Sobering Up: The Impact of the 1985-1988 Russian Anti-Alcohol Campaign on Child Health

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Abstract: This paper estimates the impact of parental alcohol consumption on child health by taking advantage of a unique shock to alcohol supply: the 1985 to 1988 alcohol prohibition campaign in Russia. This campaign was temporally short lived, and resulted in large amounts of exogenous geographic variation in its intensity and effectiveness. I construct a new data set that combines the Russian Longitudinal Monitoring Survey with regional alcohol data. Using both a differences-in-differences approach, as well as instrumental variables methods, I find significant improvements in child height, immunization rates, and chronic conditions among boys born during prohibition who also lived in regions with effective anti-alcohol campaigns. This confirms the effect of investments during a child's fetal period and first two years of life on long-term health measures, and demonstrates a potential positive effect of suppressing parental access to alcohol. Furthermore, evidence from vaccination rates suggests that the positive effect of prohibition on child health occurred through improvements in parental time, rather than income resources.

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1. Introduction

Parental alcohol abuse affects millions of children worldwide. One out of ten American children and one out of eight European children live in a household with at least one alcohol dependent or alcohol-abusing parent (Huang et al.1996, Eurocare1998). The economics literature has mostly focused on the effects of parental alcohol consumption on child abuse and mental health (Jones et al.1999; Markowitz 2000; Grossman and Markowitz 1998, 2000; Chatterji and Markowitz 2001, Nilsson 2008), but parental drinking can have a significant impact on other aspects of child health as well. In recent work, for instance, Bonu et al. (2004) document that children from Indian households that used tobacco or alcohol were more likely to have acute respiratory tract infection, more likely to be malnourished, and more likely to die before their first birthday.[1]

Despite the existence of a positive correlation between substance abuse by parents and adverse physical and mental health outcomes in children, establishing a causal relationship has proven difficult. The observed relationship may be causal if alcohol consumption has a direct impact on parenting ability or the amount of resources that parents invest in children. On the other hand, the relationship may be the result of unobserved factors that are correlated with both parental alcohol consumption and child outcomes, such as parental psychiatric disorder, stressful home environment, or living in a dangerous neighborhood. To control for these confounding factors, researchers have used (1) child and family specific fixed effects models, which control for unobserved heterogeneity at the level of the child, and parents' family of birth, respectively; (2) instrumental variables methods, which use state alcohol prices and policies to identify parents' alcohol consumption (Markowitz 2000, Grossman and Markowitz 1998, 2000, Chatterji and Markowitz 2000).

[1] See Gmel and Rehm (2003) for an extensive review of the possible effects of alcohol consumption on child and relatives' lives.

This paper extends this area of research by examining the long run impact of the 1985-1988 anti-alcohol campaign in Russia. The primary data source is the Russian Longitudinal Monitoring Survey, a rich longitudinal data set on child outcomes, parental health, and other family characteristics, which is combined with both official and unofficial regional alcohol data.

The contributions of this paper are several. First, I focus on physical (height, chronic health conditions, and immunizations) rather than mental measures of child health. This diminishes the problem of certain confounding factors—such as genes and personality—being correlated with parental alcohol consumption, since these factors are much more likely to influence child mental outcomes rather than physical health. Second, by focusing on national rather than state (local) alcohol policy and on a time period when internal migration in Russia was restricted, the endogeneity of families' location in response to changes in alcohol prices and programs is not an issue in the estimation.[2] Third, I show not only that restrictive alcohol policies can have a large positive effect on child physical health, but also that this effect occurs even in heavy drinking environments, and that it can persist in the longer run. The results in this paper therefore also add to the growing literature of the impact of early life conditions on later life outcomes (see for instance Currie 2007 for a recent review). In addition, I also present some new evidence on the channels through which parental alcohol consumption affects child health. In particular, I show that, in Russia between 1985-1988, parental time inputs might have been more important contributors to child health than parental monetary investments.

This paper also contributes to the literature on the effects of the 1985 to 1988 anti-alcohol campaign in Russia, and on the longer-term effect of prohibitions more generally. The effect of the Russian prohibition on (adult) health has been hotly debated. Some authors have argued that

[2] Nilsson (2008) also uses an alcohol policy experiment (albeit a regional one), namely the increase in alcohol availability in two Swedish regions following expansions in the marketing of strong beer in 1967-1968. He finds that children born to mothers younger than 21 years old who were exposed to the alcohol experiment in utero were more likely to have poor education and job outcomes later in life.

the prohibition was associated with dramatic decreases in adult mortality, as well as with reduced crime incidence (Chen et al., 1996, Nemtsov and Shkolnikov 1997, Cockerham, 1997, Bennett et al. 1998, Becker and Hemley 1998; Brainerd, 1998, Notzon et al., 1998; Leon and Shkolnikov 1998, Shkolnikov et al., 1998, Walberg et al., 1998, Nemtsov 2000, Brainerd 2006). Other authors, however, have argued that the beneficial health and social effects of the anti-alcohol campaign have been significantly overstated due to problems with both the official alcohol data and the mortality calculations (Treml 1991, Joyce 1992, Treml 1997, Levine 1997).

This paper adds to this literature by focusing on a previously unexplored health outcome—long-run child health—, and by using a new empirical approach that addresses the joint determination of alcohol consumption changes and health outcomes. By combining both official and unofficial alcohol data at a regional level with child outcome measures at the individual level, I show that the campaign led to significant long run improvements in child height, immunization rates, and chronic conditions among prohibition cohorts who lived in regions with effective anti-alcohol campaigns. Furthermore, these effects were strongest among the more vulnerable groups (boys), and at early ages, which confirms the effect of investments during a child's fetal period and first two years of life on long-term health measures, and demonstrates a potential positive effect of suppressing parental access to alcohol.

I use to main econometric strategies in this paper: a differences-in-differences approach, and instrumental variables estimation. I begin by exploiting the cross-regional variation in prohibition intensity in combination with cohort variation in exposure to parental alcohol consumption, controlling for time and region-invariant factors, as well as a rich set of individual and region-level covariates. Although I use several different proxies for the intensity of prohibition—measures of both registered and unregistered alcohol consumption and production—, measurement error is still a concern. Furthermore, since alcohol consumption

changes due to the prohibition and child health outcomes are both functions of government and party behavior, potential endogeneity problems remain. To address these issues, I also develop an instrumental variable procedure that isolates a source of variation in homemade alcohol—sugar consumption quotas and production—that is exogenous to child outcomes.

The paper proceeds as follows. Section 2 provides a background on the alcohol campaign, and section 3 describes the data. In section 4, I discuss the empirical strategy, and section 5 presents the results. Section 6 concludes.

2. Background

2.1 Alcohol Consumption and the 1985-1988 Anti-Alcohol Campaign

In the Soviet centrally planned economy, the state had a complete monopoly on the legal production, pricing, foreign trade, and distribution of alcohol. Since excise taxes and state profits from alcohol sales represented a large fraction of Soviet government revenues, it is perhaps not surprising that between 1960 and 1984, the sale and production of alcohol in the Soviet Union more than doubled, from 4.6 to 10.5 liters of pure alcohol per capita (Figure 1).[3]

In the 1980s, recorded alcohol consumption per capita in Russia was higher than alcohol consumption in most OECD countries.[4] Alcohol consumption was rapidly becoming a serious societal problem: the age at which people started drinking was falling rapidly, an increasing number of women and teenagers were becoming serious drinkers, and, in some cities, average consumption among working age adults was a bottle of vodka per day (White 1996).

Prior to 1985, there had been some half-hearted attempts on the part of the Soviet government to address the issue of alcohol abuse. Two anti-alcohol resolutions of the Central Committee of the Communist Party (CCCP) had been released in 1958 and in 1972 under

[3] 10.5 liters of pure alcohol per capita is roughly the equivalent of 22 liters of 100-proof vodka per person per year.
[4] The country with the highest alcohol consumption per capita in 1990 was France (12.7 liters), but most other OECD countries had per capita alcohol consumption in the 5-9 liters per capita range.

Brezhnev, and after 1982 some action was initiated by Andropov and Chernenko under the general heading of redressing "anti-social behavior". None of these measures had met with much success, however (Nemtsov and Shkolnikov 1997, McKee 1999, Richardson 1999).[5] The anti-alcohol campaigns prior to 1985 had attempted to address the alcohol issue through public health education approaches, encouraging moderate drinking, the substitution of wine or beer for vodka, and increasing intolerance towards drunk-driving and drunkenness in the workplace. When Gorbachev succeeded Chernenko in 1985, however, these measures were rejected as half-hearted, and replaced instead by an all-out war against alcohol.[6]

The anti-alcohol campaign was announced in April 1985 and was initiated in earnest in May-June 1985. It included a wide array of punitive measures: alcohol was banned at all official functions and in public places; party officials and managers who drank heavily were dismissed, earned party "demerit" points, and were publicly criticized; alcohol prices were steeply raised; the minimum age for drinking was increased from 18 to 21; the penalties for public drunkenness, drinking in the workplace, drunk driving and the production and sale of home-made *samogon* (moonshine) were raised and more strictly enforced (Ivanets and Lukomskaya 1990, McKee 1999, Tarschys 1993). Finally, and most importantly, the state production and sale of alcohol was massively reduced; by 1987, the number of stores selling wine and vodka in Russia was five times lower than in 1984, and the agricultural acreage for wine grapes was thirty percent lower (Nemtsov and Shkolnikov 1997).

These measures had some very strong and immediate effects. The queues at official alcohol outlets became as long as 3000 people each day, road traffic accidents and work absenteeism due to alcohol-related causes decreased, and state receipts from alcohol sales

[5] In 1983, following a call by Chernenko for stricter enforcement of the existing alcohol legislation, alcohol consumption finally started falling, though by very little (McKee 1999, see also Figure2).
[6] Within a few months of his designation as new Secretary General, Gorbachev became known as 'Mineral Water Secretary' due to his radical stance on drinking (Tarschys 1993).

plummeted.[7] As can be seen from Figure 1, between 1985 and 1987, recorded alcohol consumption dropped 54 percent, from 8.8 to 3.9 liters of pure alcohol per capita (Treml 1997, Ivanets and Lukomskaya, 1990). Sales of registered alcohol in Russia show a decline of a similar magnitude, from 10.5 liters to 3.9 liters during the same time period (Nemtsov 2000; Ryan 1995). Furthermore, as Figure 2 shows, the consumption of all types of state-produced alcoholic beverages fell during this time period: the consumption of vodka and wine by 55 percent, and that of beer by 26 percent, respectively.

The magnitude of the recorded drop in alcohol consumption was notable. Such a success had never been attained in any other country (apart from times of absolute prohibition or warfare) during such a short time period (Treml 1992, White 1996). The official alcohol consumption data figures do not include the consumption of homemade alcoholic beverages (*samogon*), however. Even before the start of the anti-alcohol campaign, *samogon* consumption was as high as 30 percent of the official alcohol consumption (Nemtsov and Shkolnikov 1997, Treml 1997). The anti-alcohol campaign initially included severe penalties for the production and sale of *samogon*, but as a result of the loosening of political restrictions ('glasnost') in the late 1980s, the prosecution of minor law-breaking offenses, including alcohol-related ones, declined over time. As a result, the anti-alcohol campaign was associated with an increase in *samogon* consumption, especially after June 1987 when first time convictions for home brewing of alcohol became a non-criminal offense (McKee 1999, Nemtsov 2000).

Since the production and purchase of *samogon* in Soviet Russia during most of the campaign was illegal, the exact quantification of the increase in *samogon* consumption is difficult, and has been the subject of a lot of debate in the literature. Levine and Levine (1988,1989) and Zaigraev (1997), for instance, conclude that the drop in recorded alcohol

[7] State revenues from alcohol fell by 5 billion rubles between 1984 and 1985, and by 15.8 and 16.3 billion rubles, respectively, in the following years (McKee 1999).

consumption was fully compensated by an increase in the unofficial one. By contrast, the 1989 Soviet statistical agency (Goskomstat SSR)'s estimates indicate a smaller—but nevertheless very substantial (26.5 percent)—drop in net alcohol consumption once *samogon* is taken into account. Goskomstat's estimates (at least prior to 1988) are more similar to other researchers' findings, which also suggest an overall 25 to 35 percent decline (Treml 1991, Nemtsov and Nechaev 1991, Nemtsov 1992, Lehto 1997, Shkolnikov and Mesle 1996, Cockerham, 2000).

