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Does a pint a day affect your childs pay?
The effect of prenatal alcohol exposure on adult outcomes

J Peter Nilsson

WORKING PAPER 2008:4

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Postal address: P O Box 513, 751 20 Uppsala
Visiting address: Kyrkogårdsgatan 6, Uppsala
Phone: +46 18 471 70 70
Fax: +46 18 471 70 71
ifau@ifau.uu.se
www.ifau.se

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ISSN 1651-1166

Does a pint a day affect your child's pay?
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by

J Peter Nilsson[♠]

March 11th, 2008

Abstract

This paper utilizes a Swedish alcohol policy experiment conducted in the late 1960s to identify the impact of prenatal alcohol exposure on educational attainments and labor market outcomes. The experiment started in November 1967 and was prematurely discontinued in July 1968 due to a sharp increase in alcohol consumption in the experimental regions, particularly among youths. Using a difference-in-difference-in-differences strategy we find that around age 30 the cohort in utero during the experiment have substantially reduced educational attainments, lower earnings and higher welfare dependency rates compared to the surrounding cohorts. The results indicate that investments in early-life health may have far reaching effects on economic outcomes later in life.

Keywords: Alcohol policy, infant health, education, earnings
JEL-codes: I12, I18, J24

[♠] The author gratefully acknowledges financial support from the Swedish council for working life and social research (FAS) and valuable suggestions from Douglas Almond, Matz Dahlberg, Lena Edlund, Peter Fredriksson, Erik Grönqvist, Hans Grönqvist, Lena Hensvik, Patrik Hesselius, Per Johansson and seminar participants in Uppsala, Stockholm, at STAKES, the SFI workshop on health and human capital and the Marie Curie Microdata RTN meeting in Paris. Thomas Olsson provided excellent research assistance. Jakob Sandström compiled the data superbly. The author is solely responsible for any errors. Correspond via peter.nilsson@ifau.uu.se.

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1 Introduction

Prenatal exposure to alcohol is today regarded as one of the main preventable causes of mental retardation (Abel and Sokol, 1987; West and Blake, 2005).[1] Medical and epidemiological research has since the early 1970s collected a considerable body of evidence supportive of a negative association between prenatal alcohol exposure and children's health. The range of damage include mild and subtle changes, such as slight learning difficulties or physical abnormality, through full-blown Fetal Alcohol Syndrome (FAS) including severe learning disabilities, growth deficiencies, abnormal facial features, and central nervous system disorders. While the short run effects of maternal consumption of alcohol during pregnancy on child health are relatively well covered, the extent of the long run consequences is not fully known. Evaluating the effects of prenatal exposure to alcohol, particularly in the long run, is however complex. In particular, unobserved characteristics directly related both to the child's outcomes and maternal alcohol consumption, e.g. poverty or mental health, makes the interpretation of nonexperimental estimates difficult.[2]

This paper attempts to isolate the causal relationship between prenatal alcohol exposure and adult outcomes. To do this we investigate the impact of a Swedish alcohol policy experiment on the educational and labor market outcomes of the children in utero during the experiment. During the experiment alcohol availability in two treatment regions (jointly containing 12 % of the Swedish population) increased sharply as regular grocery stores were allowed to market strong beer[3]. Prior to and after the experiment off-premise sales of strong beer, wine and spirits were only allowed in the state owned alcohol retail monopoly stores (Systembolaget). The experiment was planned to run from November 1967 until the end of 1968 but was discontinued prematurely due to

[1] Still in the US up to 50 percent of the childbearing age women drinks and 16 percent of these continue drinking during pregnancy (CDC, 2002). Göransson et al. (2003) survey pregnant women in Stockholm, Sweden regarding their consumption of alcohol. They find that 46 percent reported 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.

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

[3] Strong beer is restricted to a maximum alcohol content of 4.48 % by weight.

alarming reports of a sharp increase in alcohol consumption in the treatment regions, particularly among youths (SOU 1971:77). *Figure 1* depicts the trend 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.

