

Does a Pint a Day Affect Your Childs' Pay? Unintended and Permanent Consequences of a Temporary Alcohol Policy Experiment*

Abstract

During a policy experiment in two Swedish regions in 1967 alcohol availability increased sharply, particularly for people under age 21. The policy experiment was abruptly ended after only 8.5 months due to a sharp increase in alcohol consumption. I exploit the distinct temporal, spatial and age-specific changes in alcohol availability induced by the policy experiment to estimate the long-term effects on those exposed to it *in utero*. I find that children *in utero* during the short period of increased alcohol availability have significantly lower educational attainments, earnings and increased welfare dependency rates at age 30 in comparison with the surrounding cohorts. Any direct effects of the increased availability on birth-cohort composition (e.g. through an increase in unplanned pregnancies) are not driving the results as the richness of the data allows for a focus on exposed children conceived before the policy experiment started. The results provide compelling evidence that investments in early-life health can yield large effects on outcomes later on in life.

Keywords: Early-life conditions, natural experiment, prenatal health

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1 Introduction

How influential are prenatal conditions for later life outcomes? Providing a credible answer to this question is challenging for at least two important reasons: (i) Without explicit knowledge about what is causing the adverse prenatal conditions, it is very difficult to rule out that the same underlying causes which lead to the poor prenatal environment also lead to a poor childhood environment. Hence, distinguishing between the effects of prenatal and post-natal environment on later life outcomes is generally challenging. (ii) Even if one would know exactly what was causing the adverse conditions *in utero*, the waiting period before the adult outcomes of interest are realized is daunting. This has lead researchers to instead focus on short term outcomes such as infant health, or childhood cognitive ability tests scores. However, some insults *in utero* are not necessarily evident at birth or even during early childhood but first appear much later in life; see e.g. Barker (1998). Focusing only on immediate or short term outcomes may therefore lead to false conclusions about the full influence of early life conditions.

In order to give further insights on how important *in utero* conditions are for subsequent outcomes this study focuses on the long-run effects of *in utero* exposure to a policy experiment which exogenously and temporarily increased alcohol availability in two Swedish regions (jointly containing 12% of the population) in the end of the 1960s. During the policy experiment alcohol availability increased sharply since regular grocery stores were allowed to sell strong beer¹. Prior to and after the experiment, off-premises sales of strong beer, wine and spirits were only allowed in the state-owned alcohol retail monopoly stores (Systembolaget). With the underlying assumption that strong beer and liquor were reasonably close substitutes², the policy experiment intended to induce a shift in consumption from high alcohol (spirits) to lower alcohol (strong beer) beverages by increasing the relative availability of the lower alcohol content beverage. However, for those without the possibility to buy alcohol at Systembolaget

¹ Strong beer is restricted to a maximum alcohol content of 4.48 % by weight.

² As will become clear below this assumption seems not to have been valid in the present context.

(i.e. those under age 21), the policy shift implied that a *higher* alcohol content beverage became relatively *more* available during the policy experiment rather than the other way around.³ The experiment was planned to last from November 1967 until the end of 1968 but it was discontinued prematurely due to alarming reports of a sharp increase in alcohol consumption in the experiment regions, and a deterioration of temperance particularly among young people (SOU 1971:77). Figure 1 show the trends in strong beer sales for the treatment regions and the country as a whole from 1962 through 1972. During the first six months of 1968, strong beer consumption per capita increased ten-fold in the treatment regions as compared to the year prior to the experiment.

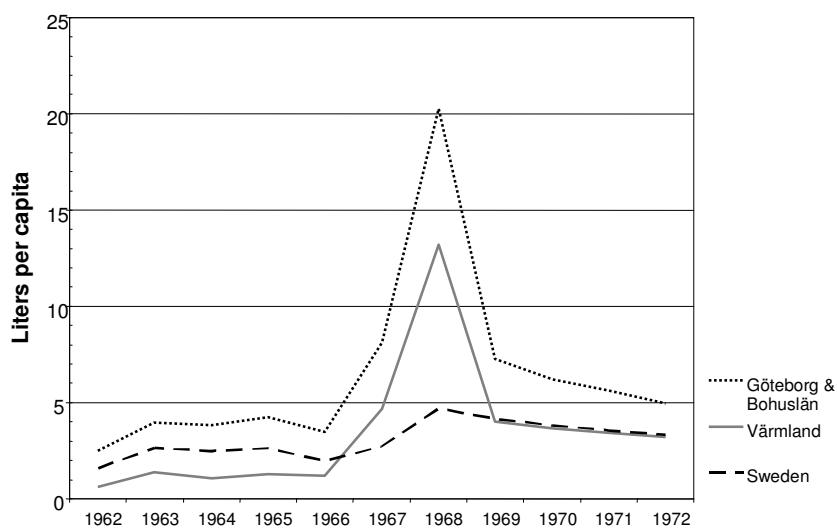


Figure 1 Yearly strong beer consumption per capita. *Source:* SCB 1962-72

At the time of the policy experiment relatively little was known about the potential negative effects of alcohol consumption during pregnancy on child development. The first general warning about the association between alcohol consumption during pregnancy and birth defects was issued by the US Surgeon General and the Swedish National Board of Health and Welfare in the early 1980s. These warnings emerged as a response to the increasing empirical evidence from the early 1970s and on indicating a negative association between heavy prenatal alcohol exposure and children's health.⁴

³ This discrepancy in changes in availability between young and old consumers were either not realized by the implementers or ignored in the following evaluation. See SFS (1967:213) and SFS (1961:159) for the rules in effect during the experiment.

⁴ The range of damage includes mild and subtle changes, such as slight learning difficulties or physical abnormality, through full-blown Fetal Alcohol Syndrome (FAS) including severe

Still, even today there exists considerable controversy (both scientifically and in the public debate) about the potential effects of low-to-moderate drinking during pregnancy and child development.⁵ The main reason is the concern that the effects of lower levels of alcohol consumption during pregnancy are biased by omitted variables correlated with both maternal alcohol consumption and children's health.

The distinct temporal, spatial and age-specific changes in alcohol availability induced by the policy experiment provide a truly unique opportunity to solve many of the identification problems present in previous work on the long-run effects of prenatal alcohol exposure. Firstly, due to its sharp restriction in time, the experiment allows for a comparison of the adult outcomes of the cohort of children born in the experimental regions who were *in utero* during the experiment with the outcomes for the surrounding "unexposed" cohorts. Secondly, the spatial restriction allows for a simultaneous comparison with the outcomes for children belonging to the same cohort but who were born in the control regions. This feature reduces the problem of general time effects confounding the estimate of the relationship of interest. Thirdly, by capitalizing on the age-specific changes in alcohol availability within the treatment regions it is possible to account for unobserved regional specific shocks affecting the outcomes of children born in treatment and control regions differentially. Finally, importantly the sharply defined time period of increased availability enables a focus on children exposed to the experiment *in utero* but conceived prior to the experiment started. This mitigates the concern that the increase in alcohol consumption also may have change the composition of births and thereby indirectly the child's outcomes. Hence, I effectively avoid attaining biased estimates of the relationship of interest due to indirect effects caused by the experiment (e.g. via an increased frequency of unplanned pregnancies).⁶

Using administrative data on all children born in Sweden between 1964 and 1972, I find that the sharp increase in alcohol consumption during the experiment has had a substantial impact on the outcomes of those still *in*

learning disabilities, growth deficiencies, abnormal facial features, and central nervous system disorders.

⁵ This is clearly reflected by the answers in surveys about drinking during pregnancy. In the US up to 50 percent of the childbearing age women drink and 16 percent of them continue drinking during pregnancy (CDC, 2002). Göransson et al. (2003) surveyed pregnant women in Stockholm, Sweden regarding their consumption of alcohol. They found that 46 percent reported a binge drinking (more than 5 standard drinks on a single occasion) episode once per month or more often in the year prior to becoming pregnant. During pregnancy 30 % reported regular alcohol use. In a Danish study, 57% of the pregnant women without previous children reported at least one binge drinking episode during the first half of the pregnancy (Kesmodel et al., 2003). See WHO (2004) for international consumption levels.

⁶ See Kaestner and Joyce (2001) for evidence of the effects of alcohol use on the probability of unwanted pregnancies. Watson and Fertig (2008) show that MLDA laws adversely affected infant health mainly through the effect on composition of births.

utero during the experiment. In particular, the children with the longest prenatal exposure to the experiment (between 5 and 8.5 months in utero) who were born by mothers under the age of 21 at delivery have on average 0.3 fewer years of schooling and lower high school and college graduation rates. They are less likely to be employed, have lower earnings and a higher welfare dependency rate compared to the surrounding cohorts. The effects on adult outcomes are in general more pronounced among males, suggesting that males are more vulnerable to adverse conditions *in utero*. Similarly, previous work has related a reduced sex-ratio at birth to adverse maternal conditions during pregnancy.⁷ In line with these studies I find that the proportion of males in the most exposed cohorts is significantly more female.

Interest in the effects of prenatal alcohol exposure might mainly be concentrated to the medical sciences, however the main contribution of this study is to the broader and growing literature focusing on the early life determinants of long-run economic outcomes.⁸ With few exceptions⁹, the previous work on effects of in utero conditions has focused short run outcomes such as infant health. This study distinguishes itself from most of the previous work on early-life conditions and adult outcomes by providing relatively clear suggestions for policy tools that potentially can reduce inequalities in long-term economic outcomes. Furthermore, the results suggest that investments in early-life health may not only be more humane compared to compensatory postnatal investment in terms of health outcomes, but potentially also an effective way of increasing human capital accumulation and reduce inequality in human capabilities (Almond, 2006; Cunha and Heckman 2006, 2009).¹⁰

The remainder of the paper is structured as follows. Section 2 provides an overview of previous work on the consequences and mechanisms of prenatal alcohol exposure on child development and details regarding the policy experiment. Section 3 describes the data and the empirical strategy. Section 4 presents the results and robustness checks and section 5 concludes.

⁷ C.f. Triver and Willard (1973), Wells (2000), Norberg (2004), Almond and Edlund (2007).

⁸ c.f. Currie (2009) and the references there in.

⁹ For example, Van den Berg, Lindeboom and Portrait (2006) investigate the impact of early life economic conditions on mortality later in life; Case, et al. (2005) quantify the lasting effects of childhood health and economic circumstances on adult health and earnings; Banerjee et al. (2007) find that economic conditions during childhood decreases stature among males but not life expectancy for females. Utilizing twin data, Black et al. (2007) shows that low birth weight (a common proxy for adverse conditions *in utero*) is strongly negatively correlated with cognitive ability and stature at age 18-20 as well as subsequent labor market outcomes. Almond (2006), and Almond and Mazumder (2005) investigate the impact of the Spanish influenza pandemic on subsequent socio-economic and health outcomes respectively of those *in utero* during the peak of the epidemic. Almond et al. (2007) study the impact of the Chernobyl accident on Swedish children exposed to the fallout while still *in utero* and finds significant negative effects on educational attainments.

¹⁰ See Currie (2009) for a recent and comprehensive review.