The differences between these studies are due to the inherent difficulties associated with estimating underground alcohol consumption, but also to the use of different timelines regarding the duration of the campaign (Reitan 2001).[8] Once the timing differences are taken into account, there seems to be much broader agreement in the literature regarding the direction of the changes in alcohol consumption—although not necessarily regarding the exact magnitudes: i) the campaign had some positive impact on net alcohol consumption, during its early stages at least ii) the effect of the campaign on alcohol consumption weakened progressively during 1987-1988 due to increases in *samogon* consumption, as well as official gradual de-escalation.

By the late 1987, Russian government finances were increasingly strained due to the absence of alcohol profits, and the anti-alcohol campaign was becoming increasingly unpopular. In January of 1988, Moscow authorities responded to the numerous complaints about the unavailability of vodka by increasing the number of outlets and trading hours, and in October 1988 the production of alcohol across the Soviet Union was increased so as to eliminate queues, effectively (if not officially) ending the anti-alcohol campaign (Tarschys 1993).

[8] The start dates for the anti-alcohol campaign vary from late 1984 (thus including Gorbatchev's predecessors measures), to the various important dates in the spring of 1985: the date when the campaign was officially announced (April 1st), that when the party guidelines were made public (May 17th), and the official implementation date (June 1st) respectively. Suggested end dates include early 1987, July 1987 (when personal *samogon* use was decriminalized), January 1988 and October 1988 (see text) and even 1990 and 1991 in some cases (since on paper the campaign was not fully terminated, and since it wasn't until 1991 that registered alcohol consumption finally rose back up to its 1985 level).

The backsliding in alcohol consumption during the late years of the campaign and in its aftermath was of large magnitude. Following the hyperinflation of the early 1990s—during which the price of alcohol decreased sharply relative to personal salaries and the general price index—alcohol consumption rose back to close to its pre-1985 level (see Figure 2).[9] Furthermore, hard alcohol now made up to 90 percent of the total intake compared to 60 percent in the early 1980s (Levine, 1997), and alcohol abuse had spread to the younger cohorts (Joyce, 1992; White, 1996). As Nemtsov (2000) concludes, "[I]t is highly probable that the positive results of the antialcohol campaign went by the board in the years after the campaign" (p. 141).

Since the magnitude of the changes in alcohol consumption due to the anti-alcohol campaign has been so hotly disputed, it is perhaps not surprising that their effect on adult health (measured by mortality and life expectancy) has been widely debated as well. The official statistics showed that mortality (especially male mortality from accidents, violence and poisonings) declined in the two and a half years after the debut of the campaign, and many researchers concur with this assessment, though the actual estimates for the size of the mortality decline vary significantly among these studies.[10] Some other researchers, however, argue that the mortality declines had started even prior to 1985—and thus the changes during the campaign represented continuations of earlier trends (Kingkade 1988, Blum and Monnier 1989); that their magnitude seems implausible and represents an artifact of the poor quality of official data (Treml 1991, 1997); and some researchers even conclude that the campaign actually "adversely affected people's health" (Butenko and Razlogov 1997).

[9] The general price index increased 1229 times between December 1992 and June 1994, whereas alcohol prices increased only 421 times during this time period. As a result, real alcohol consumption during the early 1990s increased sharply (Shkolnikov and Nemtsov 1997).

[10] The official statistics indicated a 54 percent decline in deaths from alcohol poisoning, 34 percent decline in other violent deaths, and a 26 percent reduction in deaths from liver cirrhosis (Nemtsov 2000). For assessments of the effect of the campaign on adult health see for instance Treml 1991, Shkolnikov and Vassin 1994, Chen et al., 1996; Nemtsov and Krasovsky 1996; Cockerham, 1997, Treml 1997, Bennett et al. 1998, Becker and Hemley 1998; Brainerd, 1998, Notzon et al., 1998; Leon and Shkolnikov 1998, Shkolnikov et al., 1998, Walberg et al., 1998, Nemtsov 2000, Brainerd 2006

Ultimately, evaluating the demographic and health benefits of the campaign is a complicated task, due to the difficulties in measuring unregistered alcohol consumption, as well as the fact that the mortality and health data published during the time period was most likely manipulated for political purposes in order to overstate the achievements of the campaign (Treml, 1991, Zvidrins and Krumins 1993, White 1996, Levine 1997).

In order to address these difficulties, I take a different approach from the existing literature, and focus on longer-term child health outcomes (measured in 1995), for which data reliability is not an issue. In order to isolate the effect of parental alcohol consumption during the campaign on child health seven years later, I use measures (discussed in greater detail in section 3) for which early childhood inputs are essential: height, immunization rates (which have specific age-schedules for being administered), and the incidence of chronic conditions.

2.2 Conceptual Framework

Child health is a function of genetic endowments, in utero health, as well as nutrition and other forms of health and non-health investments during childhood. Exposure to parental alcohol consumption can negatively affect physical child health in two primary ways: through alcohol consumption by pregnant mothers, and by diminishing the parents' financial inputs and time available for childcare (Bonu et al.2004).

Heavy maternal alcohol consumption during pregnancy can have a wide range of effects on fetal health and development, commonly described as fetal alcohol spectrum disorders (FASDs). The possible effects include a wide range of neurological abnormalities, behavioral and motor skills problems, as well as physical anomalies (facial abnormalities, birth defects, and growth deficiencies), and have been extensively analyzed in the medical literature (Floyd et al. 2005, Jones and Smith 1973, Goodlett and Horn 2001). While it is well recognized that some of these effects can be lifelong lasting, most of the literature has focused on the shorter run

consequences of fetal exposure to alcohol, and on ways to attenuate them.[11] The extent of the long run consequences of maternal alcohol consumption during pregnancy on child health, especially physical health, is much less known, however.[12]

Postnatal parental alcohol consumption can also negatively impact child health by reducing parental time and income resources. For instance, increased alcohol expenditures and time spent drinking reduce household income, as well as the time available for childcare. In addition, alcohol consumption can also adversely affect the health of the parents, both directly (by causing liver problems for instance) and indirectly (through domestic violence)—thus further decreasing parental income and time resources available for producing child health.

2.3 Parental Alcohol Consumption and the Anti-Alcohol Campaign

The anti-alcohol campaign did not include public health measures, or policies aimed at the underlying causes of alcohol abuse, or on prevention and treatment (Shkolnikov and Mesle 1996, White 1996).[13]Our discussion in the previous section, however, suggests that the campaign did have substantial potential to affect child health through its effect on parental alcohol consumption.

Prenatal and postnatal parental drinking in Russia in the 1980s was widespread and substantial. The extent of drinking among pregnant women, while hard to quantify precisely, was undoubtedly large during this time period. In the 1980s, ninety percent of women drank regularly, with women of childbearing age rapidly catching up with men in terms of drinking

[11] For instance, Streissguth et al. (1996) summarize several factors, including early diagnosis and special education classes, that can help reduce secondary conditions that result from fetal alcohol syndrome. Among the exceptions, Streissguth (2007) and Nilsson (2008) examine the effect of exposure to alcohol in utero on adult behavioral issues (such as judgement and distractibility), and education and income, respectively.

[12] Physical health problems sometimes associated with FASDs include heart and kidney defects, and vision and hearing problems, but the extent of these and/or other physical health conditions in the long run is much less well known.

[13] As Treml (1987) notes "the campaign offered relatively little in the way of positive policies […] the authorities *promised* to assist in the expansion of athletics and to encourage gardening, home crafts, and hobby activities […] and also ordered a rapid increase in production of soft drinks and juices […] it was expected that widely available soft drinks and ice cream would serve as substitutes for alcohol." (p.53) The emphasis is mine. Furthermore, the athletic facilities and abundant soft drinks also never really materialized in the end (White 1996, Levine 1997)

(Levine and Levine 1986). In a 2007 survey in St. Petersburg, sixty percent of women reported drinking when pregnant, and seven percent of the pregnant women reported having had more than five drinks on at least one occasion (Kristjanson et al. 2007).

Postnatal alcohol consumption was also widespread, most likely draining to a significant extent the parental time and income resources available for childcare. In the 1970s, alcohol was so important in the structure of consumer purchases, that it was dubbed "commodity number one;"[14] it accounted for between fifteen and twenty percent of disposable incomes in Russian households, a very large fraction by international standards (Treml 1982, Tarschys 1993).[15] In addition, drinking was associated with diminished wages due to very high levels of work absenteeism, as well as with income losses due to the drinking parents' increased morbidity and mortality (White 1996, p.50).[16] The impact on parental time was probably large as well. The number of workdays lost due to alcoholism was about 93 days a year on average, and accounted for over 16 percent of all working time in Soviet industries (Segal 1990). Since weekends and holidays were also associated with alcohol excesses, the loss of parental time available to spend with children was probably even larger.[17]

The potential positive influences of the campaign on parental time and income resources, however, were counteracted by negative factors as well—increases in alcohol prices and time queuing for alcohol, and the consumption of more dangerous ethanol-containing substances. To begin with, during the prohibition vodka prices—which were set by the State Committee of Prices—were raised twice, by 25 percent in August 1985, and then again in August 1986 by a

[14] No. 2 was "clothing and underwear", and no. 3 was "meat and sausages" (Krasnikov , "Commodity Number One (Part I), in Roy Medvedev, ed. *Samizdat Register*, vol. 2, Merlin Press, London, 1981, p.101.)

[15] Furthermore, Treml (1982) estimates that approximately 10 percent of households spent over 40 percent of their budgets on alcohol (p.79)

[16] Segal (1990) estimates that 30 percent of the Soviet labor force suffered from alcoholism. Similarly, a survey from a chemical plant from the early 1980s revealed that almost 25 percent of the workforce consisted of alcohol abusers (White 1996, p.50).

[17] Factory output was lower by a third on Mondays, and some factory sections weren't operational at all. During the workweek, agricultural workers in some regions in the countryside were sober only during the first half of the day (White 1996, p.49).

further 20 percent. The effect of the first price increase on household budgets was compensated by decreases in the prices of foodstuffs and household items, but the second price hike was uncompensated (White 1996). As a result, by late 1986, the cost of a half-liter of vodka was roughly equivalent to an entire workday's pay for the average Soviet worker.[18] Since even prior to the prohibition alcohol expenditures contributed to household poverty (Treml 1982), the steep price increases could have further negatively affected household income.

In addition to price increases, prohibition measures included shorter hours at the official supply stores, and the banning of alcohol sales in most places that would provide 'unnecessary temptation'—the vicinity of schools, universities, factories, medical establishments, railroads, bus stations, and essentially most public spaces. This led to very high time costs for purchasing alcohol, under the form of traveling costs, long queues (and sometimes even a bit of struggle) at the few remaining alcohol supply stores; on average, people were waiting in line two to five hours a day to purchase alcohol.[19]

Finally, the prohibition could have negatively affected child health through its effect on parents' morbidity and mortality. Since the emphasis of the campaign was on punitive and restrictive measures, during the campaign people often delayed getting medical treatment.[20] More importantly, the high time and monetary costs of alcohol led to an increased consumption of surrogate alcohols, under the form of moonshine (*samogon*), but also of ethanol containing substances not intended for drinking, such as cologne, glass cleaners, and certain forms of glue (Treml 1997).[21] Since these surrogate alcohols contained very high concentrations of ethanol (98

[18] *Pravda*, 15 November 1987.
[19] *Vestnik statiski*, no.6, p.55, 1989. A joke from the time period, for instance, has the bus driver announcing the liquor stop, and, three bus stops later, the end of the queue to the liquor store.
[20] For instance people who sustained injuries at work while intoxicated often delayed getting medical treatment until they were sober, so as to avoid the drinking penalties associated with the campaign (Treml 1987).
[21] According to a report to the Central Committee of the Communist Party (cited in Treml 1997), sales of certain types of alcohol-based glue increased from 760 tons in 1985 to 1,000 tons in 1987, and sales of glass cleaners increased from 6,500 to 7,400 tons in the same period.

percent as compared to 50 percent in vodka) and/or trace contaminants that were potentially toxic, the consequences for drinkers' health were potentially disastrous.[22]

3. Data

3.1. Long Run Measures Of Child Health

In this paper, I evaluate the effect of the anti-alcohol campaign on child health by using a new dataset that combines child and parent data from the Russian Longitudinal Monitoring Survey (RLMS) with both official and unofficial data on regional alcohol consumption.

