0.131	-0.069
Log wine sales	9	6.456	1.184	71	6.718	0.979	-0.262
Log wine price	9	5.329	0.136	71	5.406	0.215	-0.077
Log consumer basket price	9	9.063	0.109	72	9.125	0.174	-0.062
Log GRP per capita	9	12.15	0.228	72	12.42	0.707	-0.27
Share of urban population in %	9	63.83	14.47	72	70.98	12.6	-7.15
Share of laborforce in %	9	60.12	1.057	72	60.98	2.214	-0.86
Log population size	9	6.978	1.14	72	7.129	0.891	-0.151
Log mortality	9	9.66	1.157	72	9.703	0.966	-0.043
Deaths in traf. ac. per 100 th.	9	22.68	4.678	72	21.75	6.867	0.93
Average summer temperature	9	21.33	3.513	72	20.37	4.261	0.96
Average winter temperature	9	-14.5	7.257	72	-12.84	8.036	-1.66

Table A2
Means of predictor variables from the pre-treatment period

Region	Synthetic	Treated	Pskov	Kalining.	Yaroslavl	Yakutia	Murmansk	Khabar.
Weight			0.194	0.131	0.128	0.112	0.107	0.104
Log alcohol poisoning mortality	5.51	5.508	5.301	6.304	6.694	4.704	4.933	5.004
Log vodka price	5.233	5.243	5.201	5.257	5.21	5.286	5.259	5.251
Log consumer basket price	8.228	8.221	8.025	8.213	8.078	8.549	8.498	8.44
Log population size	6.986	6.991	6.655	6.854	7.233	6.882	6.844	7.291
Log GRP per capita	10.396	10.347	9.914	10.142	10.479	11.258	10.867	10.68
Log mortrality	9.713	9.695	9.756	9.598	10.162	9.157	9.284	9.956
Average summer temperature	18.719	18.689	19.68	18.94	20.2	14.52	14.82	17.18
Average winter temperature	-10.911	-11.059	-5	-1.54	-7.66	-35.52	-10.18	-22.46
Log land area	4.578	4.203	4.015	2.715	3.589	8.034	4.976	6.669
Share of urban population in %	72.17	71.222	66.6	77.533	81.067	64.233	92.1	80.683
Deaths in traf. ac. per 100 th.	23.749	24.658	30.283	26.617	26.583	17.85	11.4	23.433
Share of laborforce in %	60.854	60.835	57.61	62.16	58.98	62.84	67.3	64.06

Region	Belgorod	Krasnodar	Tuva	Kar.-Ch	Tatarstan	Kalmykia	Astrakhan
Weight	0.1	0.039	0.034	0.021	0.015	0.009	0.007
Log alcohol poisoning mortality	5.686	6.465	5.407	3.507	5.645	2.645	5.3
Log vodka price	5.215	5.175	5.304	5.075	5.071	5.053	5.101
Log consumer basket price	8.037	8.135	8.176	8.003	8.029	8.061	8.085
Log population size	7.314	8.532	5.728	6.082	8.237	5.715	6.918
Log GRP per capita	10.295	10.182	9.499	9.565	10.705	9.565	10.144
Log mortrality	10.05	11.25	8.336	8.5	10.8	8.111	9.578
Average summer temperature	21.22	24.06	18	21.18	20.56	25.54	25.8
Average winter temperature	-3.88	2.46	-25.98	-1.22	-8.5	-1.76	-1.62
Log land area	3.3	4.324	5.128	2.66	4.217	4.313	3.892
Share of urban population in %	65.417	53.25	51.55	44.083	74.05	43.917	67.6
Deaths in traf. ac. per 100 th.	19.967	24.633	35.217	22	21.517	29	18.35
Share of laborforce in %	58.05	57.89	57.9	57.74	59.1	59.29	59.97