2 Background

2.1 Alcohol policy in Sweden and the strong beer experiment

Alcohol sales in Sweden are strictly regulated by means of an off-premises retail monopoly (Systembolaget). The only alcoholic beverages permitted in regular grocery stores are those containing less than 3.5 % alcohol by volume (~2.8 % by weight). The current form of the alcohol retail system has been in effect since 1955. Since then, the consumption pattern has changed radically. Sweden traditionally belonged to the “spirit-drinking” countries, but during the last 50 years the consumption of spirits has declined substantially and has gradually been replaced by wine and beer products (Leifmann, 2001). The dominant alcoholic beverage today is the strong beer that accounts for 29 % of total alcohol consumption (SNIPH, 2005). One of the contributory factors in this changing pattern is active measures taken to encourage the substitution of consumption from spirits to beverages with lower alcohol content.¹¹

The policy experiment with free sales of strong beer (maximum alcohol contents of 5.6 % by volume, i.e. ~4.48 % by weight), running from November 1967 through July 1968 in the Göteborgs-och Bohuslän and Värmland regions is an example of a policy of this nature.¹² During the experiment, off-premises sales of strong beer were allowed in regular grocery stores as compared to only in the Systembolaget stores prior to and after the experiment.¹³ The regulations for wholesale trading in strong beer also changed. Anyone entitled to sell or serve beer was allowed to buy strong beer directly from a Swedish brewery or, in the case of imported beer, through a wholesaler.¹⁴

The original intention was to continue the experiment until the end of 1968, but soon after it was introduced reports of a sharp increase in alcohol consumption in the experimental regions, especially among young people

¹¹ See Room (2002) for a comprehensive review of Swedish and Nordic alcohol policies after 1950.

¹² The setup and results of the experiment are described in detail in the APU report from the experiment (SOU 1971:77), upon which this section draws. In the report no motivation is given as to why the two regions were selected from the pool of 25 regions.

¹³ At the end of 1968, 1 530 retail outlets were licensed for sales of beer (during the experiment also strong beer) in *Göteborg och Bohuslän* region as compared to the 26 Systembolaget stores in operation prior to and after the experiment.

¹⁴ The aim of the experiment was that the wholesaling of strong beer also was to be carried out under similar conditions as those that would exist with free sales. As a result, wholesalers were able to order goods directly from foreign breweries. All wholesalers were however obligated to report the amount of strong beer shipped to retailers.

was received. This caused the implementing authority, the Alcohol Policy Commission (APU), to propose an interruption, and in the middle of July 1968 the experiment was discontinued prematurely.

The consumption of strong beer increased dramatically in the experimental regions during the experiment. In the first half of 1968 consumption increased from the 1967 level of 1.2 million liters to 10.5 million liters in Göteborgs- och Bohuslän. In Värmland the increase was even more drastic. In the first six months of 1967 0.2 million liters were sold compared to 3.0 million liters during the same months in 1968. If summarized over both regions consumption increased by almost 1,000%. Per capita, the consumption of strong beer increased from 1.8 liters during the first six months of 1967 to 15.3 liters in the same period in 1968 in Göteborgs- och Bohuslän. The corresponding figures for Värmland were 0.7 liters and 10.6 liters per capita for the two periods. From Figure 1 it is also clear that the consumption in the country as a whole rose during the experiment. The main part of this increase is explained by the fact that the two experimental regions constituted a substantial share of the total population (12% in 1968) and hence had a large impact on the national average. If excluding the experimental regions, the rest of the country showed an increased consumption of 26% from the first half of 1967 to the same period in 1968. Figure 1 also shows that before the experiment the trends in consumption of strong beer in the two experimental regions followed the national average reasonably well. During the policy experiment, consumption boomed and afterwards it fell back again. However, note that strong beer consumption in the experimental regions remained at an elevated level compared to the pre-experiment period even after the experiment had ended. This indicates that a short-term experiment could have long-term effects on consumption (SOU 1971:77).

The geographical distribution of consumption reveals a clear connection between sales and population density. Per capita consumption was highest in Gothenburg (684,626 inhabitants) followed by Karlstad (53,208 inhabitants) and Uddevalla (36,480 inhabitants). The reason for this pattern is probably greater availability in urban areas. Another explanation might be that people living in rural areas bought strong beer when visiting the cities. However, it is also likely that some cross-border shopping for beer occurred during the experiment at least by consumers in the neighboring regions. This suggests that an experiment including the whole country would have generated a smaller increase in consumption per capita. The extent of cross-border shopping is unknown but it seems unlikely that it had any major influence on total sales.¹⁵

¹⁵ The reason is that while availability increased, prices (if anything) increased during the experiment (SOU 1971:77). In the empirical section, I also check whether the experiment generated any spill-over effects on children born in the neighboring regions.

There are excellent opportunities for evaluating the impact of the experiment on substitution between wine, spirits and strong beer. The Systembolaget stores kept exact records of the volumes sold per quarter in each region prior to, during, and after the experiment. Compared to the first half of 1967, there was a decrease in liquor sales in the first half of 1968 in the two experimental regions of ten and of five percent respectively, while the wine sales did not change to any great extent. For the rest of the country, the decline in liquor sales was four percent, while the wine sales increased by eight percent. This indicates that the experimental regions differed from the rest of the country by having larger decreases in liquor sales and no increase in wine sales. Which suggests that, in the experimental regions, liquor and wine was substituted by strong beer. The changes in liquor and wine sales were, however, rather small and did not compensate for the substantial increases in sales of strong beer.

Perhaps a more important question is how the consumption of medium beer¹⁶ was influenced. It is highly likely that the increased sales of strong beer lead to a decline in the sales of medium beer, as these products are arguably closer substitutes. Unfortunately, there are no records of the quantity of medium beer sold at the regional level. There are however data on aggregate monthly sales. The national consumption of medium beer increased by only 14% during the first six months of 1968. This should be compared with an increase of 25 % for the first three quarters of 1967 and 35 % during the fourth quarter of 1968. These figures indicate that the experiment led to a reduction in the increase of medium beer sales of 10 percentage points, and that strong beer to some extent replaced medium beer in the experiment regions. During the first six months of 1967, 91 million liters of medium beer was sold, which means that the reduction should have been around 10 million liters overall. This quantity should be compared with the extra 11.8 million liters of strong beer consumed in the experimental regions. Based on these calculations, the average increase in the experimental regions in terms of liters of 100% alcohol has previously been estimated to be around five percent (SOU 1971:77). However, potential heterogeneous consumption responses to the increased availability between different sub-populations have not been taken into consideration.

The immediate impact on harms was only assessed in terms of number of persons arrested for drunkenness. These data show no clear effects of the experiment. However, during this period there was a general increase in alcohol consumption and a general decline in the number of persons apprehended for drunkenness. There were also reports suggesting that the police authorities acted on drunkenness in ways which did not show up in the official statistics (SOU 1971:77). Moreover, in the late spring of 1968

¹⁶ Medium beer may contain at maximum 3.6 % alcohol by weight.

the implementing authority, the Alcohol Policy Commission, surveyed the local child welfare commissions (barnvårdsnämnder), the temperance commissions (nykterhetsnämnder), the local education authorities and the police authorities in the experimental regions regarding their experiences of the free sales of strong beer hitherto. The main conclusion of this survey is that there was a negative impact on temperance during the experimental period. The police authorities underscored that the temperance situation had deteriorated particularly among young people. The main nuisances reported were an increased level of disorderly conduct and littering in connection with an immense consumption of strong beer. An increase in drunken driving was also noted. Furthermore, urban areas seem to have been more affected than rural areas (SOU 1971:77).

One explanation of the particularly detrimental effects on temperance among young people is probably that they experienced the largest increase in the availability of alcoholic beverages during the experiment.¹⁷ The age limit in Systembolaget stores was set to 21, and prior to the experiment this was the only off-premise place where strong beer could be bought.¹⁸ The minimum purchasing age for beer in regular grocery stores during the experiment was 16, although the application of this law was very weak (SOU 1974:91). Hence, in line with the intention of the policy shift for the large majority in the experiment regions the policy implied that a lower alcohol content beverage became more easily available. However, this only resulted in a small reduction in consumption of higher alcohol content beverages. On the contrary for those without the possibility to buy alcohol at Systembolaget (i.e. those under age 21), the policy shift implied that a *higher* alcohol content beverage became relatively more available during the policy experiment than before or after. These age-specific differences in changes in alcohol availability provide a plausible explanation for the reported differences in the effects of the policy. Moreover, it also provides an important prior suggesting that the children *in utero* during the policy shift, who was born by mothers under age 21 are likely to have been affected most.

Estimation of the exact magnitude of the changes in consumption between the two different age-groups is however hampered by the lack of data on alcohol use among sub-populations in the experimental regions. However, from a nationwide survey among young people aged 15 through 25 conducted in the spring/summer of 1968, beer consumption was 44 %

¹⁷ For the effects of alcohol availability on consumption patterns in general see e.g. O'Malley and Wagenaar (1991) for US evidence, Carpenter and Eisenberg (2007) for Canadian evidence, and Norström and Skog (2005) for Sweden. Several previous studies focusing on young people have found responsiveness to policies pertaining to availability, such as the minimum legal drinking age (MLDA) laws, see e.g. Moore and Cook (1995).

¹⁸ On-premise consumption was in relationship to off-premise consumption very low the time of experiment.

higher among young people than in the population as a whole.¹⁹ This suggests that the average increase in consumption among young people likely exceeds the previously estimated average increase of in terms of 100% alcohol of five percent. The survey also reveals that in 1968, 90 percent of the females and 97 percent of the males reported that their alcohol debut occurred before turning 21 and that the abstainer rates in these age categories was low²⁰ (SOU 1971:77).

2.2 Consequences of prenatal alcohol exposure

While the medical professions beliefs regarding the impermeability of the placenta were shattered in the early 1960s in connection with the Thalidomide tragedy (see e.g. Dally, 1998), the first scientific support on a negative association between heavy maternal alcohol consumption during pregnancy and children's health did not emerge until 1968 in work by Lemoine et al. (1968) in France. Jones and Smith (1973) subsequently published similar findings internationally and coined the Fetal Alcohol Syndrome (FAS).²¹ In addition to confirmed maternal alcohol consumption during pregnancy, the FAS diagnosis criteria require the following conditions in infancy: growth deficiency, facial anomalies and neurological abnormalities. Other effects associated with prenatal alcohol exposure are increased risk of miscarriage and low birth weight. Many children that are not obviously physically affected, or do not show any easily defined behavioral problems may still suffer from alcohol-induced central nervous system deficits. Streissguth et al. (1991) demonstrated that there is a predictable long-term progression of disorders into adulthood resulting from prenatal exposure to alcohol. They show that, among other things, poor judgment, distractibility, difficulty in perceiving social cues and low IQ levels, were common among individuals exposed to alcohol *in utero*.²² The evidence on the consequences of medium and lower levels of alcohol consumption during pregnancy on birth outcomes is, however, less conclusive.²³ No consensus has been reached on any threshold level, either in

¹⁹ A summary of this survey can be found in SOU 1971:77. Unfortunately the raw data from this survey is not available for further analysis.