Direct information on child outcomes between 1985-1988 is not available, but since health during early childhood has significant effects on later life health (even as late as adulthood), I am able to measure the effect of the anti-alcohol campaign by focusing on child outcomes seven years after the end of prohibition.[23] For these purposes, I use the RLMS survey, which contains very detailed income, family background, and health and anthropometrical information for a nationally representative sample of households interviewed in December 1994.[24] Table 1 and 2 present summary statistics on the household controls and health outcomes used in this paper.

I also use long run measures of health that are determined at specific ages during childhood, namely height and immunizations. Height has been shown in numerous studies to be a good proxy for early life health and development (Falkner and Tanner 1986, Floud et al. 1990), and the period in a child's life between ages zero and two is considered critical to determining later life height, especially prior to adolescence (Beard and Blaser 2002).[25] The reason for this is

[22] See McKee et al. (2005) for a recent analysis of surrogate alcohol consumption in a region in Russia.
[23] See for instance Currie 2007, Haas 2007, Kuh and Wadsworth 1993 for literature reviews on the "long reach of childhood."
[24] This version of the paper uses data from round5 of the RLMS survey. Restricting the sample to children born between 1982 and 1992 (due to constraints regarding alcohol availability data) and to individuals that can be matched with their mothers, results in a sample of 1249 children. Although the RLMS survey has been conducted since 1992, data from rounds 1-4 (1992-1994) is not representative at the national level (see Zahoori et al. 1999 for details).
[25] Height deficits accumulated during childhood cannot be erased by the growth spurt during adolescence, but they can be lessened somewhat (Martorell et al. 1994, see also the review in Case and Paxson 2006)

that the speed of growth and nutritional needs are greatest during this period, and so is the risk for poor parental care-giving and (growth-retarding) respiratory and gastrointestinal infections (Martorell et al. 1994).[26] In order to compare height consistently across birth cohorts, I control for age using flexible (both parametric and non-parametric) functional forms, and I construct standardized height for age (HFA) z-scores.[27]

Since chronic conditions later in life are associated with poor childhood conditions (Barker 1995, Fogel and Costa 1997, Manton et al. 1997, Ravelli et al. 1998), I also examine the impact of prohibition on the probability of the child reporting to be in good health, having had any chronic health problems, and having been hospitalized. To control for the possibility that these health outcomes were determined by more recent income and nutrition shocks during the children' lives (rather than prohibition), I also perform placebo tests using as outcomes indicators for acute (rather than chronic) health conditions—coughing, sore throat, and diarrhea.

Finally, I also examine the impact of the campaign on immunizations, since they have specific age schedules for being administered. While in developed countries "catch-up" immunizations can be administered at older ages for children or adults who were not vaccinated at the recommended times, this was not the case in Russia prior to 1989, due to administrative and bureaucratic constraints.

3.2. Alcohol Data

The summary statistics for the alcohol measures are provided in Table 1 and Table 3. I match the data on child outcomes and parental and household characteristics with alcohol data in the child's region of birth for the time period 1970 to 1992. I use both official alcohol consumption

[26] Older, more autonomous children seem to be better equipped to protect themselves against the effects of poor parenting (Martorell et al. 1994).

[27] Height for age z-scores are standard deviations from the NCHS reference median, as suggested by the World Health Organization. A height-for-age z-score of -2.0, for instance, implies that the child is two standard deviations below the median of the reference population.

data (for 1985-1992), as well as official alcohol production data (for 1970-1994, and available separately for four alcohol categories: wine, beer, vodka, cognac). As mentioned in section 2, the official data does not include homemade alcohol (*samogon*), nor industrial alcohol and ethanol-containing substances not intended for drinking. This is an important omission in the Russian case, because *samogon* consumption in the 1970s and 1980s constituted between 30 and 60 percent of total alcohol consumption (Treml 1997).

Estimates of *samogon* consumption at a regional level are not readily available, however. After the fall of Soviet Union, Goskomstat has made available their estimates of *samogon* production between 1971 and 1989 (see Table 3). These estimates are restricted to sugar-based samogon, and exclude samogon produced from other inputs, as well as home-made fortified wines and beers. Treml (1994, 1997) also provides his own estimates of *samogon*, which are based on data from various Russian and Soviet sources and inferences from excessive purchases of sugar in retail trade, inflated to account for s*amogon* made from inputs other than sugar. Since the consumption of homemade wine was much smaller in Russia than in the wine-producing former Soviet republics (such as Georgia and Moldova), and since sugar-based *samogon* is the most common form of moonshine, the Goskomstat estimates and Treml's estimates do not differ widely prior to 1988 (see Table 3). The Goskomstat method of estimation, however, broke down beginning in the 1988, and was consequently abandoned after 1989 (Nemtsov 1992, Treml 1997, McKee 1999). Importantly therefore, Treml's estimates reveal that the increase in *samogon* consumption persisted even after the end of the campaign in 1988, and that it continued to stay at levels higher than the pre-1985 ones throughout the early 1990s.

Nemtsov (1992, 1998, 2000), Nemtsov and Nechayev (1992) and Nemtsov and Shkolnikov (1997) take a different approach, and estimate total alcohol consumption in Russia on the basis of the proportion of violent deaths (to non-violent deaths) involving the presence of

alcohol in the blood. The estimates in these studies are based on mortality data between 1982-1994 in twenty-five regions in Russia that account for over 40 percent of the total population, and that vary substantially in geography, economic development, public resources, health indicators and drinking patterns. Most importantly, the mortality data used in these studies was unlikely to be manipulated for political purposes during the campaign, since it had been deposited in the archives of the Bureau of Forensic Medicine, out of the reach of the public (Nemtsov 1988, 2000).

In this paper, I use both official alcohol production data, as well as total alcohol consumption estimates based on these latter set of studies.[28] As can be seen from both Table 3 and Figure 3, for the period 1983-1987 these estimates are very similar to both Treml's estimates as well as the sum of the Goskomstat estimates of *samogon* consumption and the recorded sales. As Nemtsov (2000) summarizes: "our latest estimates and those made earlier by other researchers […] were much the same, especially those for 1980–1987. There are no serious arguments in favour of any one of the three estimates […] That the estimates obtained by unrelated methods were so close, may be taken as their indirect verification." (p.140)

4. Econometric Specification

4.1. OLS

The ordinary least squares (OLS) specification uses a fixed effects framework, exploiting the variation across cohorts and regions in the exposure to the campaign; as discussed in sections 2.2 and 3.1, children who were in utero during the prohibition, and those who spent a larger fraction of their first two years of life under the restrictive alcohol regime had the potential to experience larger changes in outcomes compared to other cohorts. Since birth cohort variation might simply reflect the effect of macro economic shocks, however, I also exploit the variation

[28] I am extremely grateful to A. Nemtsov for graciously providing me with this alcohol data.

in the intensity of prohibition across regions. Although prohibition measures were set at the federal (Soviet Union) level, their implementation at the local (republic and *oblast*) level was uneven, resulting in large variations in the degree to which alcohol consumption changed.[29]

Most importantly for our purposes, the implementation of the prohibition across regions in Russia was driven by factors—administrative, monitoring, and political—of a nature unrelated to child outcomes. To begin with, federal directives and alcohol laws were often vague, and as a result, there was a large variation in their administration at the local level due to often-contradictory interpretations.[30]

In addition, there were also wide variations in the severity with which the sanctions were applied.[31] The reason for that was that not just jurists and the police administered the laws, but also party officials, factory managers, trade union leaders, and restaurant directors (White 1996). Pretty much anybody who was a "boss" of some kind was held responsible for the success of the campaign: trade union leaders and enterprise managers had to report on and sanction inebriated workers, restaurant and bar directors had to sanction people who held non-dry weddings, cooperative leaders had to ensure that farmers switched way from grape production etc. (Levine 1997). Most of these 'bosses,' however, had no legal training, and were often very interested in taking into account the moral aspects of the various situations (White 1996). In addition, local party supervision of alcohol policy often depended on the officials' personal ambitions, and the degree to which their career goals aligned with the various factions in the central party (White 1996, Richardson 1999, Levine 1997). As a result, the intensity of the campaign across regions

[29] For instance, during the first six months of the campaign, alcohol consumption decreased by 3.6 liters per capita in the Central Black-Earth region, and by only 0.1 liters in the North-Western region. Between 1984 and1986, alcohol consumption had fallen by 5.7 liters per capita in the Volga region, but only by 1.9 liters in the Far Eastern Siberia region.

[30] The law, for instance, forbade the drinking of 'spirits' in public places, but it did not define spirits. As a result, in some places drinking beer in public areas was allowed because beer was regarded as a "weak" alcoholic drink, whereas in other places even the sale of kefir (a yogurt drink that naturally contains a very small amount of alcohol due to fermentation) was prohibited.

[31] An inebriated worker for instance, might be dismissed in one enterprise, while another one under similar circumstances would simply be reprimanded in another enterprise.

was highly variable; as the justice minister during this time period noted, "every place, town, and even enterprise implement[ed] the legislation in its own random way."[32]

In the OLS strategy I therefore exploit this variation in the intensity of prohibition across cohorts and regions to estimate the impact of restrictions on parental alcohol consumption on long run child health. Specifically, I estimate regressions of the following form:

$$Health_{isc} = \alpha + \beta * alcohol\_w_{st} + X_{isc}\theta + X_{sc}\emptyset + S_s + C_c + R_r * T_c + \varepsilon_{isc} \quad (1)$$

where *i, c, s* and *r* index children, birth cohorts, oblasts, and regions respectively.[33] *Health* is a measure of the child's health status (such as height for age z-score, chronic health, and immunization status). *Alcohol_w* is a measure of alcohol consumption (either official or total), weighed to reflect (monthly) exposure to the effects of the campaign during periods critical to long run child health—fetal period up to two years of age. For instance, to proxy for in utero exposure, a child born on June 31st in 1988 in Moscow is assigned a weighted average of alcohol in the oblast in 1987 and 1988, with both weights being 0.5 in this case.[34] Finally, X_{isc} and X_{sc} are vectors of child and household covariates, and time-varying oblast characteristics, respectively. In addition, I also control for oblast and cohort fixed effects, as well as region specific trends. The coefficient of interest is β, which captures the differential effect of the anti-alcohol campaign on the health of "treated" (at critical stages of development during prohibition) children relative to children in the same census region. Observations are weighed using the survey sample weights, and the standard errors are clustered at the oblast level

Prior to 1990, internal migration in Russia was closely monitored and severely restricted, so it is unlikely that the households moved during the campaign in response to changes in

[32] Cited in White 1996,154.
[33] The dataset covers 11 birth cohorts (1982-1992), 32 oblasts and 8 census regions: Metropolitan areas (Moscow and St. Petersburg), Northern and North Western, Central and Central Black Earth, Volga-Vaitski and Volga Basin, North Caucasian, Ural, Western Siberian, Eastern Siberian and Far Eastern.
[34] Almond et al. (2007) employ a similar procedure to proxy for in utero exposure to the effects of the Great Famine in China.

alcohol policies.[35] After the breakup of the Soviet Union, however, mobility did increase, so comparing child outcomes during and after the campaign could result in biased estimation. To address this issue, I restrict the sample to non-movers (households that report current residence being the same as the child's birth region), but the results are not very sensitive to restricting the estimation in this way. [36]

4.2. Instrumental Variables

There are two main concerns with the OLS approach. First, as discussed in section 3, our alcohol data might measure the intensity of prohibition with error, biasing the OLS estimates towards zero. Secondly, although the regional and household covariates and fixed effects control for many determinants of child health and alcohol consumption changes due to the prohibition, omitted variable bias could still be a concern. For instance, if sites with more efficient party bureaucracies had lower alcohol consumption during prohibition and worse child outcomes after the fall of the Soviet Union, then the effect of the campaign on child health is not causal, and the OLS estimate would most likely be biased towards zero as well.

To address these issues, I also use an instrumental variable procedure that isolates a source of variation in alcohol consumption—regional sugar consumption and production—that is exogenous to long run child health. As discussed in section 3, *samogon*, whose main ingredient is usually sugar, has always been an essential component of unregistered alcohol consumption, accounting for between 30 and 60 percent of overall alcohol consumption. *Samogon* production was relatively simple and required few skills and equipment, and it expanded rapidly, especially during the later years of the prohibition period (White 1996, Treml 1987, 1997).

Our IV approach exploits this association between sugar and samogon, as well as the

[35] Siberia did experience in-migration during this period, due to both labor needs in the region, as well as political reasons. I therefore experimented with excluding Siberia from the estimation, but the results are essentially unchanged.