Appendix B. Maps

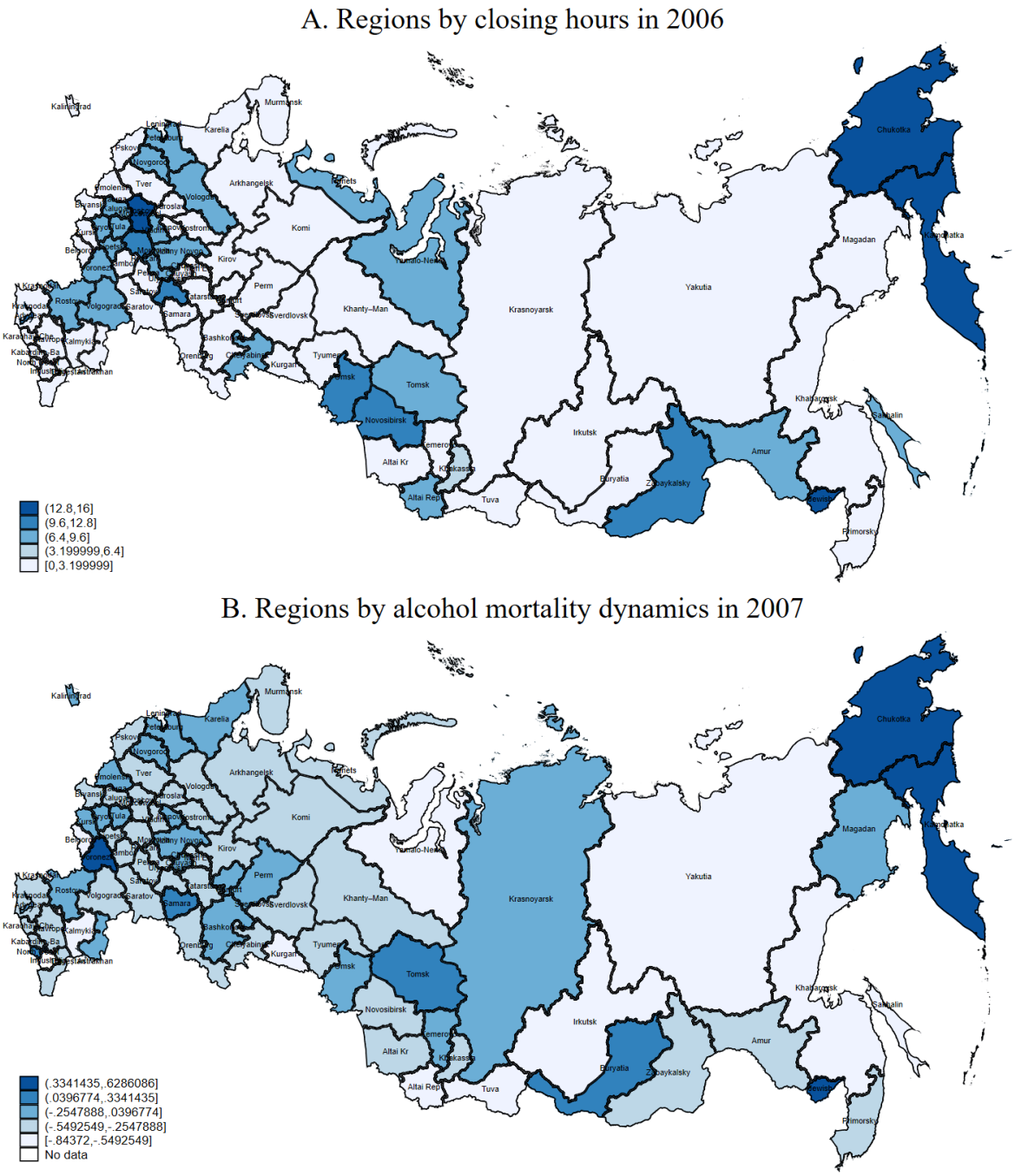


Fig. A1. Russian regions by closing hours restrictions and alcohol poisoning dynamics, 2006
Notes: Shading in map A is proportional to closing hour restrictions (in hours a night alcohol sale is prohibited), and shading in map B is proportional to alcohol poisoning mortality dynamics.