²⁰ In the highest, middle and lowest social strata 2, 8 and 10 percent of the young women (aged between 17 and 25) reported no alcohol consumption in 1968 (SOU 1971:77).

²¹ Olegård et al. (1979) is the first to study using Swedish data to estimate the effects of prenatal alcohol exposure on child outcomes. They find that alcohol exposure is related to an increased level of behavioral problems in childhood.

²² The set up and findings from this and other studies on the same single cohort of children followed from birth to the age of 25 and born in Seattle in 1974/1975 is summarized in Streissguth (2007). In common with the present study the information on maternal alcohol consumption was elicited when very little was known about the risks associated with alcohol use during pregnancy.

²³ See e.g. Rusell (1991) and Henderson et al. (2007) for reviews of this literature.

terms of the amount or incidence of alcohol consumption during pregnancy with regards to the more subtle effects on health.²⁴

West et al. (1994) and Goodlet and Horn (2001) summarize the vast medical literature focusing on the particular biological mechanisms behind the casual link between alcohol exposure and fetal development. Briefly, alcohol may affect the developing fetus directly as it readily crosses the placenta and passes to the fetal cells, but also indirectly by reducing the supply of oxygen and nourishment. In addition, the dose and pattern of alcohol use seem to be important in determining the severity of the damage. Animal experiments have suggested that a small dose consumed in a massed “binge drink” manner is more damaging than a larger but more spaced dose (Bonthius and West, 1990).²⁵ Furthermore, the detrimental effect of alcohol on fetal development is difficult to isolate to any specific timing of exposure during gestation, although the types of damage may vary between trimesters. From animal studies it has been found that the central nervous system is susceptible to damage during all three trimesters. A critical period for behavioral outcomes among human subjects is less clearly defined.²⁶ In addition to direct effects on the central nervous system and brain development, prenatal alcohol exposure may also alter the development and functioning of the immune system, leading to a higher susceptibility to infections (Zhang et al., 2005). The most critical damage inflicted by heavy exposure on organs and extremities mainly seems to occur as a result of exposure in the first trimester. Hence, prenatal alcohol exposure may reduce the health stock through several different paths.

However, since randomly administrating alcohol of different doses to pregnant women is unethical previous human studies are likely to be plagued by omitted variable bias. That is, since stated alcohol consumption patterns during pregnancy could be correlated with unobserved family characteristics directly related both to the child’s outcomes and alcohol consumption (e.g. poverty or maternal mental health), the interpretation of non-experimental estimates of the effects of prenatal alcohol exposure on child development is difficult.²⁷ When it concerns lower levels of maternal alcohol consumption and more subtle effects on child development not necessarily evident at birth, this is most likely an even greater concern.

Since randomization of treatment at the individual level is not feasible I instead focus on the exogenous changes in alcohol availability induced by

²⁴ See e.g. CDC (2004).

²⁵ This is consistent with the results from Streissguth et al. (1990, 1994) which found a binge drinking consumption pattern to be the best predictor of academic achievements.

²⁶ c.f. Coles (1994) for a discussion of the difficulties of identifying critical periods of alcohol exposure on offspring outcomes in human and Rice and Barone (2000) for a thorough review of critical periods of vulnerability for the developing nervous system.

²⁷ Additionally, eliciting correct information on maternal alcohol use during pregnancy is complicated by desirability and recall biases.

the strong beer policy experiment to mitigate the problems of omitted variable bias. The Swedish register data provides a unique opportunity to investigate the effects of an exogenous increase in alcohol consumption during pregnancy on the long-term outcomes of the child. Considering the type of weekend binge drinking pattern common in Sweden²⁸, the reports of a sharp deterioration in temperance among young people and the suggested particularly damaging effects on the fetus of binge drinking, clearly the long-run outcomes of children exposed to the experiment *in utero* may have been affected. Moreover, additional negative effects of the increase in alcohol consumption may come through changes in other behaviors that are typically associated with alcohol consumption, such as smoking, and which also are associated with poor birth outcomes.²⁹

3 Empirical strategy

The main hypothesis to be tested in this paper is whether the exogenous increase in alcohol availability during the experiment resulted in adverse adult outcomes for the children *in utero* at the time. To do this we utilize the LOUISE database assembled by Statistics Sweden covering all individuals in the age range 16-65 living or working in Sweden between 1990 and 2004. The LOUISE data are register-based and, apart from information on year and month of birth, gender and region of birth, they also contain detailed information on educational attainments, labor market outcomes and welfare payments received during the observation period. Using the so-called “multi generational” register, we have also linked each individual in the data to his/her biological parents.

In the main analysis, all first-born individuals alive in 2000 and born in Sweden between 1964 and 1972 are retained.³⁰ The children born in the 5

²⁸ The pattern of drinking in Sweden has been characterized by non-daily drinking, irregular binge drinking episodes (e.g. during weekends and at festivities), and the acceptance of drunkenness in public; see e.g. K uhlhorn et al. (1999). In general studies on fetal alcohol exposure typically consider single binge drinking episodes (i.e. not daily) as low-to-moderate exposure, which is important to consider when interpreting the estimated effects. Given that heavy alcohol abuse is fairly uncommon in Swedish youths, it seems more likely that any effects found are due to the binge-drinking type of exposure rather than the continuous daily heavy exposure typically needed for the characteristic FAS symptoms to occur.

²⁹ Attempts to assess the effects of alcohol use in comparison with the use of other drugs have however suggested that prenatal alcohol exposure may result in broader and more long lasting effects compared to other drugs, see e.g. Day and Richardson (1994). Still, to be clear the empirical strategy employed will identify the prevalence and importance of the net effect of the increase in alcohol consumption in the policy regions.

³⁰ First-borns are first of all singled out due to the assumption that people without previous children are more likely to react to a temporary increase in alcohol availability. Secondly, given the focus on mothers under age 21, adding higher order birth children will only have a marginal effect on the size of the treatment group since very few women give birth to two children before age 21.

regions neighboring the experimental regions are at first excluded in order to avoid diluting the estimates due to potential spill-over effects from the experiment. As the experiment was implemented at the regional level, this study uses panel data for regions.³¹ However, for the reasons discussed above, to allow for the age-specific differences of the policy shift on availability and consumption among young and older mothers, the sample is further partitioned with respect to the age of the mother at delivery (below/above age 21).

Based on exposure to the policy the children born in the treatment regions are divided into four groups: (1) those born prior to the initiation of the experiment, and hence only exposed after birth; (2) those exposed to the experiment *in utero* but conceived *before* the experiment started; (3) those exposed to the experiment *in utero* but conceived during the course of the experiment; and (4) those who were conceived after the end of the experiment and who, as a result, were not exposed either during pregnancy or after birth.³² In the baseline estimations, I focus in particular on children belonging to group (2). The main reason is that it seems reasonable to assume that the experiment did not affect the timing of conception for this group of children. This is important, as several studies have found an association between alcohol consumption and risky behavior among young people (Kaestner and Joyce, 2001; Carpenter, 2005; Grossman and Markowitz, 2005; Carpenter and Dobkin, 2009). Indeed Watson and Fertig (2008) find evidence suggesting that Minimum legal drinking age laws in the US affect infant health mainly through its effect on the composition of births.³³ By focusing on children conceived prior to the experiment started, biased estimates of the relationship of interest due to indirect effects caused by the experiment (e.g. via an increased frequency of unplanned pregnancies) is effectively avoided.

In order to allow for heterogeneous effects of the experiment depending on duration and timing of exposure during gestation, the children in group (2) are further divided into those whose mothers were in the first half of the pregnancy period (months 1-4), and those in the second half (months 5-9) at the start of the experiment. The empirical analysis focuses on the first group

³¹ Sweden is divided into 25 regions (Län).

³² Table A 1 in Appendix A presents a schematic overview on the estimated maximum and minimum number of weeks of *in utero* exposure, as well as the estimated gestational age at the start of the experiment.

³³ Watson and Fertig find fairly small effects on birth outcomes, although the authors also suggest that this could be due to that the MLDA only had a modest effect on consumption. Additionally, birth outcomes such birth weight is likely not an ideal measure when it comes to alcohol exposure since birth weight is mainly determined in the later stages of the pregnancy. Since drinking during pregnancy typically *decreases* sharply with gestation, it is notable that Watson and Fertig find significantly negative effects on birth-weight from the MLDA changes. This could indicate that the full effect on fetal development from the MLDA policies is larger than what the effects on birth-outcomes reveal.

since they were under risk of prenatal exposure for the longest duration. In addition the first group (months 1–4) most likely experienced a particularly high risk of being exposed to alcohol due to the experiment since a substantial proportion of the early-pregnancy mothers probably did not even realize that they were pregnant for some time during the experiment.³⁴ However, in the empirical analysis the impact of exposure to the experiment on children in late gestation and the three other exposure groups are considered as well. Again, as noted above, one should furthermore bear in mind that the awareness of the risks associated with alcohol consumption during pregnancy was very low at the time of the experiment.

The baseline empirical model used to test the outlined hypothesis is the following difference-in-difference-in-differences (DDD) model,

$$\begin{aligned} \text{OUTCOME}_{c,t,mom<21} = & \alpha_0 + \beta_1 \text{EXPOSURE}_{c,t,mom<21} + \eta_c + \delta_t + \phi_{mom<21} \\ & + \gamma_{c,t} + \lambda_{c,mom<21} + \mu_{t,mom<21} + \varepsilon_{c,t,mom<21} \end{aligned} \quad (1)$$

which is estimated by OLS on data aggregated by birth quarter, age of mother (below/above 21) and region of birth.³⁵ In equation (1) OUTCOME is the outcome of interest (average years of schooling, share of high school graduates, share on welfare, average earnings etc.). EXPOSURE is equal to 1 if the child is born by a mother under the age of 21 at delivery in the treatment regions and conceived between July and October 1967, and otherwise 0.³⁶ Thus β_1 is the parameter of interest and it reflects the impact of the experiment on the outcomes of the children *in utero* at the time in adulthood. δ_t and η_c are period (quarter/year) and region of birth effects respectively. $\phi_{mom<21}$ is a parameter indicating whether the child was born by

³⁴ Today the average pregnancy is recognized 5-6 weeks after conception (see Floyd et al., 1999). It seem reasonable to assume that in the late 1960s recognition of pregnancy most likely occurred even later on average due to the lack of readily available pregnancy test. This may also have increased the probability of changing their consumption pattern due to the increase in availability. Even in the absence of information of the direct adverse effects of alcohol consumption on child development this seems likely since mothers in late pregnancy are presumably aware of the risk associated with intoxication in general (e.g. increased risk of accidents etc.).

³⁵ The aggregated data is used instead of individual level data as the treatment varies at this level. The aggregate data is preferred in order to avoid problems of within-region correlations in the error term which may otherwise result in underestimated standard errors as Donald and Lang (2007) show. Using raw aggregated data, as is done in this case yields qualitatively similar results as when using the residual aggregation method, and hence adjusting for background characteristics available in the data as suggested by e.g. Bertrand et al. (2004).