[36] This is not surprising given that internal migration started increasing significantly only after 1994, a time period which is outside my sample. Even during the early period of transition, entry to cities and certain regions was still restricted; these restrictions were only eliminated during the second half of the 1990s (see Gang and Stuart 2002 for more details).

variation in the exposure to the campaign across cohorts. Specifically, I use as instruments for alcohol consumption the interactions between the change in sugar availability between 1985 and 1989 in a child's oblast of birth, and dummies for the year of birth.[37]

The correlation between sugar and alcohol consumption during prohibition is very strong. Sugar consumption increased in tandem with *samogon* production, especially during the later years of the campaign—between 1987 and 1988, for instance, sugar sales increased by as much as they had done during the entire decade from 1970 to 1980 (*Pravda*, September 1988). The key for the IV estimation, however, is to use a source of variation in regional sugar availability that is exogenous to child health. To do so, I take advantage of the fact that regional sugar availability in the Soviet Union was determined via a complex system of central planning involving quotas. The sugar quotas (much like the quotas for other foodstuffs), were typically set to last five years, and were in principle based on observable regional characteristics like population, income, and degree of urbanization. In practice, however, due to both planning errors and (very local) bureaucratic discretion, the initial allocation of quotas across broad regions had a large random component (Alexeev and Treml 1993; Schroeder 1992).

For our purposes, the existence of the quota system—dating long before the prohibition period—means that children born during prohibition in regions that had been allocated greater sugar quotas prior to 1985 would have experienced a greater exposure to (sugar-based homemade) alcohol compared to children from other regions. Since official sugar quotas measures for each oblast are unavailable, the instruments that we use are oblast and year specific sugar predictions, $sugcons\_pred_{sc}$, constructed using 1970-1984 data on sugar consumption and regional characteristics used in official planning reports.[38]

[37] Note here that only the interactions can be considered exogenous.

[38] The regional characteristics used in official planning reports are income, population, roads, degree of urbanization, and share of population of working age. I therefore obtain 1985-1992 sugar trend predictions on the basis of linear regressions (for 1970-

It is important to note here that the exclusion restriction will still fail if sugar quotas were jointly determined (by the central planners) with those for other foodstuffs that constitute essential inputs into child health. I have checked this possibility directly, and found no evidence of a correlation between sugar consumption and that of meat, bread, potatoes, milk, and fruits and vegetables during this period.[39] This is not surprising given the frequent (and rather random) planning failures, inefficiencies, and reporting problems in the Soviet system (Alexeev and Treml 1993, Schroeder 1992).

To further check the validity of the results, I also perform estimations using a different set of instruments, namely the interactions between measures of sugar production (rather than sugar consumption quotas) in a child's region of birth and dummies for the year of birth. Since sugar production in Soviet Russia was heavily dependent on natural conditions for the growing of sugar beets (such as precipitation, temperature, and soil quality), these instruments are more plausibly exogenous to child health.

Regional sugar beet production did not automatically translate into regional sugar availability, however, since sugar beet processing often occurred far away from the original growing area.[40] Beginning in the 1980s, however, when republics and local units (oblasts) acquired greater autonomy due to glasnost, they began imposing restrictions and embargoes on the shipment of goods outside their administrative boundaries (Schroeder 1992).[41] Since these restrictions ensured a much tighter relationship between the local production and consumption of various goods during the time period under study—including sugar—, our instruments based on

1984) of sugar consumption per capita in each oblast on year, real income per capita, log of population and its square, log of road length per capita, and on the percentage of the population that is urban, and of working age respectively. Results using as instruments sugar predictions constructed on the basis of 1980-1984 data are essentially the same as those presented in the paper. Appendix Table 1 contains the results using the 1970-1984 data.

[39] Results available from the author upon request. Note here that if the correlation between the consumption of other foodstuffs and sugar was positive, then the negative effect of alcohol consumption on child health would be mitigated by the nutritional impact of other foodstuffs, and our estimates would be *underestimated*.

[40] This was the result of central planning sometimes, and other times simply the end result of various inefficiencies in the agricultural system (Hultquist 1965).

[41] These restrictions were officially ended in the early 1990s, after the dismantling of the former Soviet Union.

sugar production are not only exogenous, but also strong.[42]

5. Results

5.1. The effect of the anti-alcohol campaign on child height

As discussed in the previous sections, the anti-alcohol campaign resulted in large changes in alcohol consumption across regions and birth cohorts. Did these changes result in improved health outcomes for the children who were most vulnerable to the effects of prohibition? The comparison of height for age (HFA) z-scores for different birth cohorts in high (above the median) and low alcohol consumption regions provides some suggestive evidence. Figure 4 reveals that both prior to and after the anti-alcohol campaign, the HFA z-scores for boys born in high and low regions generally followed similar patterns, and that boys born in low regions were on average taller than boys from high regions during both these periods—which is consistent with parental alcohol abuse being detrimental to child health (panel A). During the anti-alcohol campaign, however, these patterns were reversed; since the intensity of the prohibition was generally higher (and thus the alcohol consumption decline larger) in the high regions compared to lower ones, height deficits among prohibition cohorts were much smaller in high regions. Furthermore, panel B of Figure 4 suggests that HFA z-scores for girls did not follow the same patterns as the HFA z-scores for boys.

Table 4 shows this more formally. Columns 1-3 and 4-6 present the OLS results from estimating equation 1, for boys and girls, respectively with height for age (HFA) z-scores as the dependent variable. Since as discussed in section 2.2 and 3.1, the children most likely to be affected by the changes in alcohol consumption were those at critical stages in their development

[42] To check for the possibility that local sugar production after 1985 might have been responsive to increased demand during prohibition—in which case the exclusion restriction would still fail—I also performed specifications using as instruments the interactions between cohort dummies and sugar production during "normal times", just before the prohibition, but results are essentially the same. This is not surprising given the heavy dependence of sugar beet production on natural conditions, and the notoriously sluggish (and sometimes complete lack of) responsiveness to incentives of the Soviet agricultural system.

during the campaign, the variable of interest is alcohol consumption during the fetal period and first two years of life.[43]

The vector of controls includes household correlates of child height and parental alcohol consumption suggested by the literature (maternal age[44] and educational attainment,[45] family size, urban setting), as well as region-level characteristics (regional income, population, and availability of health resources). Of these variables, the only significant correlates of child height are the availability of health resources (dispensary capacity and doctors per capita) and being located in an urban setting (column 1). In columns 2-3 and 5-6, I also control for current and longer run measures of the economic status of the household—current total household income (adjusted for differences in the cost of living across regions), and a living conditions index, constructed using principle components methods[46] — but the results are essentially unchanged.

Overall, the OLS regressions from columns 1-6 suggest that the lower alcohol consumption during the campaign was associated with improvements in child height for boys, but not for girls. The estimate of β in columns 1-3 is negative and statistically significant. It implies that a decrease of alcohol consumption of 1 liter per capita (one regional standard deviation across regions) increased male height by 0.25 standard deviations relative to the WHO reference median. By contrast, the differences-in-differences estimate of β in columns 4-6 is statistically insignificant, suggesting that the decline in alcohol consumption during the prohibition had no effect on girl height.

[43] For instance, a child born on January 31st in 1988 in Moscow is assigned a weighted average of alcohol consumption in region 1 in 1987, 1988, 1989, and 1990, with the weights being 8/33 (in utero), 12/33 (1 month in utero +11 months during early childhood), 12/33 (months during early childhood) and 1/33 (remaining month during early childhood) respectively.

[44] Specifically, I include indicators for whether the mother was less than 18 or over 35 years of age at the time of the child's birth, since this has been shown to be correlated with various health-related problems (see Royer 2004).

[45] The regressions in Table 4 use mother's educational attainment as controls. Results controlling for father's educational attainment are very similar to these, but some collinearity problems occur due to the fact that the educational variables are very highly correlated with the maternal ones—most likely due to assortative matching in Russian marriages.

[46] The variables that I use in the principal components analysis are indicators for the availability of central heat from boiler, of central cold and hot water supply, of metered gas/electric stove, and of central sewage disposal. Filmer and Pritchett (2001) use a similar method using NHFS data, and argue that the asset index might be a better proxy of household permanent wealth compared to current income and consumption measures.

As discussed in section 4, both measurement error and the endogeneity of the intensity of the campaign could bias the estimate of β towards zero. To address this issue, I next estimate equation (1) by instrumental variables, using as instruments for alcohol consumption the interactions between the change in sugar consumption quotas in a child's oblast of birth and dummies for the year of birth. Columns 7-10 of Table 4 present the IV results with and without the household income measures for boys and girls, respectively.

The estimate for β is very similar in both specifications for each gender; statistically significant and negative (and much larger in magnitude than the OLS estimate) for boys, and statistically insignificant for girls. The estimate of β from columns 7-8 implies that a decrease of alcohol consumption of one liter per capita would have improved male height outcomes by half a standard deviation.[47] To get a better sense of the magnitude of these estimates, I re-estimate equation (1) with log child height (rather than HFA score) as the dependent variable. The results, presented in Table 5 (columns 3-4) are qualitatively similar to those in table 4.[48] The estimate for β in this specification (-0.03) implies that, at the mean, a decrease in alcohol consumption of one liter per capita—equivalent to moving a child from the Urals region to Moscow for instance—would increase child male height by 3.3 cm.[49] These estimates are of a similar order of magnitude to those in the literature of the impact of early life conditions on later life outcomes.[50]

[47] One liter of alcohol per capita (or roughly 2 liters of 100-proof vodka per person per year), constitutes roughly one standard deviation of regional alcohol consumption in our sample. The coefficient on β is -0.8, which implies an increase in HFA score of 0.8 (or roughly half of a standard deviation in HFA scores in our sample).

[48] To facilitate comparisons, columns 1 and 2 of Table 5 simply present the results from columns 7 and 9 of table 4.

[49] The effect on height is a proportional decline of $(1-e^{-0.036*1})=3\%$, which represents 3.3 cm at the mean in our sample.

[50] For instance, Duflo et al. (2007) found that the effect of the decline in regional GDP (following the destruction of grape vines by phyloxera) on height at age 20 was equivalent to half a century (19^{th}) worth of growth. Chen and Zhou (2007) and Meng and Qian (2007) found that the Great Famine in China decreased (adult) height by 3.04 and 3.38cm, respectively. Brainerd(2006) found that the average adult stature gain between 1937 and 1982 was between 1.5- 1.9 cm each decade for men, and 1.2cm each decade for women. Note here that our IV estimate is a local average treatment effect (Angrist et al.1996). Under the assumption that the effect of parental alcohol consumption on child health is heterogeneous due to unobservable characteristics, the IV estimates provide the effect for the groups affected by the anti-alcohol campaign. Since, as discussed later in the paper, the effect of the anti-alcohol campaign seems to have been strongest for the most vulnerable groups, our larger estimates compared to Brainerd (2006) simply reflect the larger potential for health improvements among more vulnerable children.

By contrast, however, the results in table 4 and 5 suggest that the prohibition had no effect on female height. These results are consistent with studies from the medical and biological literature that suggest a greater susceptibility of boys to early life conditions (Trivers-Willard 1976, Wells 2000, Edlund and Almond 2007, Drevenstedt et al. 2008). Since male infants typically have higher rates of morbidity and mortality than female infants, they may suffer to a greater extent the physical consequences of parental alcohol abuse.[51]

To get a better sense of the impact of the prohibition by timing of exposure, I also estimate the effect of the campaign separately during the fetal and postnatal periods. Columns 5-8 of Table 5 show the results of estimating equation 1 using as campaign proxies the alcohol consumption in a child's birth place, weighed separately by exposure in utero, and between ages 0 and 2 respectively. The estimates of β from both specifications for boys are negative, statistically significant, and large in magnitude, confirming the crucial impact of health investments during both the fetal and the early childhood periods.

5.2. Alcohol consumption and child height: validity and robustness checks

In Table 6 I analyze the validity of the IV results for boys in greater detail.[52] Column 1 simply reproduces the main IV results from column 7 of Table 4 for convenience, and column 2 presents the first stage results. The instruments are strong; the first stage F test statistic is 49, which is much higher than the critical values (20 and 40, respectively) required for TSLS unbiased and correct size estimation (Stock and Yogo 2001). Since sugar availability changes act as proxies for *samogon* consumption and thus for the intensity of the campaign, the key prediction is that the first stage coefficients should be negative for children born during the prohibition years, and Figure 5 shows that this is indeed the case. Furthermore, as discussed in section 4, the regional variation in sugar between 1985 and 1989 across cohorts—stemming from

[51] We will analyze this issue in the Russian context in greater detail in section 6.
[52] The results for girls (available from the author upon request) are qualitatively similar to those in tables 4 and 5.

quotas prior to the prohibition—is unlikely to be directly related (other than through its effect on alcohol consumption) to child outcomes in 1994.