Appendix C. Counterfactuals in particular regions

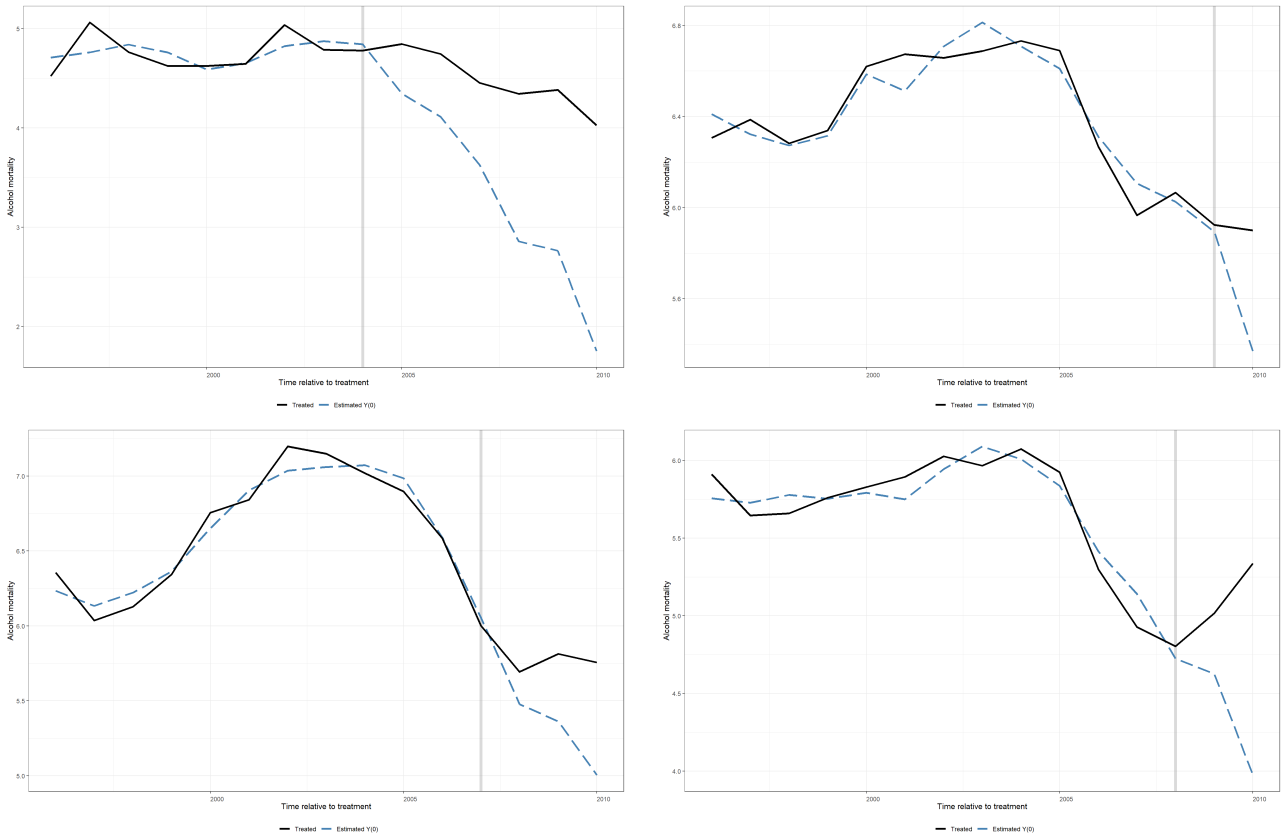
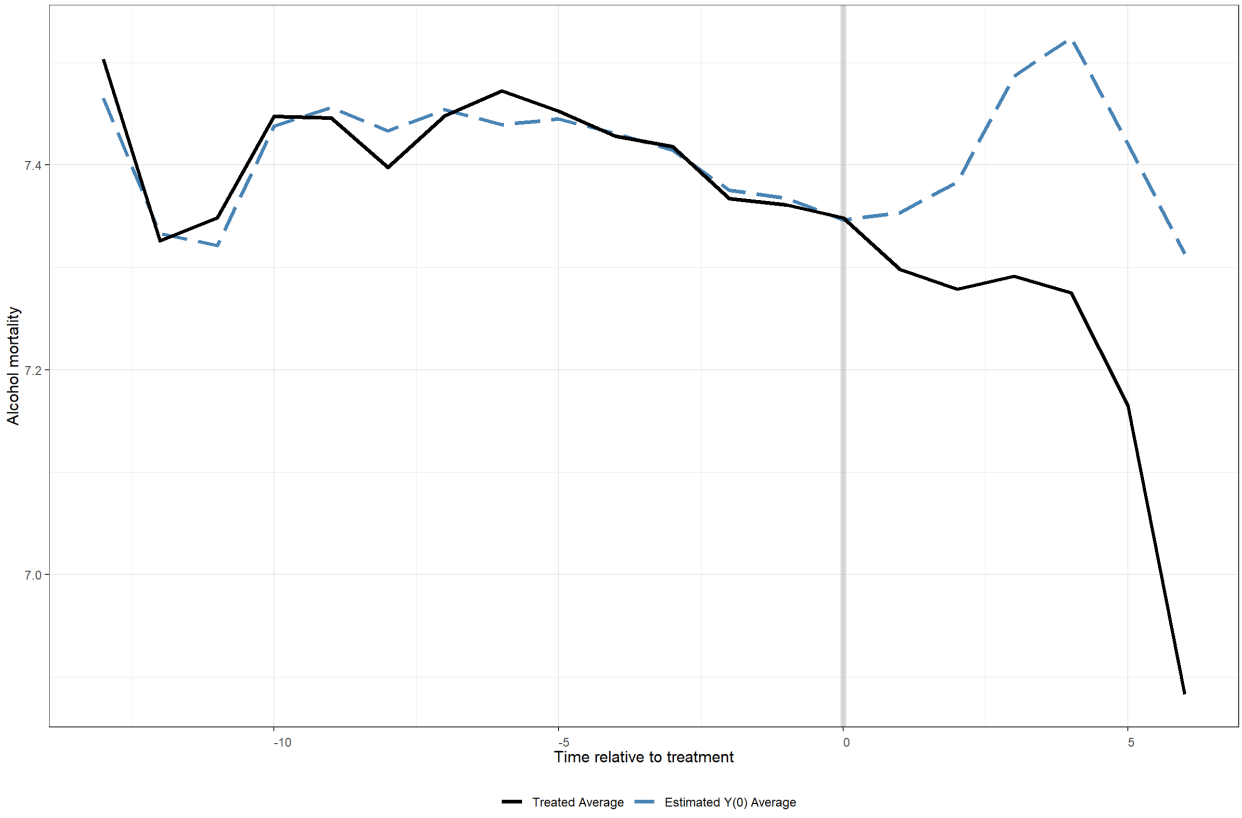