³⁶ Hence in the main estimations the “quarter” of birth is defined as Q1=Jan.-March, Q2=April-July, Q3=Aug.-Sept., Q4=Oct-Dec, so as to be in a better position to capture the full effect on those conceived just prior to the experiment.

a mother under the age of 21 at the date of birth. The time (δ_t) and region (η_c) parameters control for region and quarter of birth specific effects affecting adult outcomes.³⁷ The $\phi_{mom<21}$ parameter accounts for fixed differences in outcomes between children born by mothers under the age of 21 and those above. The interaction terms $\gamma_{c,t}$, $\lambda_{c,mom<21}$ and $\mu_{t,mom<21}$ account for many other factors that are also related to the outcomes of interest. For example, as seen in Table 1, over the observation period the number of mothers under the age of 21 decreased somewhat and hence the composition of these mothers may have changed in terms of parental ability. The quarter*youngmom effect ($\mu_{t,mom<21}$) account for such and similar compositional changes throughout the observation period. The region*young mom effects ($\lambda_{c,mom<21}$) control for fixed regional differences in the composition of mothers giving birth to children under the age of 21. $\epsilon_{c,t,mom<21}$ is the error term.

Note that the DDD model accounts for many possible confounders, and perhaps most importantly also regional common shocks coinciding with the experiment also affecting the children's outcomes. Hence, in order for a contemporary local shock to bias the estimate of β_1 in equation (1) not only must the timing of the temporary unobserved shock precisely coincide with the timing of the temporary policy experiment; but it must also only affect the adult labor market outcomes of children born by mothers under the age of 21 and *not* children born by older mothers.³⁸ While it is not possible to provide a direct test of this assumption, in the following sections, besides the baseline DDD estimates, results from a number of robustness checks assessing the plausibility of this identifying assumption is also reported. Moreover, there are no indications of that a type of shock fulfilling these conditions or other changes in policy occurred simultaneously as the policy experiment in either the treatment or control regions.

³⁷ See Buckles and Hungerman (2008), Costa and Lahey (2005) and Dobelhammer and Vaupel (2001) for evidence on the importance of season of birth effects on adult outcomes.

³⁸ Note also that the same conditions must hold in order for a common shock *later in life* to bias the estimates. In addition, the use of quarter of birth data and the fact that Swedish children born during the same calendar year typically start school at the same time, potential disruptive behavior of a few exposed class mates will not bias the estimate through peer effects, unless the peer effect only affects the children born in the same quarter of the year and not earlier or later. Something that seems highly unlikely.

4 Results

To preview the central results, the cohort of children born by young mothers and who were exposed to the experiment for the longest duration *in utero* have significantly lower earnings, higher probability of no earnings at all, lower educational attainments and higher welfare dependency rates, compared to the surrounding cohorts. For most outcomes the effects of the experiment are more pronounced for males than for females, suggesting that males are particularly affected by adverse conditions *in utero*. In line with these results the cohort *in utero* is furthermore significantly more female, and while there is no significant effect on the cohort size or month of birth of females, there is a negative effect on both the month of birth and cohort size of males. These findings indicate that those most heavily exposed were more likely to be either spontaneously aborted or born prematurely. The results are furthermore robust to a number of specifications checks, such as the inclusion of maternal fixed effects, changes in the definition of timing of exposure and placebo estimates where children born in the neighboring regions are pretended to be exposed to the policy changes instead.

4.1 A first look at the data

Table 1 presents descriptive statistics for the adult outcomes of children born in the control and treatment regions for the cohorts *in utero* prior to, during and after the experiment. All averages are calculated using data aggregated to the region-by-quarter of birth-by-old/young mother-level and weighted by the number of children in each cell. In all there are 1,748 cells including 353,742 children. The first panel of Table 1 reports the mean of the outcome variables for children born in the treatment regions and the control regions. Columns 1-6 report averages for children born in the experimental regions (columns 1-3) and the control regions (columns 4-6). Columns 7-12 report the corresponding characteristics for children of mothers under the age of 21 at the date of birth. The statistics in Table 1 are calculated for the cohorts born during the first two quarters of each year. Table 1 also presents the fathers and mothers ages at the date of birth, the fraction of mothers with post secondary education (measured in 1990), and the average number of children in each cell. From these background characteristics an increasing age trend among mothers may be noted, and also that the number of young mothers decreases over time in both the treatment and the control regions. Looking at the average outcomes, it appears that the children of the young mothers exposed to the experiment (i.e. born in 1968) tend to have a less favorable development in terms of educational and labor market outcomes compared to the other cohorts.

To get a clearer view of the trend in the outcomes of children born around the time of the experiment, Figure 2 plots average years of schooling

completed in 2000 for children born between 1966:Q1 to 1970:Q4 by mothers under the age of 21 in the treatment and control regions. The average years of schooling of the treatment region children conceived just prior to the experiment (born during the second quarter of 1968) deviate clearly from the pattern displayed by the adjacent cohorts and the control region cohorts.

A similar pattern is found in Figure 3 in which the comparison group is now children born in the treatment regions, but with mothers older than 20 at the date of birth. There is no visible change in the educational outcome for children with older mothers, but the dip in years of schooling is still apparent for the young mothers' children. The pattern in the two figures is clearly in line with the police reports suggesting that young people's alcohol consumption increased most during the experiment. The timing also corresponds well with the estimated duration of exposure as presented in Table A1.

Figure 4 plots the average earnings³⁹ at age 32 for the children whose mothers were under the age of 21 on delivery in the control and treatment regions. As in the case of education there is a distinct decrease in relative earnings between treatment and control region children that coincides with the timing of the experiment. In order to get a better picture of where the variation in average earnings stems from Figure 5 delve deeper into the differences in earnings for the most exposed cohort. On the left hand side of Figure 5 the cumulative earnings distribution of men and women born during the second quarter of 1968 is shown. The cumulative earnings distributions suggest that men at the lower end of the distribution seem to have been particularly strongly affected as the distribution is pushed to the left for the exposed cohort. In contrast, the earnings differences between those born in the control and treatment regions earning above the 50th percentile are relatively small. Under the assumption that in the absence of the experiment the treated children would have ended up at the same position of the distribution, the experiment seems to mainly have affected low-SES children.⁴⁰ For comparison, the same distributions are shown on the right hand side of Figure 5 for individuals born one year prior to the experiment. Again, the difference in distribution between the control and treatment regions for this cohort is minimal.

³⁹ The data used in the figure have been trimmed so as to omit individuals with yearly earnings below the 1st percentile (SEK 1400) and above the 99th percentile (SEK 563,700).

⁴⁰ The invariant rank assumption may however be a strong assumption in this context. A survey among young people aged 15-25 conducted in the spring of 1968 revealed a clearly positive correlation between alcohol usage among young women and the father's socio-economic status (see e.g. SOU 1971:77), suggesting that children of more well-off mothers may actually have been those with the highest exposure.

Table 1 Means of background characteristics and outcomes (first two quarters of each year)

	Treated			Control			Treated			Control		
	All mothers			All mothers			Young mothers			Young mothers		
	(I)			(II)			(III)			(IV)		
	Born <1968	Born 1968	Born >1968	Born <1968	Born 1968	Born >1968	Born <1968	Born 1968	Born >1968	Born <1968	Born 1968	Born >1968
Outcomes:												
Education (years)	12.28	12.36	12.52	12.26	12.4	12.50	11.48	11.40	11.55	11.52	11.59	11.49
Fraction high school graduates	0.92	0.93	0.93	0.91	0.93	0.93	0.87	0.85	0.86	0.86	0.88	0.87
Fraction college graduates	0.16	0.16	0.17	0.16	0.16	0.17	0.06	0.05	0.06	0.07	0.07	0.06
Average log (yearly earnings) at age 32	7.20	7.34	7.40	7.21	7.33	7.41	7.09	7.14	7.30	7.14	7.25	7.29
Fraction w. zero earnings (age 32)	0.12	0.10	0.10	0.11	0.09	0.09	0.14	0.14	0.13	0.13	0.10	0.12
Fraction on welfare in 2000	0.04	0.04	0.05	0.04	0.04	0.04	0.09	0.08	0.09	0.06	0.06	0.09
Fraction males	0.51	0.52	0.51	0.51	0.52	0.52	0.50	0.49	0.53	0.51	0.52	0.52
Family characteristics:												
Age of father at delivery	27.1	26.9	27.1	26.9	26.8	27.2	22.5	22.4	22.8	22.6	22.4	22.8
Age of mother at delivery	23.9	24.1	24.4	23.7	24.0	24.4	18.9	18.9	18.9	19.2	18.9	18.9
%Mothers w. Post-secondary education	22	24	29	22	24	29	11	11	11	13	11	10
Average number of children in cells	464	443	430	310	300	279	238	197	158	168	126	103

Note: The table reports weighted averages over cells.

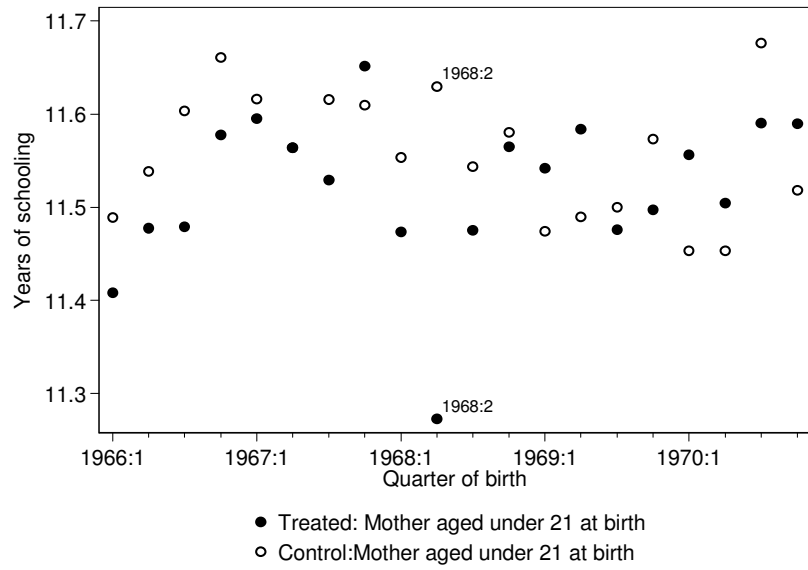


Figure 2 Average years of schooling, treated vs. control.

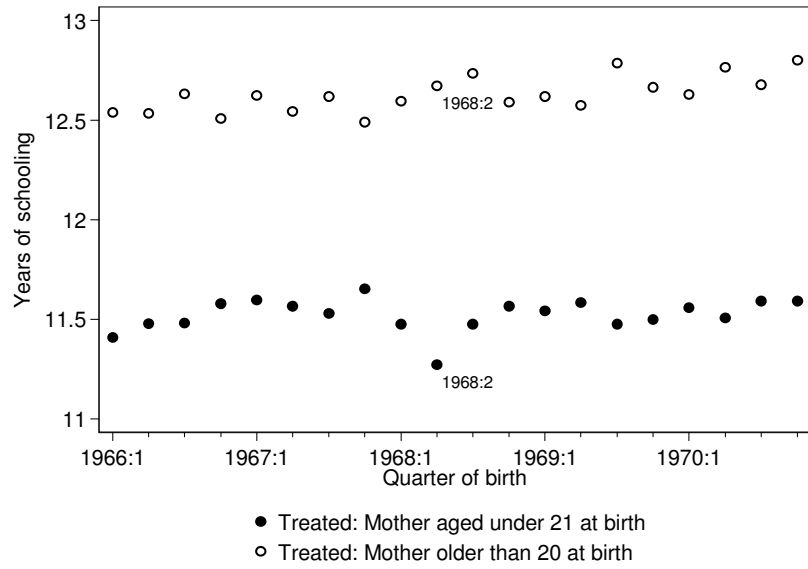


Figure 3 Average years of schooling, young vs. old mother.