To further confirm the validity of the IV results, I also estimate equation (1) using as instruments for alcohol consumption the interactions between sugar production and birth cohort dummies. As noted in section 4, sugar production is arguably more exogenous to long run child health, being largely determined by natural conditions. Furthermore, due to increased local autonomy, sugar consumption and production were tightly correlated during the 1980s.

Since data on sugar production is only available for regions where natural conditions are suitable for sugar beet production, sample sizes are slightly smaller. Furthermore, sugar production data is only available at the regional level, so estimations using sugar production instruments contain region (rather than oblast) fixed effects—but are otherwise similar to those using sugar quotas.[53] Column 3 of Table 6 shows the IV results, and column 4 shows the first stage. The instruments are strong, and the patterns of the coefficients in the first stage are similar to those for sugar quotas.[54] Furthermore, using this different set of instruments, the estimate of β is still statistically significant, but slightly smaller (-0.5). When sample differences are taken into account, however, the implied magnitude of the effect of prohibition on male HFA z-scores is essentially the same regardless of whether we use sugar quotas or production as instruments.[55]

Columns 5-7 present some further robustness checks by using as our alcohol measures official alcohol production of vodka, wine and beer (rather than total alcohol consumption). These results are consistent with the fact that our instruments identify the variation in alcohol consumption during the campaign stemming from changes in *samogon* use. The coefficient on

[53] Standard errors are also clustered at the regional level in these estimations. Furthermore, the sample size is also smaller since sugar is not produced in all regions.
[54] The first stage F statistic is 42, and the coefficients on the prohibition cohort dummies are negative and individually and jointly statistically significant.
[55] To make results comparable between columns 1 and 3, I have re-estimated equation 1 using regional (rather than oblast) sugar quotas as instruments on the sample in column 3. The estimated coefficient on β in this specification is –0.42.

beer consumption is statistically insignificantly different from zero, and that on wine is negative, but very small in magnitude (columns 6-7). By contrast, the coefficient on vodka (s*amogon*'s closest substitute) in column 5 is negative, strongly significant, and large in magnitude (-0.7). Furthermore, the implied effect of the prohibition on male HFA z-scores in the estimation using vodka production is very similar in magnitude to the (total alcohol consumption) IV estimate in column 3. The similarity of all the IV estimates across the different specifications in columns 1, 3 and 5 of Table 6 (using different alcohol measures and different sets of instruments), is reassuring, providing further confirmation of the validity of the IV approach.

Since child height is measured seven years after the end of the campaign, however, one might still be concerned that the results are driven by the impact of post-prohibition factors, like persistent parental alcohol consumption over time or the market reforms of the 1980s, rather then by the prohibition per se.[56] I address these issue in two ways. In column 8, I perform a placebo test by using weight for age (WFA) z-scores as the dependent variable. Unlike height for age, WFA z-scores represent a short-run measure of child health, reflecting current flows of health investments, rather than their accumulation over time (Falkner and Tanner 1986), and thus could not have been affected by the anti-alcohol campaign. In column 9, I use 1995 parental drinking status as my measure of alcohol consumption, and I check whether persistent household drinking habits (correlated with the intensity of the campaign) are driving the effect on child height. The placebo estimates of β in columns 8 and 9, are not statistically different from zero, however, which provides further confirmation that our estimates really capture the effect of prohibition (rather than post-1988 changes in household's socio-economic circumstances) on child health.

[56] The evidence in the literature suggests that the early years of the transition to a market economy were generally associated with factors detrimental to health: stress, smoking (especially among women), and increased alcohol consumption in the presence of low real alcohol prices following the hyperinflation of the 1990s (See Stillman 2006 for a review). Our estimates would therefore be *underestimated* if these factors were really driving the results.

5.3. Distributional Effects of the Anti-Alcohol Campaign

The estimates so far will not capture the full distributional impact of the campaign unless its effects are similar at the mean and in the tails of the distribution. Panels A-C of Figure 6, however, which show the HFA densities across high and low regions by birth cohort, suggest that this is unlikely to be the case. This figure reveals that there was a right shift in the entire distribution of HFA z-scores in high intensity regions relative to low intensity ones for children born during the prohibition (relative to children born during other time periods in the sample), and that the effects in the tails of the distribution seem to have been stronger than those at the center.

Table shows this more formally. In these estimations, I aggregate the individual and household level variables to the bottom, middle and top terciles of (mother's) height in each oblast-birth cohort cells, and perform IV estimations on this modified data. Since our instruments and alcohol variables vary only at the oblast-birth cohort level, they are not affected by these changes. Essentially, this method estimates the average effect of the campaign on the tails of the distribution, and, unlike quantile regression, it enables us to use fixed effects.[57] The results, presented in columns 1-3, show that the effect of the campaign was strongest for the most vulnerable groups, namely those with statures in the bottom percentiles of the distribution; in the topmost tercile of the height distribution, the effect of the campaign on child height was statistically insignificant.

5.4. The Effect of Prohibition on Chronic Health and Immunization Status

Although the results so far show that the campaign had a substantial protective impact on the health of boys (as measured by height), it is important to learn whether it also had an impact on other long run health indicators as well. In the first three columns of table 8, I therefore assess

[57] By contrast, quantile regression would estimate the effect of the campaign on the entire distribution of height outcomes.

the impact of the campaign on the likelihood that the child was hospitalized in the past year, of the parent reporting that the child had serious health problems during the past three months, and on parent-reported child health status.[58] The results in panel A indicate that for boys, the alcohol campaign decreased the likelihood of adverse chronic health conditions (under the form of hospitalizations and serious health concerns), and increased the probability of parents reporting the child to be in good health. By contrast, the results in panel B show that for girls, the alcohol campaign increased the likelihood of the parent reporting the child to be in good health status (though by a much smaller magnitude compared to boys), and had no effect on hospitalizations.

To confirm that these effects on child health are due to the campaign, I also perform several falsification tests. Specifically, I use as dependent variables acute health outcomes—indicators for coughing, sore throat, and gastric problems during the past week—which should not have been affected by past events like the alcohol campaign. The results in columns 3-6, show that this was indeed the case (for both girls and boys).

Finally, in the last column of Table 8, I examine the impact of the anti-alcohol campaign on the probability of the child being immunized. As discussed in section 3, since vaccinations have an age-specific schedule, they can provide us with a cleaner identification of the effect of alcohol consumption during the campaign on child health. Specifically, if I find that children from higher intensity regions, who were "eligible" to be vaccinated during the campaign, had different immunization rates (when measured in 1995), we can be confident that this effect was indeed due to the prohibition. The results in column 7 show that this was indeed the case for boys; a decrease in alcohol consumption of one liter per capita increased the probability that a child would have all the age-specific required immunizations (DTP, polio, measles, and

[58] Self-reported health has been shown in numerous studies to be a good predictor of both short-run and long-run health (see Miilunpalo et al. 1997).

tuberculosis) by over 24 percent. By contrast, there is no evidence that the anti-alcohol campaign had a discernible effect on the immunizations for girls (panel B, column 7).

The results in column 7 suggest that immunizations can provide a plausible mechanism for the effect of prohibition on height and chronic conditions for boys that we found in section 5.2 and in this section. The reason for this is that increased immunization rates are associated with declines in childhood infectious diseases, which, in turn, are associated with increases in growth, and decreases in chronic conditions later in life (Martorell et al. 1994, Blackwell et al. 2001, Barker 1995, Costa 2000). [59]

5.5 Why Are The Effects of the Prohibition Stronger For Boys?

Together, the results in the previous sections suggest that the anti-alcohol campaign had a large impact on the health of boys in the regions that experienced large drops in alcohol consumption, possibly due to the effect of campaign on immunizations. The campaign, however, had much smaller effects (if any) on long-term health for girls. Why were Russian boys so much more susceptible to the effects of the anti-alcohol campaign?

As noted in section 5.1, the medical and biological literature suggests one possible reason, namely the fact that male fetal and infant survival rates are more vulnerable to early life conditions than female ones (Wells 2000, Drevenstedt et al. 2008). Since adverse early life conditions can increase the fraction of pregnancies that are female (Anderson and Bergstrom, 1998, Nilsson 2008, Mathews et al. 2008), the prohibition could have positively affected the long-term health for boys through its effect on male survival. To check for this possibility, I estimate equation 1 in an IV framework, using an indicator for live male birth as the dependent variable, and using a polynomial in mother's age at first birth as additional controls (in addition

[59] Frequent and severe infections during early childhood have been shown to impair growth (Martorell et al. 1994), since they can both lead to and exacerbate inadequate dietary intake (Scrimshaw et al. 1968). In addition, childhood infections are also associated with chronic conditions later in life such as heart diseases, cancer and lung conditions (Blackwell et al. 2001, Barker 1995, Costa 2000).

to the household and regional controls in table 4). The estimate for β is statistically significant and negative (-0.12), suggesting that a decrease in alcohol consumption of 1 liter per capita during prohibition increased the likelihood of a live male birth by 12 percent.

To interpret the results, however, it would be useful to know whether the selection on survival was positive or negative. If the male prohibition babies are weaker compared to non-prohibition babies, then our results are underestimated, whereas the opposite is true if male prohibition survivors are stronger. Although data limitations do not allow me to examine this issue more fully in the Russian context, we should note here that most studies in the literature of the effects of early *adverse* life conditions typically find *positive* selection on mortality (Qian 2007, Nilsson 2008, Edlund and Almond 2007). Furthermore, our discussion of the distributional effects of the prohibition on height outcomes is also suggestive of male prohibition survivors being more vulnerable.[60]

Together, these findings suggest that our estimates are probably most relevant for shedding light on the effect of the prohibition on the most vulnerable groups. In addition, even in the absence of survival bias, our results are most likely underestimates of the cumulative effect of the campaign over the life of a child. The reason for this is that poor health in childhood (which we measure during pre-teen years) is associated not just with worse chronic adult health, but also with adverse health trajectories; by middle age, the cumulative impact of childhood shocks on chronic health could be 4-6 times larger than that earlier in life (Haas 2007)

5.6. The Effect of Prohibition on Child Health: Time or Money?

As discussed in section 2, reductions in parental alcohol consumption can affect child outcomes by increasing parental time and monetary investments in child health during both the fetal and early childhood time periods. Parsing out the relative contribution of parental time and

[60] Anecdotal evidence (as well as author's preliminary research) also suggest a change in the *composition* of births towards more vulnerable groups, with mothers from lower socio-economic status being more likely to be pregnant during the campaign.

financial resources to the improvement in child outcomes during the campaign is a very difficult task, and cannot be answered definitively in the absence of detailed survey data from the time period.

Nevertheless, we can provide some suggestive evidence in this respect based on the effect of prohibition on immunizations. In the 1950s and 1960s, the health care system in the Soviet Union had been particularly successful in reducing infant deaths from infectious diseases through a variety of public health and mandatory mass immunization campaigns (Ryan 1988, Brainerd and Cutler 2005). By the late 1970s, however, the worsening of the economic conditions in Russia started putting a strain on the resources available for immunizations (Vitek and Wharton 1998, Spika et al. 2006, CDC 1994).[61] The result was an increase in rationing for some vaccines (like DTP (diphteria-pertussis), polio, and measles) that had been mandatory and widely available in previous decades. Furthermore, other vaccines (for hepatitis, and a newer version of the measles vaccine for instance), which had not been part of the general immunization campaigns in the 1950s-1960s, were available only on an "optional" basis, and usually required an informal (bribe) payment (Vitek and Wharton 1998, Ryan 1998, Spika et al. 2006). Furthermore, due to the centralized nature of the healthcare provision system, these changes in the provision of vaccines were determined by federal (rather than local) factors, and their effect was therefore rather uniform across the Soviet Union.