Fig. A2. Real and counterfactual dynamics of alcohol poisoning mortality in particular regions
Note: The GSC estimates are given clockwise from upper-left for following regions: Mordovia, Penza, Irkutsk, Primorsky.

Appendix D. Counterfactuals for alcohol sales



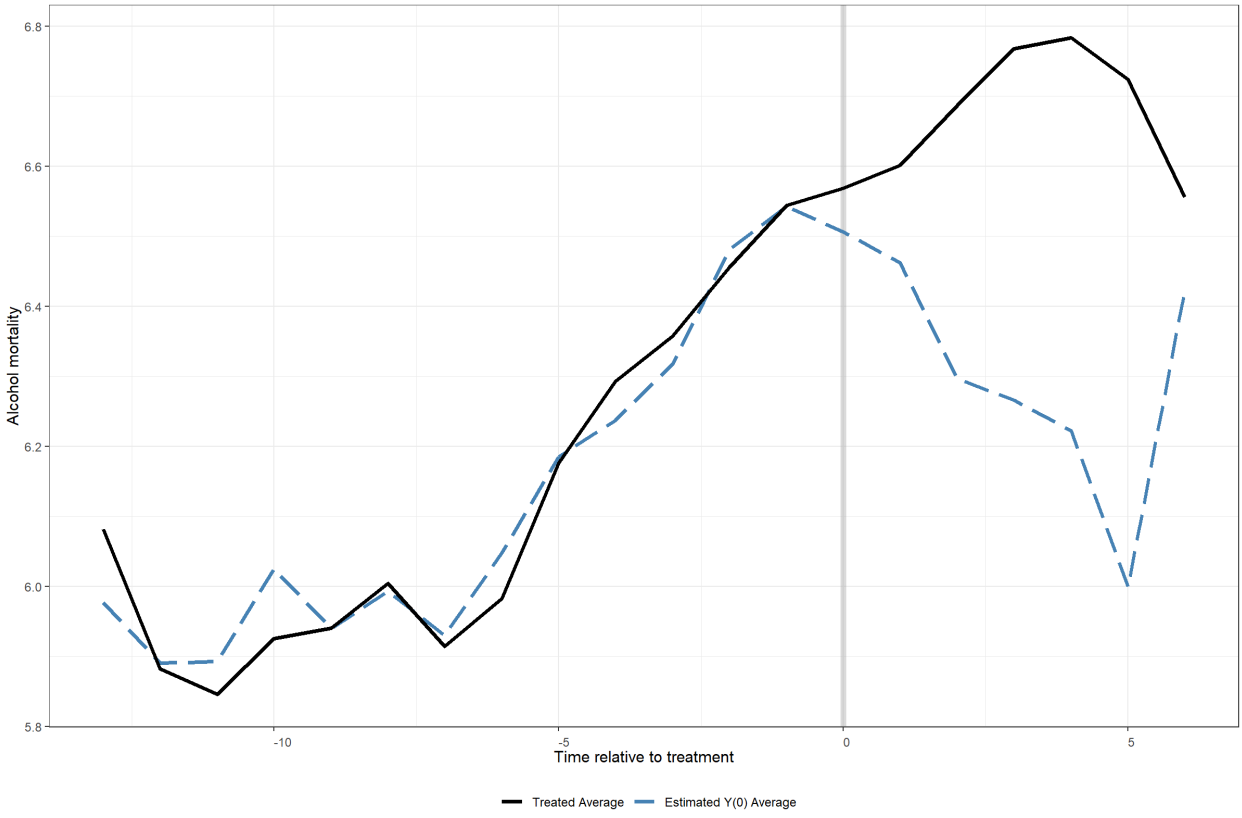


Fig. A3. Real and counterfactual dynamics of alcohol sales in the treated regions
Note: The GSC estimates above and below are given for vodka and wine sales, respectively.