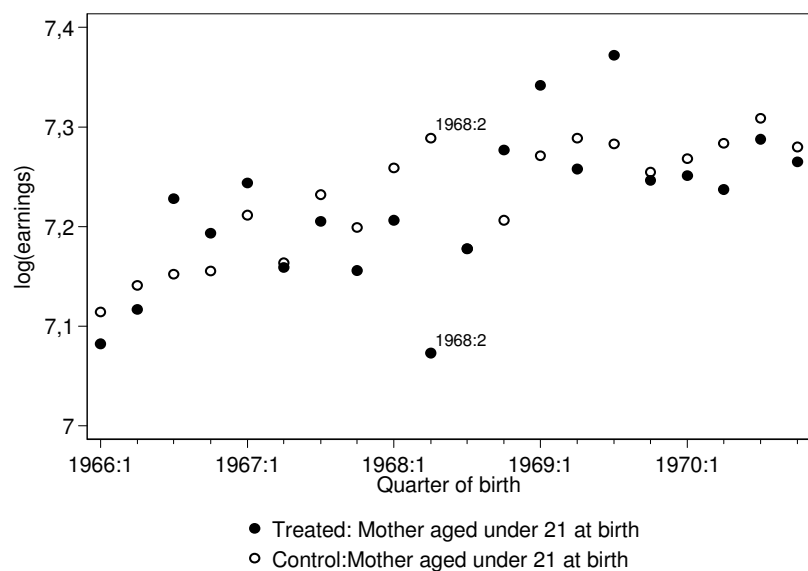


Figure 4 Average $\ln(\text{earnings})$ at age 32, treated vs. control.

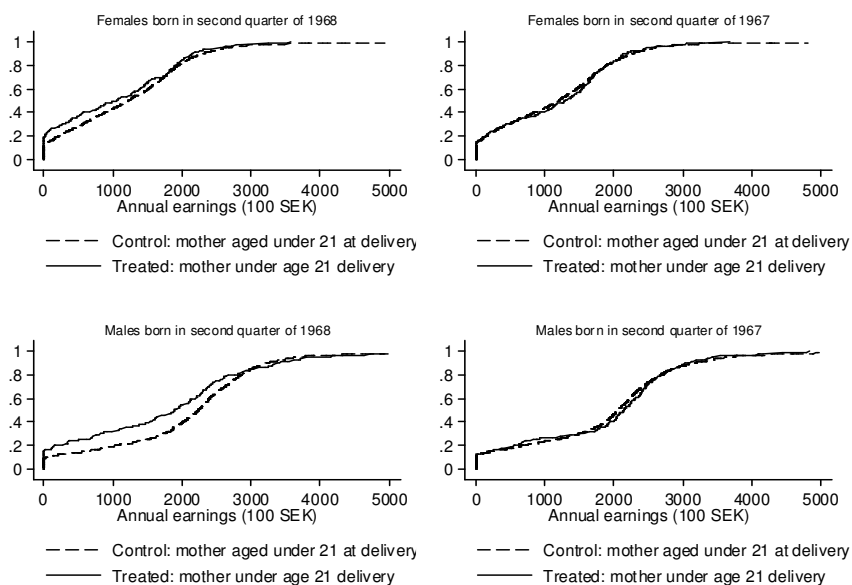


Figure 5 Cumulative earnings distribution at age 32. Left column presents earnings for women (top) and men (bottom) born during the second quarter of 1968. The right column shows the same distributions for children born during the second quarter of 1967 (i.e. before the experiment).

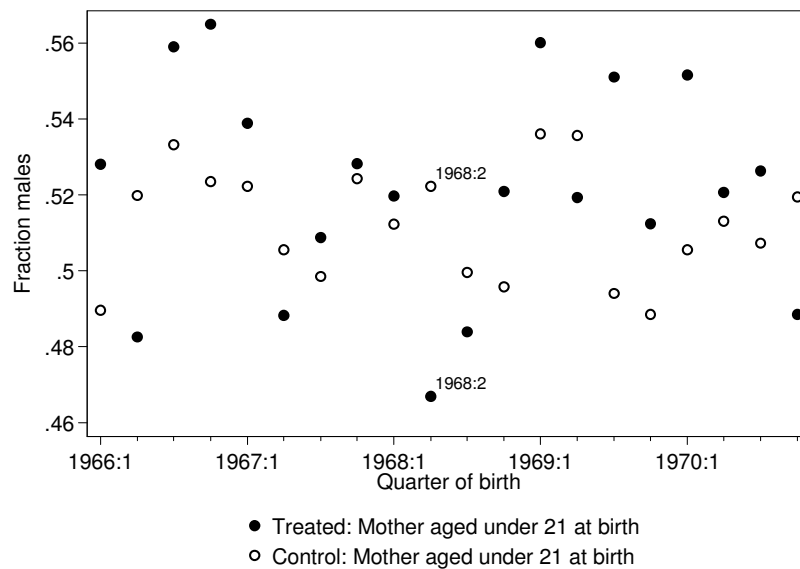


Figure 6 Proportion of males in 2000

Finally, Figure 6 plots the proportion of males in corresponding cohorts. Clearly the variance is higher in this case; but still there is a distinct drop in the proportion males, coinciding with timing of the experiment and the changes in the other outcomes. Previous studies have found that a reduced sex-ratio at birth is indicative of adverse maternal conditions during pregnancy (see e.g. Trivers and Willard, 1973; Lee et al., 1998; Wells, 2000). This finding is explored in more detail below.

4.2 Regression results

The descriptive analysis above does indeed suggest substantial drops in average outcomes, coinciding with *in utero* exposure to the experiment. To gauge more formally to what extent this drop is indeed caused by the experiment, we now turn to the OLS difference-in-difference-in-differences estimates of equation (1).

4.2.1 Baseline estimates

This section reports baseline results from regression analysis based on the specification in equation (1). Panel A, B and C of Table 2 report estimates of β_1 using the average years of schooling, the proportion high school graduates and the proportion with at least 3 years of higher education as the dependent variable, respectively. Columns (1)-(3) in each panel provide the estimates employing the full sample, the male sample, and finally the female sample. Educational attainment is measured in 2000 when the children in the sample were aged between 28 and 36. All regressions are weighted by the number of children in each cell. The reported standard errors are robust with respect to heteroscedasticity.

As seen in Table 2, the impact of the experiment on educational outcomes is substantial. In the full sample, the coefficient suggests that the number of years of schooling is reduced by 0.27 years on average. Among males, this effect is even stronger - males from the cohort *in utero* during the experiment have on average 0.47 fewer years of schooling, and among females this effect is somewhat weaker (0.10 years), and not statistically distinguishable from zero. Turning to the proportion who graduated from high school, it appears that the children in the exposed cohort are about 4 percentage points less likely to have completed high school. Again, this effect is driven by a lower high school completion rate of 10 percent with respect to the mean among males (-0.09/0.9). The proportion of males who has graduated from higher education is also significantly reduced by 3.9 percentage points, and by 2.1 percentage points for females, but imprecisely estimated. The effect on the proportion of males graduating from higher education is even larger than the effects on the high school completion rates, which support the notion that many children who are not obviously affected by prenatal alcohol exposure may still suffer from cognitive deficits. With respect to the mean, exposed males are about 35 percent (-0.039/0.11) less likely to have graduated from higher education.

Table 2 The impact of the experiment on educational attainments

Sample			
A. Dependent variable:	All	Men	Women
	(1)	(2)	(3)
Years of schooling			
<i>In utero</i> (month 1-4)	-0.266*** (0.049)	-0.473*** (0.124)	-0.101 (0.151)
Observations	1350	1350	1350
R-squared	0.98	0.96	0.95
Mean	12.33	12.18	12.49
Sample			
B. Dependent variable:	All	Men	Women
Fraction high school graduates	(1)	(2)	(3)
<i>In utero</i> (month 1-4)	-0.039*** (0.009)	-0.092*** (0.017)	0.015 (0.014)
Observations	1350	1350	1350
R-squared	0.90	0.85	0.82
Mean	0.92	0.91	0.93
Sample			
C. Dependent variable:	All	Men	Women
Fraction graduated from higher education	(1)	(2)	(3)
<i>In utero</i> (month 1-4)	-0.025** (0.012)	-0.039*** (0.013)	-0.021 (0.014)
Observations	1350	1350	1350
R-squared	0.95	0.92	0.92
Mean	0.16	0.14	0.18
Quarter of birth dummies	YES	YES	YES
Region of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES

Notes: Each column and panel represents a separate regression. The dependent variable is years of schooling, fraction with higher education or fraction who have completed high school. The unit of observation is all first born children alive in 2000 either by mothers aged ≥ 21 or below in a given year, quarter and region. “*In utero*(month 1-4)” is a dummy equal to 1 if the child was born by a mother under age 21 and exposed to the experiment while *in utero* from early until late pregnancy (see section 3.1 for details). All regressions include year of birth, quarter of birth, region of birth, mother under age 21 at delivery dummies and a set of interaction terms between these variables (see equation 1). All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. Heteroscedasticity robust standard errors are reported in parenthesis.

Table 3 The impact of the experiment on labor market outcomes

A. Dependent variable:	Sample		
	All (1)	Men (2)	Women (3)
ln(earnings)			
<i>In utero</i> (month 1-4)	-0.241*** (0.053)	-0.228*** (0.081)	-0.177** (0.097)
Observations	1350	1350	1350
R-squared	0.88	0.87	0.79
Mean	7.26	7.57	6.93
B. Dependent variable:	Sample		
	All (1)	Men (2)	Women (3)
Fraction with zero earnings			
<i>In utero</i> (month 1-4)	0.071*** (0.012)	0.069*** (0.017)	0.069*** (0.013)
Observations	1350	1350	1350
R-squared	0.76	0.71	0.67
Mean	0.10	0.09	0.11
C. Dependent variable:	Sample		
	All (1)	Men (2)	Women (3)
Fraction welfare participants			
<i>In utero</i> (month 1-4)	0.036*** (0.009)	0.051*** (0.016)	0.021 (0.021)
Observations	1350	1350	1350
R-squared	0.84	0.74	0.76
Mean	0.042	0.039	0.046
Quarter of birth dummies	YES	YES	YES
Region of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES

Notes: Each column and panel represents a separate regression. The dependent variable is years of schooling, fraction with higher education or fraction who have completed high school. The unit of observation is all first born children alive in 2000 either by mothers aged ≥ 21 or below in a given year, quarter and region. “*In utero*(month 1-4)” is a dummy equal to 1 if the child was born by a mother under age 21 and exposed to the experiment while *in utero* from early until late pregnancy (see section 3.1 for details). All regressions include year of birth, quarter of birth, region of birth, mother under age 21 at delivery dummies and a set of interaction terms between these variables (see equation 1). All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. Heteroscedasticity robust standard errors are reported in parenthesis.