In table 9, we take advantage of this difference in time and monetary costs among vaccines to shed further light on the channels through which the campaign affected child health. Columns 1-2 show that prohibition had a strong impact on the likelihood of the child receiving all rounds of DTP and polio immunizations, a process that was time-intensive for the parents due to queuing, as well as finding out when and where the vaccines will be available. Column 3

[61] In fact, the outbreak of a diphteria epidemic in Russia during the 1990s, for instance, was largely attributed to decreased immunizations in the 1980s (Vitek and Wharton 2005).

shows that the effect of prohibition on the likelihood of being immunized against measles—which required both queuing (for the older vaccine type) and monetary resources (for the newer type)—was positive, but only weakly statistically significant, and much smaller in absolute value. By contrast, vaccination outcomes that were more intensive in parents' money—those for the newer vaccines, mumps and hepatitis—were unaffected by the campaign, or even decreased (columns 4-5).[62]

Together, the results in table 9 provide suggestive evidence that time factors might have played a larger role in improving child health compared to parental income, at least where immunizations were concerned. Furthermore, these results also suggest an additional reason for the differential effect of the campaign on males discussed in section 5.4. Recent research suggests that although mothers divide their time inputs equally among their children (regardless of child gender), fathers do not; paternal time investments are significantly bigger for boys compared to girls (Lundberg et al. 2008). Since in pre-prohibition Russia the frequency and intensity of drinking was larger among men (Treml 1987), the campaign probably increased relative paternal time available for childcare—and thus relative time investments in boys (relative to girls). In turn, this would provide another mechanisms for the very strong effects of exposure to the prohibition both in utero as well as during the post-natal period. Investigating these issues in greater detail would be an interesting avenue for future research.

6. Conclusions

This paper evaluates the impact of parental alcohol consumption on long run child health by taking advantage of a unique shock to alcohol supply—the 1985-1998 anti-alcohol campaign in

[62] To check for the possibility that government, rather than parental effort, drove the immunization results, I have also performed two further estimations. First, I restricted the sample to children who reported not having been immunized at school, since vaccination outcomes for these children were more likely to be driven by government (rather than parental) action. Second, I also checked to see whether the effect of prohibition on immunization outcomes was higher for families with more children, since these families had an increased chance of governmental health intervention (and immunization) through social worker involvement. The results from these estimations, however, are very similar to those in table 9.

Russia. The campaign's effects on alcohol consumption and health have been the subject of a lot of debate in the literature, and we offer several contributions. First, by using a new data set that combines survey level data with regional alcohol consumption and focusing on a previously unexplored outcome (long run child health), we provide new evidence that the campaign had some large positive effects in the long run. Second, by documenting and combining several separate insights from the literature (the large regional variation in the intensity of the campaign and the correlation between sugar production and consumption and samogon), we are able to use a new (instrumental variables) method to address both the problem of joint determination of the campaign intensity and health outcomes, as well as that of data limitations regarding unregistered alcohol consumption.

We find significant improvements in height, immunization rates, and chronic conditions among boys born during prohibition who also lived in regions with effective anti-alcohol campaigns. However, we find no evidence that the prohibition had a significant long-term impact on the health status of girls. These results are consistent with a growing body of evidence in the medical literature that suggests a stronger sensitivity of male health outcomes to early life conditions. Together, the results in this paper show that parental investments (especially time) – during the fetal period, as well as during the child's first two years of life—can have significant consequences on long-term child health outcomes. This has important policy implications, demonstrating a potential positive effect of suppressing parental access to alcohol.

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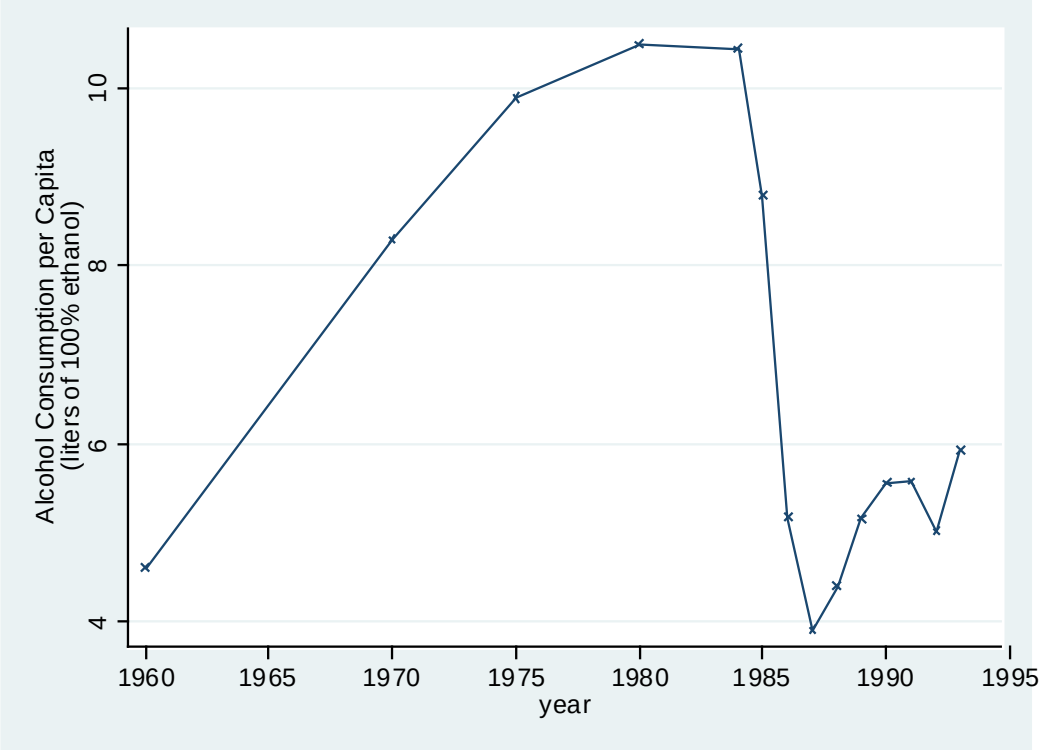
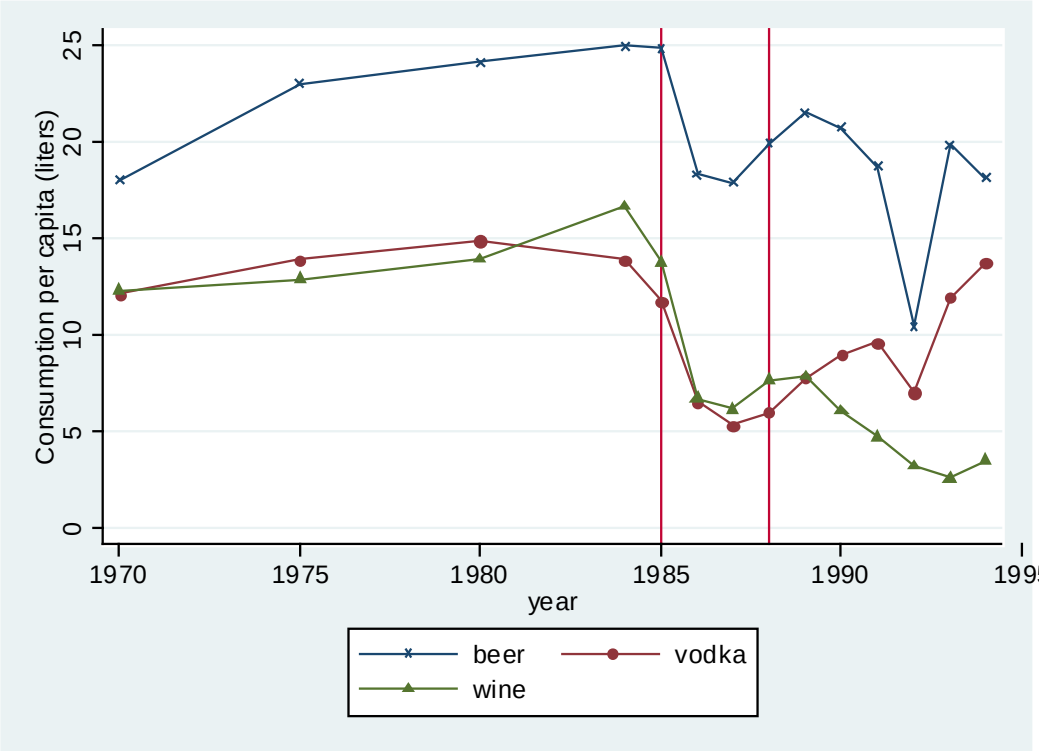
Figure 1. Official Alcohol Consumption Per Capita In Russia, 1960-1995



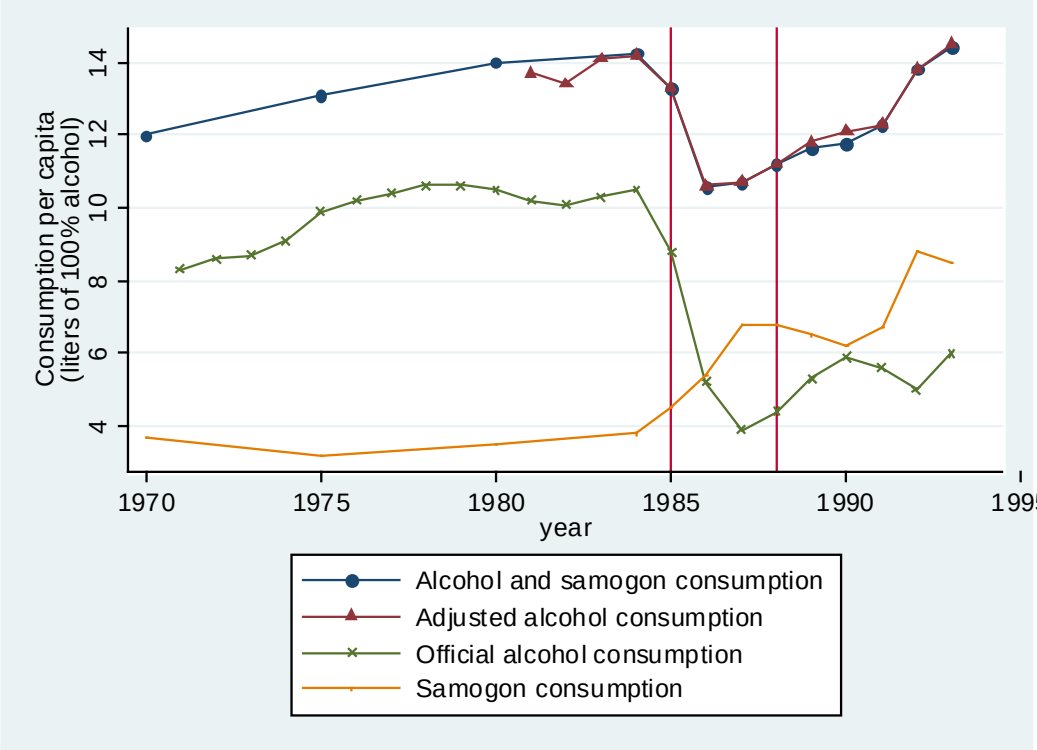
Note: Alcohol consumption data was published by Goskomstat Rossii (1992, 1993, 1995) and Treml (1982), and reproduced in Treml (1997). Alcohol data is measured in liters, and was derived from sales of all state-produced alcoholic beverages, i.e., vodka, fruit wine, grape wine, cognac, champagne, and beer—converted to 100% alcohol.

Figure 2 Per Capita Consumption of State-Produced Alcoholic Beverages in Russia, 1970-1995



Note: The data was published by Goskomstat Rossii (1992, 1993, 1995) and Treml(1982), and reproduced in Treml (1997). Home-distilled samogon and home-made wine are excluded. Per capita consumption of fruit wine, cognac, and champagne is not shown separately.