Appendix E. Alternative control group

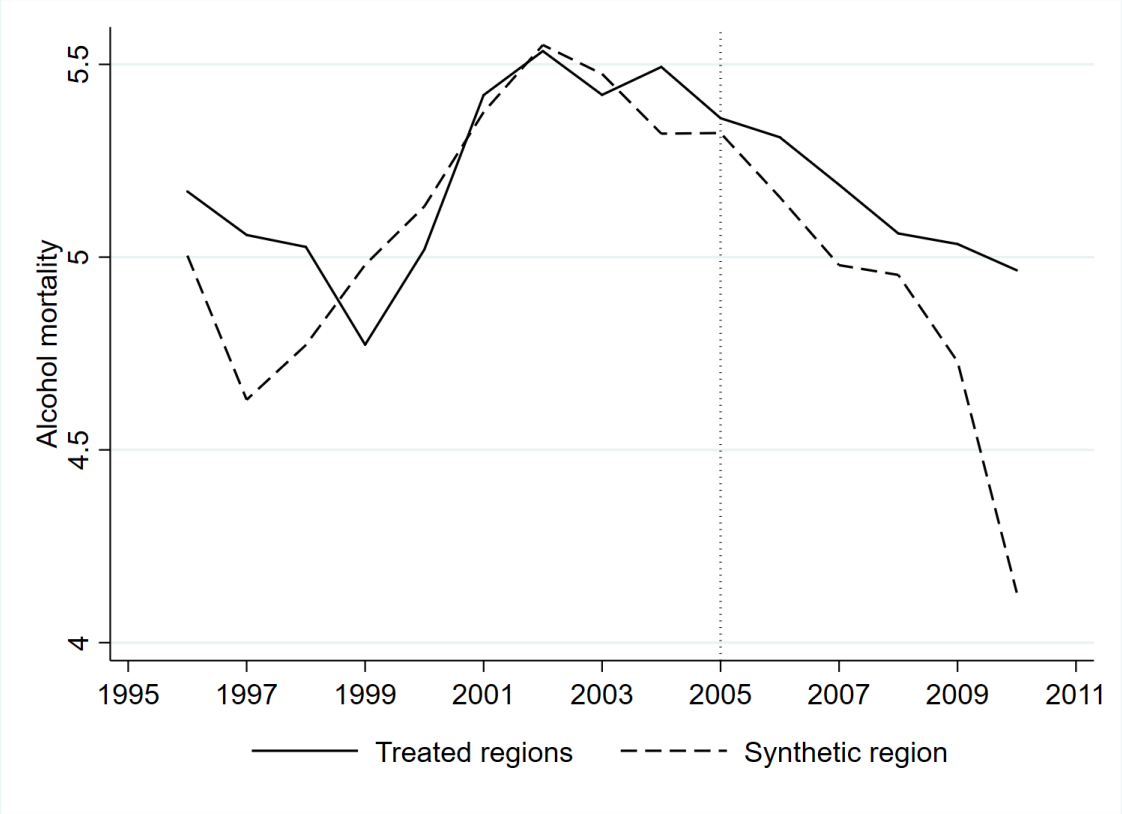


Fig. A4. SC counterfactual with control regions imposing the restriction after 2010