Moving on to the impact on labor market outcomes presented in Table 3, we see that males and females in this case are similarly affected. On average the exposed cohort has close to 24 percent lower earnings at the age of 32. Again, males seem to have been somewhat more strongly affected than females. However, the assumption that women's earnings at the age of 32 are an accurate measure of their permanent earnings is questionable. Böhlmark and Lindqvist (2006) estimates of life-cycle biases shows that, in the case of Sweden, the ideal solution for women would be to use earnings after the age of 40 in order to get a good proxy for permanent earnings.

Panel B in Table 3 presents the results of a regression using the fraction with zero earnings as the dependent variable. In this case, the experiment increased the risk of having no labor income at all at age 32 for both men and women with around 7 percentage points. The last panel in Table 3 reveals that the proportion on welfare among the exposed males is 5 percentage points higher in the exposed cohort. The proportion of females on welfare is also higher, but the point estimate is not statistically different from zero.

To summarize, for education and labor market outcomes the estimated impact of the experiment is considerable. In the case of education, the effects are comparable with the estimates for other types of insults *in utero* on subsequent educational attainments (see e.g. Almond et al., 2007; Barreca, forthcoming). Moreover, as suggested by Figure 5 the greatest impact on earnings is found at the lower end of the earnings distribution. In a recent study Heathcoate, Perri and Violante (2009) show that changes in earnings in the bottom of the earnings distribution to a much larger extent reflect changes in hours worked rather than changes in wages. The opposite is true for changes in earnings in the top of the distribution. This indicates that the relatively large effects of the experiment on average annual earnings likely reflect reductions in hours worked rather than low wages. Unfortunately I do not have access to data on wages, which is potentially a better measure of skills. Moreover, while the natural log transformation of the earnings simplifies interpretation, it also emphasizes differences at the lower end of the earnings distribution. Running the same regression on the non-logged earnings (still excluding the zeros) reduces the point estimate significantly to around 15 percent, which is still a sizeable effect.

4.2.2 *Differential effects by socioeconomic status*

A higher level of parental resources may potentially mitigate some of the negative effects of health shock early in life (Currie and Hyson, 1999; Case et al., 2002). Panel A in Table 4 reports estimates for the sample of children with mothers with at least one semester of post-secondary education. The point estimates for education and labor market outcomes tend to be larger for children of mothers without a higher education and are more precisely estimated. Panel B in Table 4 presents the results from the same specifications, but for mother with above or below the median income level in 1990. The pattern is similar in this case with smaller estimated effects for higher income-level mothers.

These set of results indicates that parental resources may mitigate the effects of poor health in childhood on outcomes later in life. However, while suggestive these results should be interpreted with care as the standard errors are in some cases fairly large. Moreover, the highest educational level and earnings of the mother (both measured in 1990), might potentially be endogenous with respect to the health of the child (see e.g. Powers, 2001). Finally, it is difficult to rule out that the heterogeneous effects are due to differential consumption responses to the increase in availability i.e. that low SES mothers drink more. However, survey evidence from the same period indicate that, if anything, young people from higher SES backgrounds drink more and are less likely to completely abstain from alcohol use (SOU 1971:77).⁴¹

⁴¹ To further test for potential differences in consumption between low SES groups and high SES groups I also estimated differences in the impact on the sex-ratio (see discussion in section 4.2.3) for the two different educational groups. After splitting the sample into high and low SES the estimated effects on the sex-ratio is larger for the high SES group, indicating higher consumption, but the precision is not good enough to draw conclusions regarding which group that responded most to the policy.

Table 4 The impact of the experiment by maternal education and earnings: labor market and educational outcomes

Panel A.	Dependent variables											
	Years of schooling		High school graduates		Higher education		Earnings		Zero earnings		Welfare	
	High	Low	High	Low	High	Low	High	Low	High	Low	High	Low
Education of mother (1990)												
<i>In utero</i> (month 1-4)	-0.347*	-0.200***	-0.020	-0.038***	-0.016	-0.027**	-0.218	-0.235***	0.010	0.078***	0.058	0.025**
	(0.182)	(0.063)	(0.044)	(0.010)	(0.090)	(0.010)	(0.185)	(0.046)	(0.064)	(0.008)	(0.038)	(0.013)
# observations	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350
Panel B.	Years of schooling		High school graduates		Higher education		Earnings		Zero earnings		Welfare	
Mothers labor earnings (1990)	Above median	Below median	Above median	Below median	Above median	Below median	Above median	Below median	Above median	Below median	Above median	Below median
<i>In utero</i> (month 1-4)	-0.071	-0.360***	-0.028	-0.045*	-0.022	-0.021*	-0.226	-0.248	0.042***	0.092***	0.017*	0.047***
	0.103	(0.083)	(0.028)	(0.025)	(0.027)	(0.011)	(0.216)	(0.187)	(0.015)	(0.020)	(0.009)	(0.014)
# observations	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350

Notes: Each reported column represents a separate regression. The outcomes are measured within each region of birth/year of birth/quarter of birth/mom<age 21 at delivery cell. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. Heteroscedasticity robust standard errors are reported in parenthesis. High education of mother implies post-secondary education, median earnings calculation is based on maternal earnings distribution in 1990.

4.2.3 Further results and robustness checks

Health related outcomes

The pattern in the tables above is clear. The children exposed to the policy experiment *in utero* seem to have significantly worse adult outcomes than the surrounding cohorts. Notably, males seem to have been particularly affected. Table 5 provides some guidance as to why males could be expected to be more strongly affected by an increased prenatal exposure to alcohol than females. The table reports the estimated effects of exposure on three health-related outcomes that yield some insights into the underlying mecha-

Table 5 The impact of the experiment on health related outcomes

	Dependent variables:				
	Fraction of males	Month of birth	In cohort size	Month of birth	In cohort size
	(1)	(2)	(3)	(4)	(5)
	All	Men	Men	Women	Women
<i>In utero</i>	-0.072*** (0.024)	-0.240** (0.122)	-0.166*** (0.055)	0.042 (0.146)	0.130* (0.072)
Year/Quarter dummies	YES	YES (YEAR)	YES	YES (YEAR)	YES
R.O.B dummies	YES	YES	YES	YES	YES
Mom age<21 dummy	YES	YES	YES	YES	YES
Observations	1350	342	1350	342	1350
R-squared	0.56	0.65	0.98	0.63	0.98
Mean(not logs)	0.515	4.00	125	4.00	118

Notes: Each column and panel represents a separate regression. Except for when the dependent variable is “month of birth” the outcomes are measured within each region of birth/year of birth/quarter of birth/mom<age 21 at delivery cell. In the “month of birth” case instead the analysis each cell refers to region/year of birth/mother under age 21 cell averages. Furthermore, in this case only those born between January through July is retained. “*In utero*” is a dummy equal to 1 if the child was born by a mother under age 21 and exposed to the experiment while *in utero* (see text for details). All regressions include year of birth, quarter of birth, region of birth, mother under age 21 at delivery dummies and the corresponding interaction terms. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable, except for the cohort size outcome. Heteroscedasticity robust standard errors are reported in parenthesis.

nism explaining the differences in outcomes between males and females.⁴² Column (1) presents the point estimate from a regression using the baseline model from equation (1) on the full sample with the proportion of males in each cell as the dependent variable. The coefficient is statistically significant and suggests that the proportion of males is 7.2 percentage points lower in the exposed cohort. Columns (2) and (4) present the results of a regression in which the dependent variable is the average month of birth for children born between January and July in each year, for males and females separately. While the coefficient reveals that the exposed males were born on average 1 week earlier (0.24 months), the experiment does not seem to have had any similar effect on the average birth month of females. Similarly, the cohort of men born in the wake of the experiment is significantly smaller, while no such effect is recognized for females (columns 3 and 5).

These results are in line with several medical and biological studies suggesting that male are more sensitive to adverse conditions in early life than females (see e.g. Lee et al. 1998; Wells, 2000). Moreover, these estimates are consistent with results first found by Little et al. (1986) who, after controlling for a number of maternal background characteristics, found “a greater vulnerability of the male to alcohol exposure in the late first and early second trimester...” as measured by birth weight.⁴³

Spill-over effects to neighboring regions

The instigators of the experiment suggested that at least some of the increased sales of strong beer were due to cross-border shopping by individuals from neighboring regions. Next I examine to what extent such cross-border shopping also resulted in adverse outcomes for the children born in these regions. Remember that in the previous regressions these children were excluded from the sample. Table 6 reports coefficients from the same specifications as in the tables above but now the “in utero” dummy is equal to 1 for the cohort of children born between April and July 1968 by

⁴² The ideal data for such as exercise would be the medical birth register with its highly detailed data on birth weight, prematurity etc. Unfortunately the Swedish medical birth register started to get digitalized in 1973.

⁴³ Using a sample of non-smoking, non-alcoholic women Little et al. related average daily consumption both in the week before pregnancy recognition (week 6 on average) and in the week prior to the first prenatal visit (between week 8 through 16, mean: 11.2) to birth weight. The differing effects between males and females on birth weight are particularly strong in the later case. Interestingly, the fraction of male births in their sample is also strongly negatively correlated with consumption during the same period of gestation. Furthermore, the results are consistent with differences in sensitivity to binge alcohol exposure displayed among male and female rats found by Goodlett and Peterson (1995). Moreover, these results also provide evidence on one mechanism explaining the results Balsa (2008) finds. Using the NLSY79 Balsa show that having a problem-drinking parent is associated with longer periods out of the labor force, lengthier unemployment, and lower wages, in particular for males.

mothers under the age of 21 in one of the five regions neighboring the experiment area.⁴⁴

The results from this exercise suggest that cross-border shopping did not affect the outcomes of the children in the neighboring regions to any major extent. None of the coefficients is significantly different from zero at any conventional level of significance. Given that the neighboring regions and the treatment regions are highly interdependent and constitute a local labor market, this exercise also strengthens the case for the main identification strategy.

⁴⁴ The experiment-region children are excluded from these regressions.