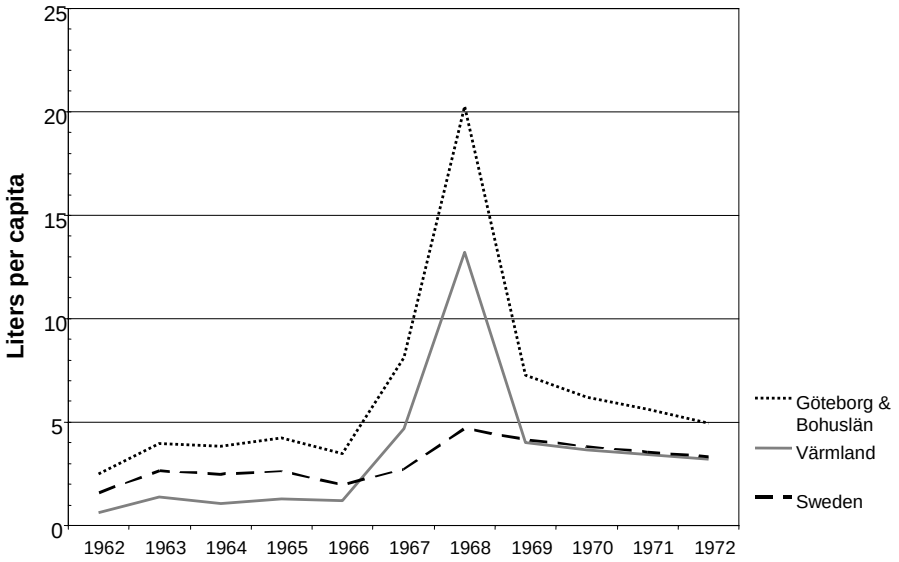

Figure 1 Yearly strong beer consumption per capita.
Source: SCB 1962-72

The temporary and exogenous increase in alcohol availability during the experiment provides us with a unique opportunity to solve many of the identification problems present in previous work. First, 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 counties who were in utero during the experiment with the outcomes of the surrounding "unexposed" cohorts. Second, the spatial restriction allows for a simultaneous comparison with the outcomes of children belonging to the same cohort but who was born in the control counties. This feature reduces the problem of general time effects confounding the estimates of the relationship of interest. Third, we capitalize on the fact that the experiment increased alcohol availability relatively more for youths under age 21 compared to older individuals. This is due to a minimum alcohol purchasing age law prohibiting youths below age 21 from buying strong beer

(and other spirits) at the Systembolaget stores prior to and after the experiment.[4] During the experiment the age restriction was 16 in grocery stores.[5] By comparing the outcomes of the children born by mothers below 21 at birth with the outcomes of children born by older mothers, any differing trends in outcomes between children born in treatment and control counties is taken into account. Finally, by focusing on the outcomes of the cohort in utero during the experiment but conceived *before* it started we are able to mitigate the concern that the experiment also might have altered family composition and thereby the child's outcomes.[6]

Using administrative data on all children born between 1964 and 1972 we find that the sharp increase in alcohol consumption during the experiment has had a substantial impact on the outcomes of those still in utero during the experiment. In particular we find that the children with the longest prenatal exposure to the experiment (between 5 and 8.5 months in utero) who was born by mothers under age 21 at delivery have on average less years of schooling, 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.

This is the first study focusing on the long term economic impact of prenatal exposure to alcohol. We also contribute to an emerging literature examining the importance of early life health conditions for subsequent outcomes.[7] With a few exceptions[8], the previous work has focused on infant health. Our study distinguishes itself from most of the previous work on early-life conditions and adult outcomes by

[4] Several previous studies on youth consumption have found responsiveness to policies pertaining to availability, such as the minimum legal drinking age, see e.g. Moore and Cook (1995).
[5] See SFS (1967:213) and SFS (1961:159) for the rules in effect during the experiment.
[6] See e.g. Kaestner and Joyce (2001) for evidence of the effects of alcohol use on the probability of unwanted pregnancies.
[7] c.f. Currie and Hyson (1999) and the references there in.
[8] For example, Van den Berg, Lindeboom and Portrait (2006) investigate the impact of early life economic conditions on mortality later in life; Case, Fertig and Paxson (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 of 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, Edlund and Palme (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.

providing relatively clear suggestions for policy tools that legislators promoting equal opportunities in health and education may use. The results furthermore suggest that investment in early-life health may not only be more humane compared to post-natal investment in terms of health outcomes, but potentially also a more efficient way to increase human capital accumulation in comparison with investments later in life.

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 also the implementation and results of the policy experiment. Section 3 describes the data and the empirical strategy. Section 4 presents the results and robustness checks. Section 5 concludes.