Appendix F. Estimation with validation period

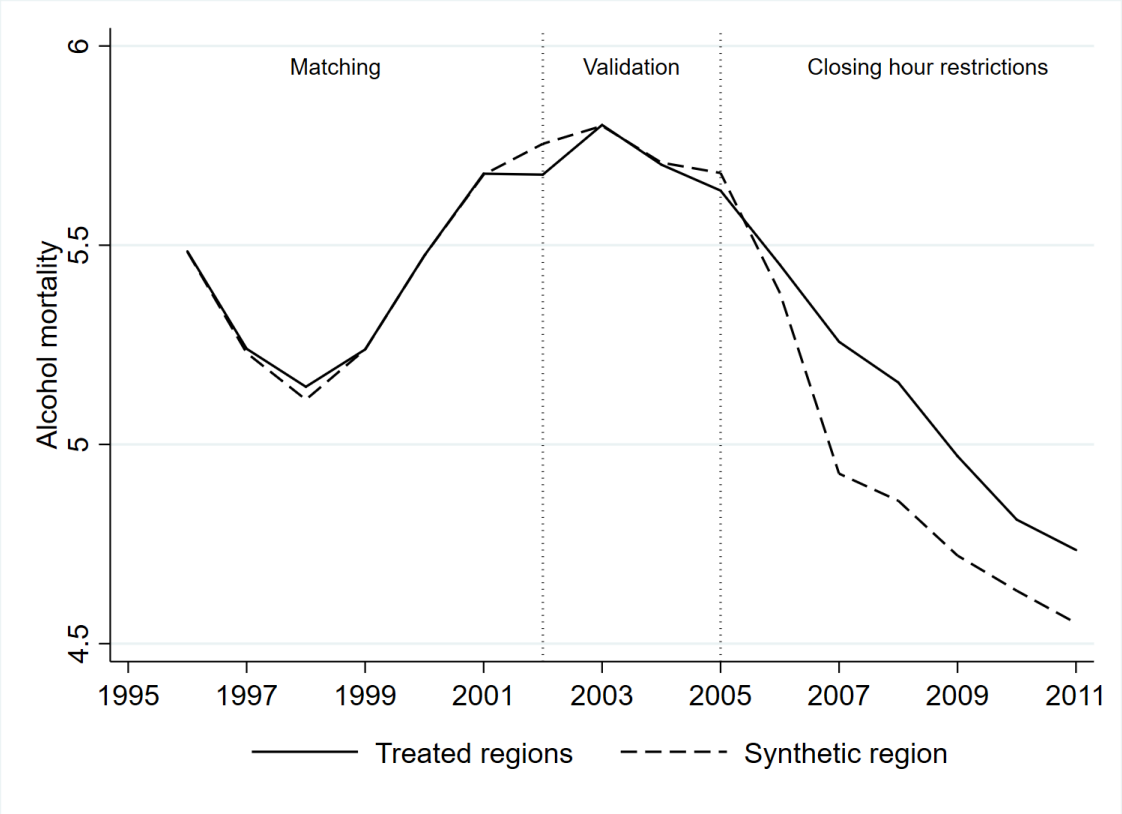


Fig. A5. SC counterfactual with the validation period

Appendix F. In-place-placebo with a smaller group of control regions

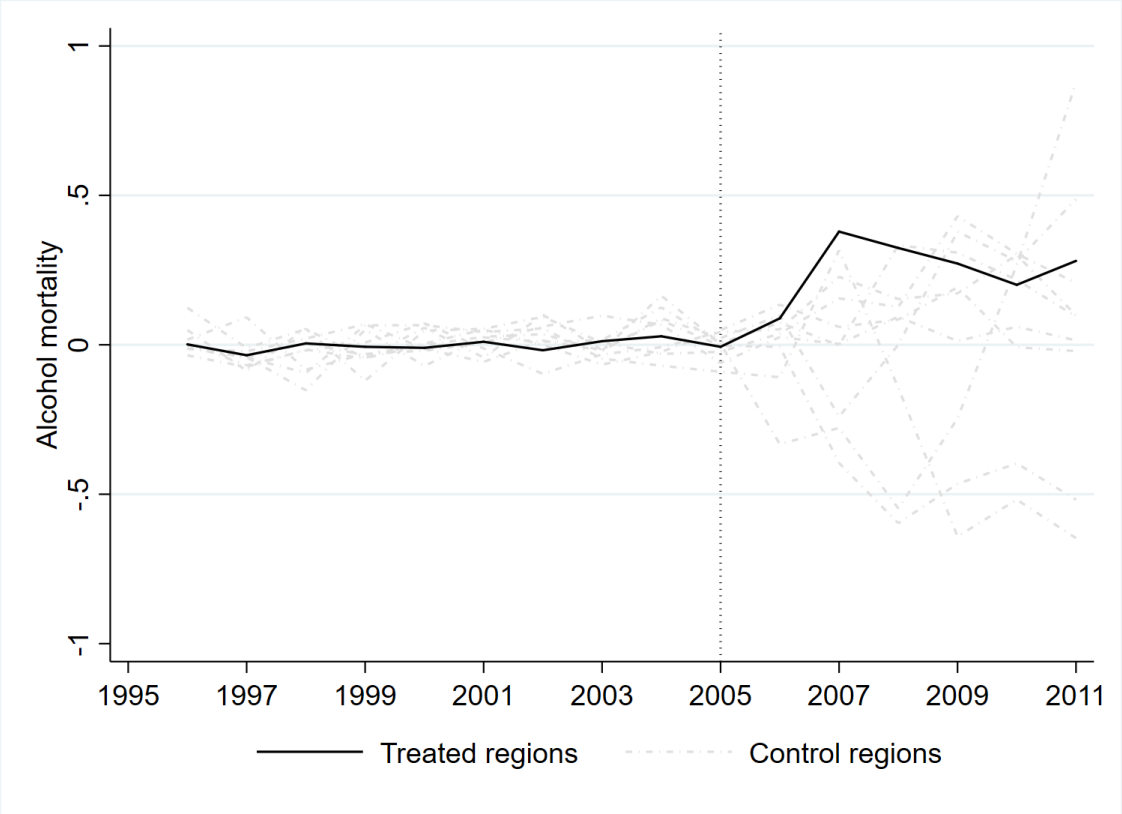


Fig. A6. In-place-placebo robustness check
Notes: The analysis excludes regions with the pre-treatment MSPE twenty times higher than the treated one's.

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