Table 6 The impact of the experiment on neighboring regions: labor market, educational and health outcomes

Dependent variables: Labor and education												
A.	Years of schooling		High school graduates		Higher education		Earnings		Zero earnings		Welfare	
	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
Sample												
<i>In utero</i>	-0.106	-0.140	0.006	-0.026	-0.021	0.007	0.040	0.101	0.016	0.029	0.003	0.017
	(0.135)	(0.088)	(0.027)	(0.022)	(0.014)	(0.023)	(0.082)	(0.092)	(0.019)	(0.018)	(0.019)	(0.016)
# of obs.	1598	1598	1598	1598	1598	1598	1598	1598	1598	1598	1598	1598
Dependent variables: Health												
B.	Fraction males	Month of birth		ln(cohort size)								
	ALL	Men	Women	Men	Women							
Sample												
<i>In utero</i>	-0.006	0.119	-0.037	0.022	0.037							
	(0.025)	(0.085)	(0.123)	(0.097)	(0.074)							
# of obs.	1598	413	408	1598	1598							

Notes: Each column and panel represents a separate regression. Except for when the dependent variable is “month of birth” the outcomes are measured within each region of birth/year of birth/quarter of birth/mom<age 21 at delivery cell. In the “month of birth” case instead the analysis each cell refers to region/year of birth/mother under age 21 cell averages. Furthermore, in this case only those born between January and July are retained. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable, except for the cohort size outcome case. Heteroscedasticity robust standard errors are reported in parenthesis.

The timing of exposure

Table 7 examines the impact of the experiment on those who were between 1 to 12 months (panel A), and 13 to 24 months old (panel B) at the start of the experiment. Besides including dummies for the new cohorts of interest the original “in utero” dummy is also included in order to examine to what extent the baseline results are sensitive to the change in specification. Interestingly the experiment does not seem to have had an effect on the outcomes of children born just prior to its implementation. This finding suggests that it is in fact prenatal exposure to the policy rather than an increased incidence of detrimental postnatal events that drives the main results. Moreover, the augmented model yields qualitatively similar results as the baseline model which is reassuring.

Table 8 reports the impact of the experiment on children of mothers in late pregnancy (months 5-9) at the start of the experiment vs. the original exposure cohort. Only the probability of having graduated from high school seems to have been significantly affected among those exposed late in pregnancy, whereas the estimated impact on the original cohort are virtually identical to the baseline results. One might be tempted to interpret the results of this exercise as evidence that alcohol exposure during the first and second trimester is more detrimental than exposure later on. However, these findings could also merely reflect heterogeneous consumption responses to the increase in alcohol availability between mothers in early and late pregnancy. Unfortunately, the estimation strategy employed does not allow for a distinction between these two mechanisms.

In order to attain a clearer picture of the dynamics of the impact of the experiment, Table 9 reports estimates from regressions using monthly rather than quarterly data. Specifically I now let the treatment window glide over the cohorts potentially affected by the experiment. Hence, rather than just looking at those with the maximum amount of *in utero* exposure to the experiment, I now start with those born between November 1967 and February 1968, continuing with December 1967 through March 1968, up to those born between September 1968 and December 1968. The treatment window used in the main analysis, April through July 1968, is highlighted in bold. The treatment windows to the left of the vertical dashed line (columns I-VI) only contain cohorts estimated to have been conceived before the experiment started. To the right of the dashed line, at least some of the children in the treated cohorts were conceived during the course of the experiment. The parameter estimates reported follow a clear pattern. While there are no significant differences for the children with the least amount of exposure (reported in column I and II), there is an increasingly negative trend in outcomes as the treatment window is rolled towards the most expos-

Table 7 The impact of the experiment on children aged 1-12 months and 13-24 months at the start of the experiment: Labor market and educational outcomes

		Dependent variables:				
Panel A.	Years of schooling	High school graduates	Higher education	Earnings	Zero earnings	Welfare
Age at start of Experiment:	All	All	All	All	All	All
I(1-12 months)	-0.034 (0.045)	-0.0003 (0.009)	0.0004 (0.010)	0.041 (0.034)	0.0004 (0.010)	-0.006 (0.011)
<i>In utero</i> (month 1-4)	-0.271*** (0.050)	-0.039*** (0.009)	-0.025** (0.012)	-0.240*** (0.053)	0.071*** (0.0122)	0.035*** (0.009)
Number of observations	1350	1350	1350	1350	1350	1350
		Dependent variables:				
Panel B.	Years of schooling	High school graduates	Higher education	Earnings	Zero earnings	Welfare
Age at start of Experiment:	All	All	All	All	All	All
I(13-24 months)	-0.056 (0.071)	-0.002 (0.016)	-0.007 (0.009)	-0.014 (0.030)	0.002 (0.012)	0.002 (0.006)
<i>In utero</i> (month 1-4)	-0.263*** (0.053)	-0.039*** (0.009)	-0.024** (0.012)	-0.240*** (0.046)	0.071*** (0.012)	0.036*** (0.009)
Number of observations	1350	1350	1350	1350	1350	1350

Notes: Each column and panel (A & B) represents a separate regression. The outcomes are measured within each region of birth/year of birth/quarter of birth/mom<age 21 at delivery cell. Robust standard errors in parenthesis. The *I(1-12)* take the value 1 if the child was born in 1966Q4-1967Q3 and zero otherwise. The “*In utero*” dummy is equal to 1 if the child was born between April and July 1968. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. Heteroscedasticity robust standard errors are reported in parenthesis.

Table 8 The impact of the experiment on children of mothers in depending on gestational age.
Late pregnancy (month 5-9) vs. early pregnancy (month 1-4) at start of experiment: Labor market and educational outcomes

	Dependent variables: Labor and education					
	Years of schooling	High school graduates	Higher education	Earnings	Zero earnings	Welfare
Gestational age at start of experiment:	All	All	All	All	All	All
<i>In utero</i> (month 5-9)	0.036 (0.097)	-0.019** (0.009)	0.014 (0.021)	0.032 (0.075)	-0.005 (0.016)	0.004 (0.006)
<i>In utero</i> (month 1-4)	-0.256*** (0.063)	-0.043*** (0.010)	-0.023* (0.013)	-0.242*** (0.053)	0.070*** (0.013)	0.033*** (0.010)
Number of observations	1350	1350	1350	1350	1350	1350

Notes: Each column represents a separate regression. The outcomes are measured within each region of birth/year of birth/quarter of birth/mom<age 21 at delivery cell. “*In utero* (month 5-9)” is equal to 1 if the child the child was born between November 1967 and March 1968. “*In utero* (month 1-4)” refers as above to the original treatment cohort, those born between April and July 1968. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. Heteroscedasticity robust standard errors are reported in parenthesis.

Table 9 The impact of the experiment on children depending on gestational age at start of experiment (monthly data)

Dependent variables: Educational, labor market and health-related outcomes											
	(I)	(II)	(III)	(IV)	(V)	(VI)	(VII)	(VIII)	(IX)	(X)	(XI)
Period of Birth	Nov-Feb	Dec-Mar	Jan-Apr	Feb-May	Mar-Jun	Apr-Jul	May-Aug	Jun-Sept	Jul-Oct	Aug-Nov	Sept-Dec
Est. gestational age (months) in Nov. 1967	(6-9)	(5-8)	(4-7)	(3-6)	(2-5)	(1-4)	(n.c.-3)	(n.c.-2)	(n.c.-1)	No one conceived	No one conceived
<i>Outcome:</i>											
Yrs. of Schooling	0.065 (0.134)	-0.063 (0.079)	-0.173** (0.074)	-0.224*** (0.075)	-0.240*** (0.082)	-0.266*** (0.083)	-0.300*** (0.095)	-0.220** (0.112)	-0.110 (0.108)	-0.130 (0.101)	0.043 (0.097)
High School grad.	-0.002 (0.015)	-0.014 (0.017)	-0.030* (0.016)	-0.026* (0.015)	-0.044*** (0.012)	-0.037** (0.016)	-0.036* (0.020)	-0.019 (0.022)	-0.013 (0.019)	-0.009 (0.017)	0.007 (0.016)
University grad.	0.012 (0.025)	-0.011 (0.017)	-0.017 (0.016)	-0.018 (0.015)	-0.015 (0.016)	-0.023** (0.012)	-0.036*** (0.013)	-0.030* (0.018)	-0.010 (0.020)	-0.017 (0.020)	0.001 (0.020)
Labor earnings	-0.012 (0.043)	0.026 (0.086)	-0.035 (0.102)	-0.163 (0.119)	-0.204* (0.118)	-0.290*** (0.092)	-0.203* (0.109)	-0.081 (0.068)	-0.040 (0.072)	0.014 (0.076)	0.011 (0.079)
Zero earnings	-0.008 (0.021)	0.016 (0.018)	0.051* (0.029)	0.071*** (0.024)	0.076*** (0.024)	0.072*** (0.026)	0.034** (0.017)	0.011 (0.023)	-0.008 (0.021)	-0.016 (0.018)	-0.036*** (0.013)
Welfare dep.	-0.001 (0.016)	0.005 (0.013)	0.017 (0.018)	0.012 (0.017)	0.022 (0.016)	0.034** (0.015)	0.017* (0.010)	0.013 (0.013)	0.007 (0.013)	0.003 (0.013)	0.002 (0.013)
Fraction males	-0.002 (0.028)	0.004 (0.024)	-0.008 (0.028)	-0.058** (0.027)	-0.064** (0.028)	-0.073** (0.029)	-0.039 (0.033)	-0.025 (0.031)	-0.015 (0.040)	-0.001 (0.041)	0.003 (0.043)
# of obs	4086	4086	4086	4086	4086	4086	4086	4086	4086	4086	4086

Notes: Each column and panel represents a separate regression using the model in equation (1). The outcomes are averages/fractions within each region of birth/month of birth/mom<age 21 at delivery cell. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. Heteroscedasticity robust standard errors are reported in parenthesis. The estimates from using the original treatment window are reported in bold (column VI).

ed cohorts. For the educational outcomes, the strongest negative effect is reached somewhere between March and August 1968 (columns V-VII), as is the case for earnings.

In the case of years of education and earnings, I have performed the same analysis for each cohort born from three years before the main cohort until three years after. The parameter estimates from these regressions is summarized in Figure 7. The estimates reported between the two vertical dashed lines contain at least one cohort exposed to the experiment *in utero*. Firstly, from this figure it can clearly be seen that the timing in the dip in relative outcomes among the highest exposed cohorts is unusually large and fits very well with the number of weeks of exposure. Secondly, while there are also dips for other cohorts for each one of the outcomes, during the experiment both the estimated impact for both educational outcomes and earnings move in concert unusually well. Thirdly, interestingly in the case of education the estimates suggest that the children conceived at the end of the experiment period (i.e. born in the spring of 1969) have a relatively higher level of educational attainments ($p < 0.05$). This effect could in part be due to a positive effect of the experiment on parental composition among the children conceived during the policy experiment. As discussed above previous research has shown that that alcohol consumption increases risky behavior among young people. Hence, if the higher alcohol consumption increased fertility relatively more among high ability parents this may explain the relative increase in educational attainments among the cohort conceived at the end of the experiment period.⁴⁵

To be able to test this hypothesis directly, one would ideally like to have some parental quality indicator measured prior to birth of the child. As such a measure is not available, I look at whether the fraction of children born by a mother with post secondary education (measured in 1990) is higher among those conceived during last part of the experiment.⁴⁶ This exercise indeed indicates that parental composition improved significantly for those children conceived during the later part of the experiment as the fraction of children born by educated mothers by 3.3 percentage points (mean=0.13, $p < 0.05$).⁴⁷

⁴⁵ In the absence of legalized abortions (not freely available until 1975), there are several potential reasons for such effects to occur. One reason is that highly skilled women are assumingly less likely to become pregnant at an early age, as the cost of having a child is higher in terms of lost future earnings relative to low skill women. Hence, increased alcohol availability may have a larger *relative* affect on the pregnancy rate among highly skilled women than low-skilled women.