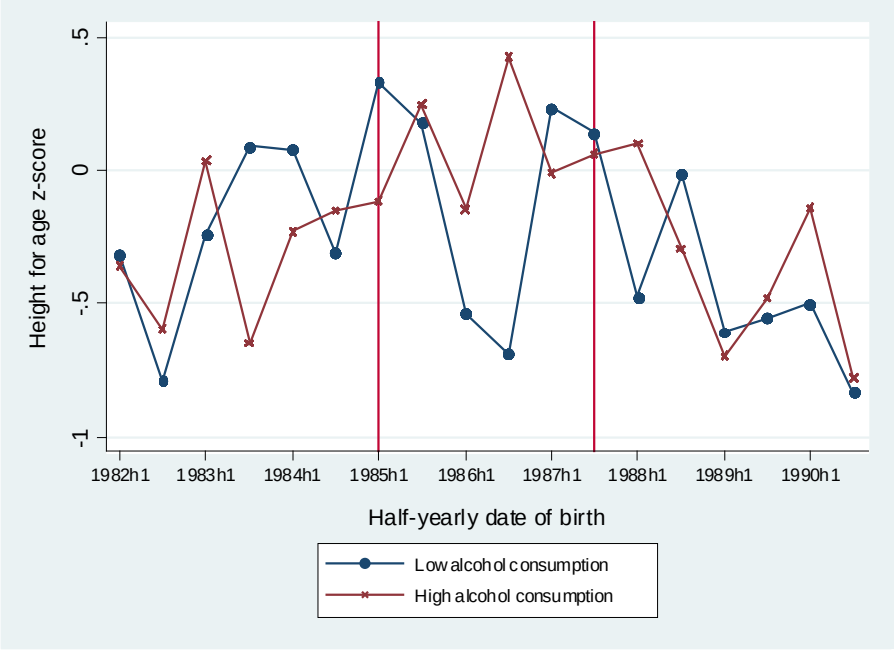
Figure 3 Official and Actual Per Capita Alcohol Consumption in Russia, 1960-1995



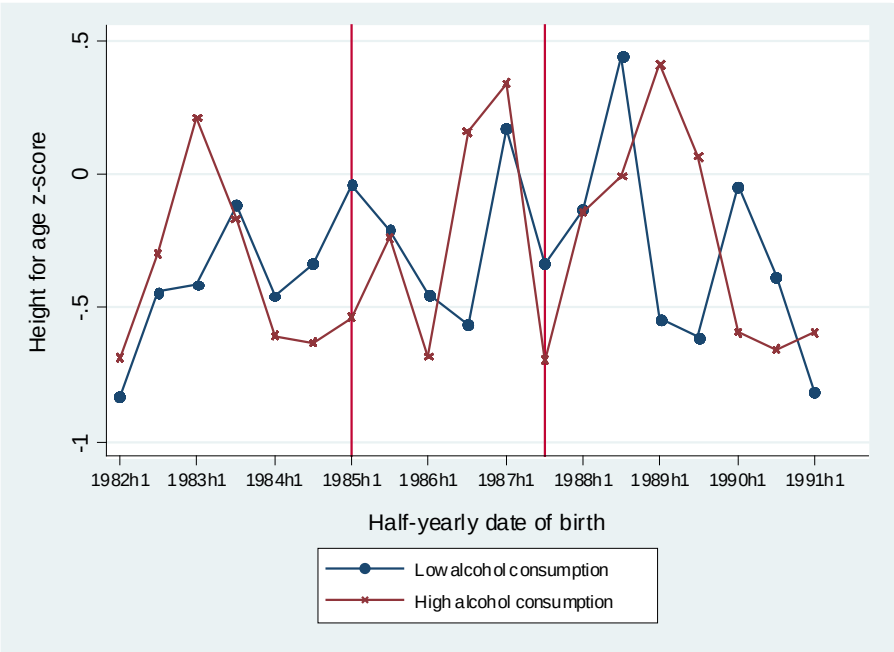
Note: Official alcohol consumption data was published by Goskomstat Rossii (1992, 1993, 1995) and Treml(1982), and reproduced in Treml (1997). It was derived from sales of all state-produced alcoholic beverages, i.e., vodka, fruit wine, grape wine, cognac, champagne, and beer, converted to 100% alcohol. Alcohol and samogon consumption is the sum of official alcohol consumption and samogon consumption. The samogon data is from Goskomstat estimates and was estimated by Treml (1997). Samogon consumption refers to sugar-based samogon only. The estimates exclude samogon produced from other inputs, such as potatoes, grain, and fruits, as well as home-made wines and beers. Adjusted alcohol consumption is total alcohol consumption (samogon included) as estimated by Nemtsov(1992, 1997), and Nemtsov and Nechaev (1991).

Figure 4 Height For Age Z-Scores, in Regions with High and Low Alcohol Consumption Levels

Panel A: Height For Age Z-Scores : Boys

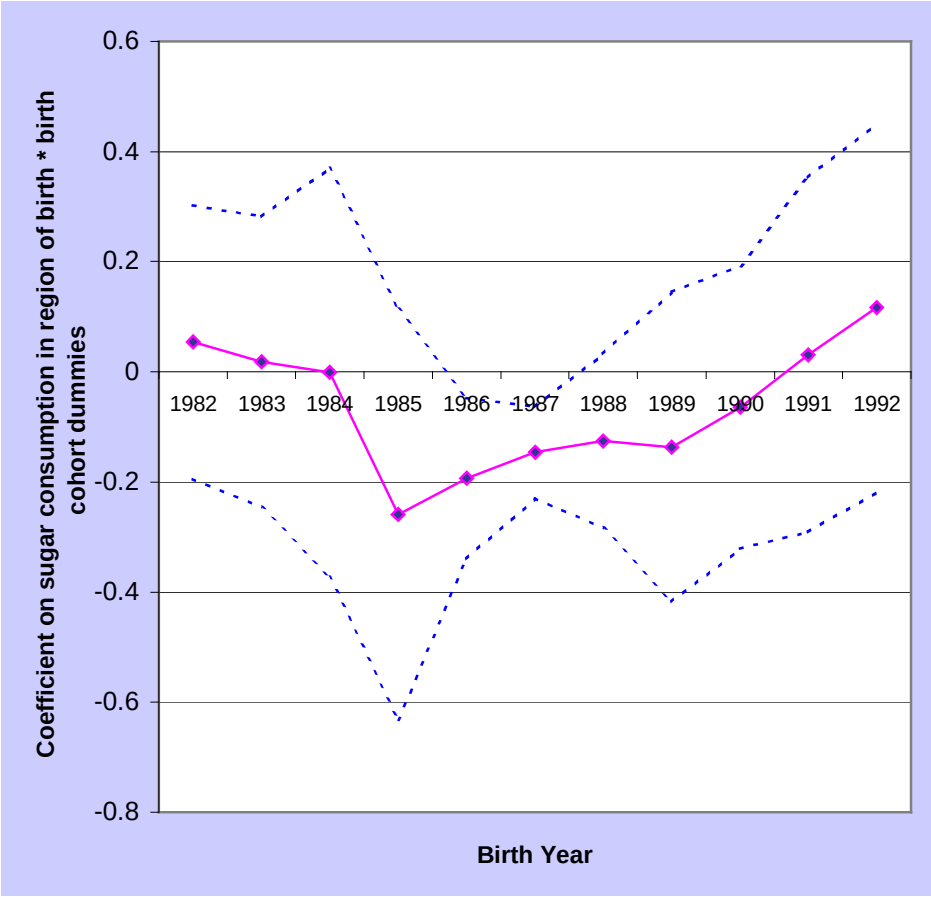


Panel B: Height For Age Z-Scores : Girls



Note: The figures shows average height for age (HFA) z-scores for children born in regions with high (above the median) and low (below the median) total alcohol consumption. Data on height and age is from round 5 of the RLMS. The HFA z-score represents the number of standard deviations from the WHO reference median height for a given age. The vertical lines represent the dates for the start and end of the anti-alcohol campaign.

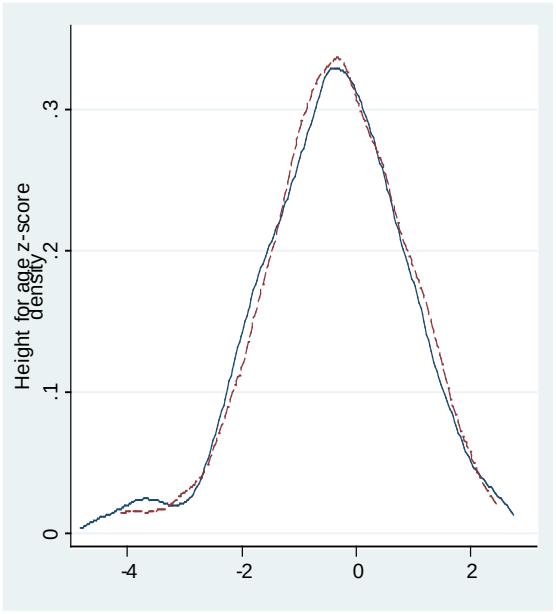
Figure 5 The First Stage Effects Of Changes in Sugar Consumption On Alcohol Abuse



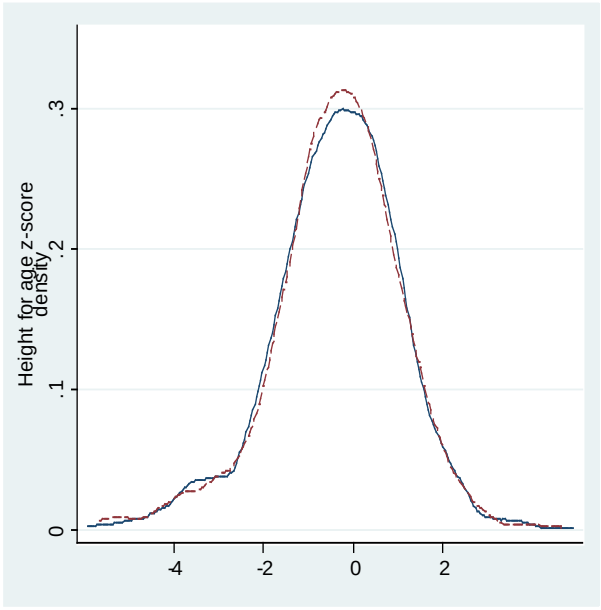
Note The solid line depicts the coefficients on the interactions between changes in sugar consumption and birth cohort dummies from the first stage estimation of equation (1) by IV in column 2 of Table 6. Dotted lines depict the 95 percent confidence bands

Figure 6 HFA Z-Score Densities In High And Low Regions, By Half-Yearly Date Of Birth

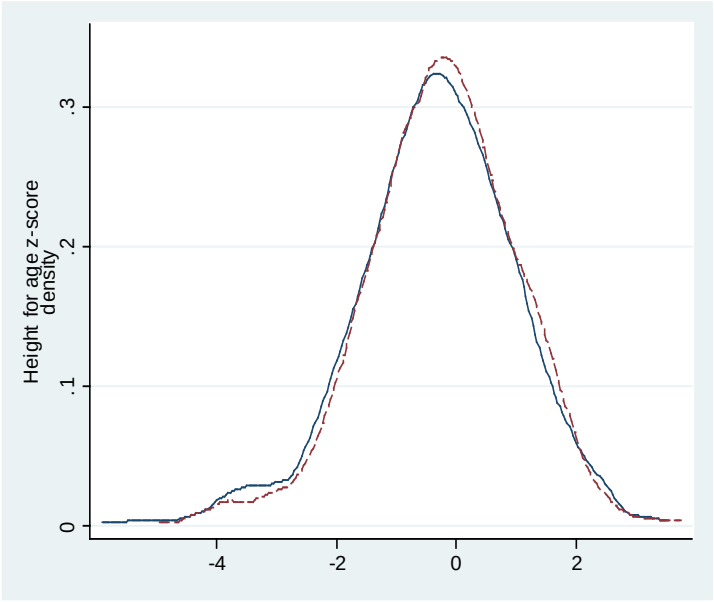
Panel A: Children Born between 1983h1-1984h2



Panel B: Children Born between 1987h1-1992h1



Panel C: Children Born between 1985h1-1987h2



Note: Solid and dotted lines represent low and high alcohol consumption regions respectively.

Table 1 Summary Statistics (I): Household and Regional Controls

Variable	# Obs	Mean	Std. Dev.
Oblast and regional data			
apt area per inhabitant (sq m)	1254	15.05	1.57
phones per capita	1254	11.31	5.32
% pop older than working age	1254	0.01	0.00
% pop of working age	1254	0.03	0.01
pop (th)	1254	2375.91	1191.92
road length per capita (km)	1254	77.02	56.65
income per capita (th)	1254	0.70	0.06
doctors per capita	1254	37.59	5.59
dispensary capacity per capita	1254	200.97	38.95
Alcohol cons per capita (weighed)	1254	12.25	1.53
Sugar prod (th)	771	175.3	261.7
Sugar cons (per capita)	1254	44.7	5.34
Simulated sugar cons (per capita)	1254	41.82	5.9
Wine production (mill)	1254	1.86	1.81
Beer production (mill)	1254	5.45	3.37
Vodka production (mill)	1254	2.27	1.44
Individual data			
nb of kids of age<5	1254	0.71	0.78
nb of kids of age>=5	1254	1.28	1.02
1= urbanized village	1254	0.06	0.23
1=rural	1254	0.30	0.46
1=community medical center	1254	0.30	0.46
1= mother's education: secondary /vocational	1254	0.22	0.42
1= mother's education: technical	1254	0.44	0.48
1= mother's education: college and above	1254	0.25	0.39
Living conditions index	1254	0.44	1.9
Asset index	1254	0.33	1.12
1=mother is a drinker	1254	0.56	0.49
1=father is a drinker	1254	0.77	0.41
1=boy	1254	0.51	0.49

Note: Data on households controls is from round 5 of the RLMS. Data on regional controls is from various official Russian statistical publications. The alcohol consumption data is from Nemtsov(1992, 1997), and Nemtsov and Nechaev (1991) and refers to total alcohol consumption (*samogon* included) per capita in the indicated years (expressed in liters).