⁴⁶ Note that for these children conception was potentially affected by the experiment, although the time *in utero* during the experiment was short.

⁴⁷ This effect is driven by a 35 percent increase (est. 0.35, std.err 0.15) in the number of children born by a high school educated mother rather than a decrease in the number children born by a less educated mother. The estimates are attained by running the baseline regression with the fraction of mothers with a high school diploma as the dependent variable. The last

An additional finding that indirectly supports the idea that the relative increase in educational attainments are caused by the experiment is that the positive effect on education dies out directly after the last “treated” cohort leaves the treatment window (the cohorts just after the right vertical dashed lined in Figure 9). Finally, as we saw in table 2 while there was a substantial difference in the impact on educational attainments for women and men in the main exposure group (presumably from potential sex differences in susceptibility to damage *in utero*), for the cohorts born in the spring of 1969 the years of schooling point estimates are virtually identical between males (est: 0.268 std.err: 0.159) and females (est: 0.269 std.err: 0.126). These two sharply contrasting patterns suggest that the improvement in years of schooling outcomes for those cohorts conceived towards the end of the policy experiment more likely are due to social causes rather than biological causes.

The pattern in Figure 9 furthermore suggests that in order to identify the effects of a given alcohol policy intervention on young peoples consumption (and their children’s outcomes), it seems crucial to investigate *who* is actually affected by the policy, i.e. to what extent parental composition and fertility rates are effected. Neglecting such effects may potentially *underestimate* the true effect of the policy. However, in the present case for the cohorts were direct effects on conception rates can be ruled out (i.e. for those conceived before the experiment started), increased alcohol exposure does indeed seem to have significant and economically important effects on adult outcomes.

cohort in which all children were conceived during the experiment (children born between January and April 1969) is used as the treatment group and I also include a dummy for children born in the same months of 1968 in the specification. For the cohort size outcomes separate regressions are estimated for children born by a mother with/without post-secondary education.

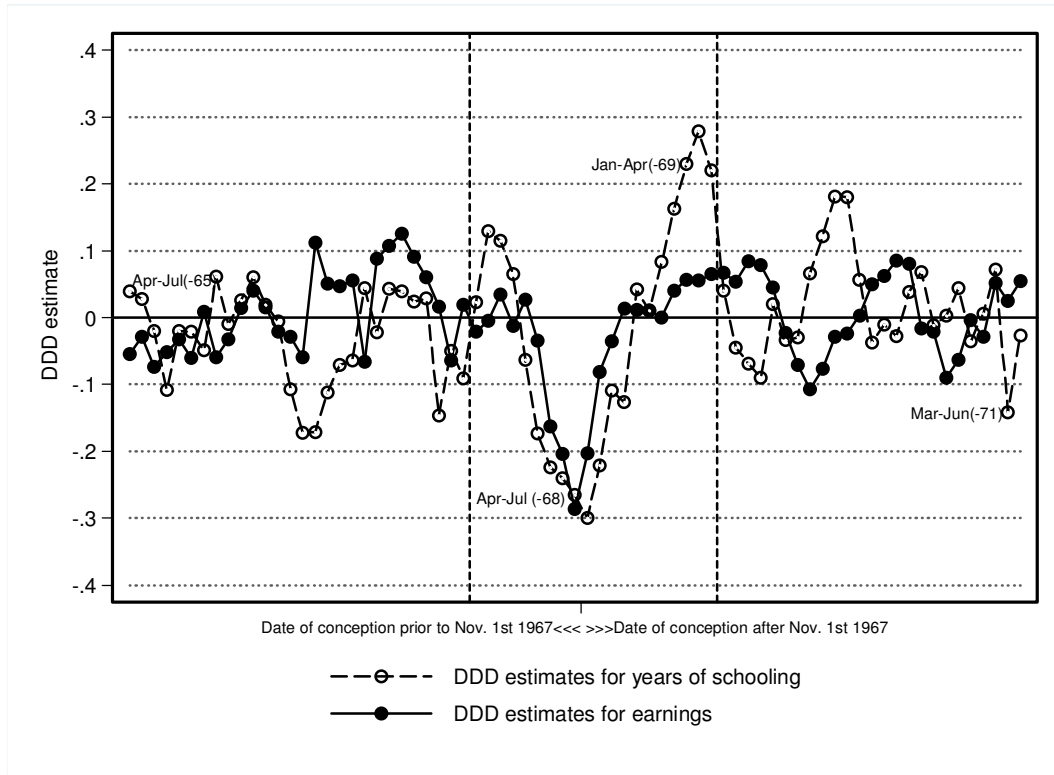


Figure 7 DDD estimates for years of schooling and earning

Sibling fixed effects estimates

As a final check to investigate if the results are due to changes in unobserved parental characteristics coinciding with the timing of the experiment, I have also estimated a maternal fixed effects model in which the outcomes of children in the baseline exposure group are compared to the outcomes of their unexposed siblings. That is, I keep the children belonging to the main exposure group who have a sibling who was also born in the between 1964 and 1972 window. This sample contains around 2,000 sibling pairs. I then re-estimate the baseline model, but now also add a maternal specific parameter which controls for all time-invariant characteristics shared by the siblings. The results from this model are presented in Table 10.

Table 10 Sibling fixed effects estimates

	Dependent variables: Labor and education					
	Years of schooling	High school graduates	Higher education	Earnings	Zero earnings	Welfare
	ALL	ALL	ALL	ALL	ALL	ALL
Exposed sibling	-0.392*** (0.137)	-0.044 (0.032)	-0.037** (0.019)	-0.151 (0.121)	0.063** (0.031)	-0.013 (0.025)
<i>R</i> -squared	0.64	0.62	0.64	0.58	0.56	0.57
Number of siblings	4,428	4,428	4,428	4,428	4,428	4,428

Notes: The table reports sibling fixed effects estimates where the exposure variable is equal to 1 if one of the siblings were exposed to the experiment in utero and born by a mother young than 21. The control variables are the maternal fixed effects, month of birth fixed effects (year*month), region of birth fixed effects and an indicator variable for if the mother was aged under 21 at delivery.

The estimated impact on adult outcomes from the maternal fixed effects model is highly similar to the estimates provided by the baseline model. The adult outcomes of the sibling who was exposed to the experiment in utero in general are considerably worse than the outcomes of the sibling who was not exposed. With the exception of welfare dependency the effects are, in general, even stronger suggesting again that it is not family composition that is driving the main results. This exercise provides strong support for the validity of the main identification strategy.

5 Summary and conclusions

I investigate the long run effects of *in utero* exposure to a temporary “liberalization” of alcohol sales following an alcohol policy experiment in two Swedish regions in the late 1960s. Young people under the age of 21 experienced the largest increase in alcohol availability during the experiment, and according to reports increased their alcohol consumption most. In line with these reports I find that the cohort of children exposed to the experiment *in utero* and born by mothers under the age of 21 has significantly reduced earnings, higher welfare dependency rates, and lower educational attainments as adults in comparison with the surrounding cohorts.

This is the first study applying a quasi-experimental estimation strategy to identify the effects of maternal alcohol consumption during pregnancy on the child’s long-term outcomes. Importantly the analysis allows me to rule out one of the most important alternative explanation behind the correlation between maternal alcohol consumption and children’s development; the potential effects of high consumption on changes in composition of births (i.e. unplanned pregnancies). The results provide compelling evidence on the effects if poor prenatal conditions on adult outcomes.

This study also provides suggestive evidence of an overlooked and potentially important mechanism behind teenage motherhood and children’s outcomes.⁴⁸ Given the findings in this study, and the survey evidence suggesting that about 90% of the alcohol consumed by youths under the age of 21 in the United States is in the form of binge drinks (OJJDP, 2001), identifying effective policy tools to reduce binge drinking among young people may not only improve the health of the individual, but potentially also the outcomes of children born by young mothers.⁴⁹ ⁵⁰ Finally, the

⁴⁸ See e.g. Levine et al. (2001), Francesconi (2007), Hunt (2006) for evidence on the effect of teenage childbearing on offspring outcomes.

⁴⁹ Tsai et al. (2007) use survey data to estimate the prevalence of binge drinking among women of child bearing age (18-44) in the US. In 2003 an estimated 7.2 million women (13 %) in these age categories engaged in binge drinking. In the early 1990s it was about 10 %. While binge drinking decreased among youths up until the mid 1990s there are now signs of a reverse in this trend (Serdula et al., 2004).

⁵⁰ Carpenter et al. (2007) use data from 1976 through 2003 to estimate the impact of a variety of policy measures such as minimum legal drinking age laws, “zero tolerance” under age drunk driving laws and beer taxes on alcohol use among youths. They find that MLDA seems to have had significantly reduced alcohol consumption among high school seniors. Carpenter and Dobkin (2009) use a RD design to identify the effect of the MLDA alcohol consumption

differences in the impact on long-term outcomes found for boys and girls clearly calls for future research investigating if other prenatal conditions also induce similar types of sex-specific interactions effects.

on mortality and suggest that public policy interventions to reduce youth drinking can have substantial direct public health benefits.

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Appendix A: Estimated exposure

Table A 1 Estimated prenatal exposure to the experiment

Month of birth	Est. date of conception [†]	Est. gestational age at start of experiment (month)	Min./Max. number of weeks in utero during experiment		Trimester under exposure:	Experiment may have affected conception rate?
Before Nov. -67	Before Feb. 1967	born	0	0	-	NO
Nov. -67	Feb. 1967	8-9	0	4	3	NO
Dec. -67	Mar. 1967	7-8	4	8	3	NO
Jan. -68	Apr. 1967	6-7	8	12	3	NO
Feb. -68	May 1967	5-6	12	16	2, 3	NO
Mar. -68	June 1967	4-5	16	20	2, 3	NO
April -68	July 1967	3-4	20	24	2, 3	NO
May -68	Aug. 1967	2-3	24	28	1, 2, 3	NO
June -68	Sep. 1967	1-2	28	32	1, 2, 3	NO
July -68	Oct. 1967	0-1	32	34	1, 2, 3	NO
Aug. -68	Nov. 1967	-	30	34	1, 2, 3	YES
Sept. -68	Dec. 1967	-	26	30	1, 2, 3	YES
Oct. -68	Jan. 1968	-	22	26	1, 2, 3	YES
Nov. -68	Feb. 1968	-	18	22	1, 2	YES
Dec. -68	Mar. 1968	-	14	18	1, 2	YES
Jan. -69	Apr. 1968	-	10	14	1, 2	YES
Feb. -69	May 1968	-	6	10	1	YES
Mar. -69	June 1968	-	2	6	1	YES
Apr. -69	July 1968	-	0	2	1	YES
After Apr. -69	After July 1968	-	0	0	-	NO

[†]These estimates all assume that conception occurred 9 months prior to birth. Experiment started on November 1st 1967 and ended on July 14th 1968. The cohorts highlighted in bold are those defined as treated in the main analysis.