Table 2 Summary Statistics (II): Health Measures

	(1)			(2)		
	Boys			Girls		
Variable	*Obs*	*Mean*	*Std. Dev.*	*Obs*	*Mean*	*Std. Dev.*
Height (cm)	649	123.7	19.4	605	124.9	20
Weight (kg)	676	26.7	9.9	605	26.5	10.6
HFA z-score	676	-0.34	1.4	605	-0.36	1.5
WFA z-score	676	0.08	1.3	605	-0.21	1.07
1=hospitalized [a]	676	0.04	0.18	605	0.03	0.17
1=health problems	676	0.38	0.44	605	0.42	0.45
1=sore throat[b]	676	0.18	0.38	605	0.17	0.38
1=diarrhea[b]	676	0.02	0.15	605	0.03	0.18
1=Self reported (good) health	676	0.54	0.49	605	0.49	0.53
1=immunized	676	0.67	0.48	605	0.62	0.49

Note: . [a] during the past year; [b] during the past three months. The data on health variables is from round 5 of the RLMS. HFA (WFA) z-scores represent standard deviations from the NCHS reference median height (weight) for a given age, as suggested by the World Health Organization

Table 3 Recorded and unrecorded alcohol consumption in Russia, 1970-1999

Year	Goskomstat Data		Researcher Estimated Alcohol Consumption	
	Registered alcohol	*Registered + samogon*	*Treml (1997)*	*Nemtsov (1992 and 2000)*
1970	8.3		12	
1971	8.4			
1972	8.6			
1973	8.8			
1974	9.5			
1975	9.9		13.1	
1976	10.2			
1977	10.4			
1978	10.6			
1979	10.6			
1980	10.5	13.5	14	13.8
1981	10.2	13.3		14.1
1982	10.1	13.1		13.9
1983	10.3	13.3		14.1
1984	10.5	13.8	14.2	14.2
1985	8.8	12.3	13.3	13
1986	5.2	10.2	10.6	10.5
1987	3.9	10	10.7	10.6
1988	4.4	8.3	11.2	11.4
1989	5.3	8.7	11.7	11.9
1990	5.6		11.8	12
1991	5.6		12.3	12.5
1992	5.0		13.8	13.5
1993	5		14.4	14
1994	6.8 (6.8)			14.6
1995	6.5 (9.3)			14.5
1996	(7.2)			14.4
1997	(7.5)			14.2
1998	(7.3)			13.9
1999	(7.6)			14.3

Table 4 The effect of the anti-alcohol campaign on child height

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Dep var=HFA z-score	***HFA z-score***									
	boys			*girls*			*boys*		*girls*	
	OLS	*OLS*	*OLS*	*OLS*	*OLS*	*OLS*	*IV*	*IV*	*IV*	*IV*
alc cons	-0.25	-0.27	-0.26	0.038	0.037	0.038	-0.76	-0.8	0.9	0.94
	[0.12]+	[0.15]+	[0.14]+	[0.14]	[0.14]	[0.14]	[0.35]*	[0.37]*	[0.6]	[0.7]
living cond.index			0.18 [0.1]		-0.001 [0.05]			0.04 [0.05]		0.03 [0.05]
ln (hh inc)		0.03 [0.07]				0.01 [0.07]				
mom edu :secondary	0.24	-0.23	-0.21	-0.2	-0.16	-0.2	0.29	0.28	0.14	0.12
	[0.33]	[0.34]	[0.35]	[0.14]	[0.14]	[0.14]	[0.32]	[0.34]	[0.34]	[0.3]
mom edu:vocational	0.24	-0.28	-0.24	0.75	0.75	0.74	0.26	0.27	0.27	0.29
	[0.21]	[0.2]	[0.21]	[0.26]**	[0.26]**	[0.27]**	[0.21]	[0.21]	[0.21]	[0.21]
mom edu: college+	1.4	1.3	1.38	3.2	3.0	3.2	1.5	1.51	1.51	1.47
	[0.47]**	[0.52]*	[0.50]*	[0.5]**	[0.52]**	[0.5]**	[0.52]*	[0.48]*	[0.47]*	[0.49]*
Dispensary capacity per capita	0.01 [0.006]*	0.01 [0.006]**	0.01 [0.006]**	0.007 [0.01]	0.007 [0.01]	0.007 [0.01]	0.04 [0.01]**	0.048 [0.2]**	-0.002 [0.1]	-0.004 [0.1]
Doctors per capita	0.10 [0.05]+	0.12 [0.6]+	0.10 [0.52]+	-0.08 [0.07]	-0.08 [0.07]	-0.07 [0.07]	0.12 [0.06]+	0.11 [0.05]+	0.13 [0.1]	-0.12 [0.1]
% pop working age	238.2 [168.1]	294.4 [155.2]+	237.2 [165.1]	226.1 [138.1]	226.2 [138.1]	225.1 [138.1]	187.2 [88.0]+	192.2 [89.0]+	271.2 [170.1]	249.3 [169.1]
% pop older working age	-132.2 [150.1]	-157.4 [172.3]	-231.2 [150.1]	-66.5 [180.1]	-66.5 [180.1]	-66.3 [178.1]	-132.3 [377.2]	-142.3 [323.2]	214.3 [170.5]	143.2 [165.5]
ln (pop)	13.7	17.2	13.9	-2.6	-2.7	-2.6	14.2	18.4	1.6	0.37
	[6.1]*	[6.3]*	[6.1]*	[6.1]	[6.5]	[6.2]	[4.1]**	[3.9]**	[8.3]	[8.3]
ln (real inc pc)	-3.7	-3.9	-3.9	4.8	4.78	4.8	-2.4	-2.3	4.6	4.8
	[3.1]	[4.1]	[4.1]	[3.2]	[3.2]	[3.3]	[3.1]	[3.2]	[4.1]	[4.3]
rural	-0.34+	-0.23+	-0.24+	-0.24	-0.24	-0.23	-0.34	-0.35	-0.12	0.24
	[0.18]	[0.11]	[0.13]	[0.19]	[0.19]	[0.2]	[0.19]	[0.17]**	[0.2]	[0.27]
Observations	649	649	649	605	605	605	649	649	605	605
Adj. R-squared	0.31	0.34	0.34	0.27	0.27	0.27	0.29	0.29	0.21	0.2

Note: +significant at 10%; * significant at 5%; **significant at 1%. Standard errors (in parentheses) are clustered at the oblast level. All regressions include oblast, birth year fixed effects and region specific trends. Observations are weighed using survey sample weights. Additional controls include road length per capita, phones per capita and dummies for number of kids younger than 5, and for number of kids between 5 and 17.

Table 5. The effect of the anti-alcohol campaign on child height: further results

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Dep var=HFA z-score	***HFA z-score***		***Log (height)***		***HFA z-score***		***HFA z-score***	
	boys	*girls*	*boys*	*girls*	*boys*	*girls*	*boys*	*girls*
	IV	*IV*	*IV*	*IV*	*IV*	*IV*	*IV*	*IV*
exposure	*combined*	*combined*	*combined*	*combined*	*utero*	*utero*	*Age 0-2*	*Age 0-2*
alc cons	-0.76	0.9	-0.03	0.019	-0.67	0.015	-0.9	-0.02
	[0.35]*	[0.6]	[0.012]*	[0.02]	[0.37]+	[0.21]	[0.32]**	[0.2]
Observations	649	605	649	605	649	605	649	605
Adj. R-squared	0.29	0.21	0.25	0.19	0.2	0.14	0.28	0.17

Table 6. The effect of the anti-alcohol campaign on child height: IV validity and robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Dep var=	**HFA z score**							**WFA z-score**	**HFA z-score**
Alc cons measure	***Alc cons***	***Alc cons***	***Alc cons***	***Total alc***	***Vodka prod***	***Wine prod***	***Beer prod***	***Alc cons***	***Current drinker***
Instruments	Sugar quotas	IV first stge	Sugar prod	IV first stage	Sugar prod	Sugar prod	Sugar prod	Sugar quota	Sugar quota
alc cons	-0.76 [0.35]*		-0.5 [0.21]*		-1.6 [0.6]*	-0.2 [0.1]+	-0.1 [0.1]	0.13 [0.36]	0.17 [0.4]
sug*1982		0.07 [0.09]		-0.01 [0.08]					
sug*1983		0.02 [0.12]		-0.015 [0.09]					
sug*1984		0.01 [0.16]		-0.02 [0.08]					
sug*1985		-0.25 [0.15]		-0.04 [0.07]					
sug*1986		-0.19 [0.05]*		-0.07 [0.04]+					
sug*1987		-0.14 [0.03]**		-0.09 [0.03]*					
sug*1988		-0.13 [0.07]+		-0.08 [0.04]+					
sug*1989		-0.13 [0.11]		-0.05 [0.08]					
sug*1990		-0.07 [0.11]		-0.02 [0.09]					
sug*1991		0.04 [0.13]		0.01 [0.1]					
sug*1992		0.12 [0.14]		0.05 [0.12]					
Observations	649	649	433	433	456	456	456	649	649
Adj. R-squared	0.31	0.89	0.29	0.62	0.28	0.15	0.1	0.2	0.2

Note. + significant at 10%; * significant at 5%; **significant at 1%. Standard errors (in parentheses) are clustered at the oblast level in columns 1-2 and 8-9 and at the region level in columns 3-7. Regressions in columns 1-2 & 8-9 (3-7) include oblast (region) and birth year fixed effects, as well as region specific trends, and all the controls from table 4. Estimation is by IV in all columns (with instruments as specified in the table), except for columns 2 and 4 where estimation is by OLS.

Table 7. The distributional effect of the anti-alcohol campaign on child height

	(1)	(2)	(3)
HFA z-core percentile	*bottom tercile*	*2nd tercile*	*Top tercile*
alc cons	-0.68 [0.35]+		
alc cons		-0.22 [0.12]+	
alc cons			0.02 [0.21]
Observations	250	189	210
Adjusted R-squared	0.39	0.42	0.31

Table 8. The effect of Prohibition on Chronic and Acute Health Conditions

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Dependent var	***Hospitalized past yr***	***Any health problems last 3 months***	***Self health status (1=good +)***	***Coughing***	***Sore throat***	***Gastric problems***	***Immuniz status***
	Panel A: Boys						
alc cons	0.13	-0.24	-0.35	-0.12	0.06	0.05	-0.24
	[0.09]+	[0.11]+	[0.02]+	[0.11]	[0.09]	[0.04]	[0.07]+
Observations	662	662	662	662	662	662	662
Adjusted R-squared	0.16	0.2	0.25	0.15	0.13	0.17	0.29
	Panel B: Girls						
alc cons	0.05	-0.05	-0.09	0.13	0.05	-0.03	-0.09
	[0.04]	[0.03]+	[0.5]+	[0.1]	[0.04]	[0.05]	[0.12]
Observations	621	621	621	621	621	621	621
Adjusted R-squared	0.17	0.2	0.22	0.18	0.14	0.12	0.2

Note. * significant at 10%; ** significant at 5%; *** significant at 1%. Standard errors (in parentheses) are clustered at the oblast level. All regressions include oblast and birth year fixed effects, as well as region specific trends, and all the controls from table 4. Estimation is by IV (using sugar quotas as instruments) in all columns.

Table 9 The effect of the campaign on immunizations for boys, by type of vaccine

	(1)	(2)	(3)	(4)	(5)
Vaccine type	***DTP***	***Polio***	***Measles***	***Hepatitis***	***Mumps***
alc cons	-0.21	-0.12	-0.04	-0.05	-0.09
	[0.08]*	[0.05]*	[0.03]+	[0.05]	[0.08]
Observations	621	621	621	621	621
Adj R-sq	0.31	0.37	0.33	0.29	0.33

Note. * significant at 10%; ** significant at 5%; *** significant at 1%. Standard errors (in parentheses) are clustered at the oblast level. All regressions include oblast and birth year fixed effects, as well as region specific trends, and all the controls from table 4. Estimation is by IV with sugar quotas as instruments.