2 Prenatal alcohol exposure and the policy experiment

2.1 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 emerged not before 1968 in work by Lemoine et al. (1968) in France. Jones and Smith (1973) subsequently published similar findings and coined the Fetal Alcohol Syndrome (FAS).[9] The FAS diagnosis criteria require, besides confirmed maternal alcohol consumption during pregnancy, 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 behavioural problems may still suffer from alcohol-induced central nervous system deficits. Streissguth et al. (1991) demonstrated that there exists a predictable long-term progression of disorders into

[9] 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.

adulthood resulting from prenatal exposure to alcohol. They show that, among other things, poor judgment, distractibility, difficulty perceiving social cues and low IQ levels, were common among individuals exposed to alcohol in utero.[10] The evidence on the consequences of medium and lower level of alcohol consumption during pregnancy on birth outcomes is however less conclusive.[11] Yet, no consensus has been reached on any threshold level neither in terms of amount nor incidence of alcohol consumption during pregnancy with regards to the more subtle effects on health.[12]

West et al. (1994) and Goodlet and Horn (2001) summarize the vast medical literature focusing on the particular biological mechanisms having a part in the casual link between alcohol exposure and fetal development. Briefly, alcohol may affect the developing fetus directly as it readily crosses the placenta and distributes to the fetal cells, but also indirectly by reducing the supply of oxygen and nourishment. The dose and pattern of alcohol use seem in addition 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).[13] The detrimental effect of alcohol on fetal development is furthermore difficult to isolate to any specific timing of exposure during gestation, although the types of damage may vary between trimesters. While the central nervous system is susceptible to damage during all three trimesters, animal studies suggest that the third trimester is an extra sensible period for the brain (Marcussen et al., 1994). On the other hand for behavioural outcomes among human subject this pattern is less clear cut.[14] Besides direct effects on central nervous system and brain development prenatal alcohol exposure may also alter immune system development and functioning, leading to a higher susceptibility to infections (Zhang et al., 2005).

[10] The set up and findings from this and other studies on the same cohort of children followed from birth to age 25 and born in Seattle in 1974/1975 is summarized in Streissguth (2007). Similar to this study the information on maternal alcohol consumption was elicited when very little was known about the risks associated with alcohol use during pregnancy.
[11] See e.g. Rusell (1991) and Henderson et al. (2007) for reviews of this literature.
[12] See e.g. CDC (2004).
[13] This is consistent with these results from Streissguth et al. (1990, 1994) which found a binge drinking consumption pattern to be the best predictor of academic achievements.
[14] c.f. Coles (1994) for a discussion on the difficulties of identifying critical periods of alcohol exposure on offspring behavioral outcomes in humans.

Damage inflicted on other organs and extremities mainly seem to occur due to exposure in the first trimester. Hence, prenatal alcohol exposure may reduce the health stock through several different pathways.

2.2 Swedish alcohol policy and the strong beer experiment

Alcohol sales in Sweden are strictly regulated through an off-premise retail monopoly (Systembolaget). The only alcoholic beverages allowed to be sold 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 have changed radically. Sweden traditionally belonged to the "spirit-drinking" countries, but during the last 50 years spirit consumption has decreased substantially and gradually been replaced by wine and beer products (Leifmann, 2001). The dominant alcoholic beverage today is the strong beer that accounts for 29 % of the total alcohol consumption (SNIPH, 2005). One of the contributing causes of the changing pattern is active measures taken to encourage substitution of consumption between spirits and beverages with lower alcohol content.[15]

An example of such a policy was the experiment with free sales of strong beer (maximum alcohol contents of 5.6 % by volume, i.e. □ 4.48 % by weight), running between November 1967 and July 1968 in *Göteborgs-och Bohuslän* and *Värmland* counties.[16] During the experiment off-premise sales of strong beer was allowed in regular grocery stores as compared to solely in the Systembolaget stores prior to and after the experiment.[17] The regulations for wholesale trade with strong beer also changed. Anyone with rights to sell or serve beer were allowed to buy strong beer directly from a Swedish brewery or, if it concerned foreign beer, through a wholesaler. The intention of the experiment was that also the wholesale of strong beer was to be

[15] See Room (2002) for a comprehensive review of Swedish and Nordic alcohol policies after 1950.
[16] The experiment's setup and results 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 counties were selected from the pool of 25 counties.
[17] At the end of 1968, 1 530 shops were licensed for sales of beer (during the experiment also strong beer) in *Göteborg och Bohuslän* county as compared to the 26 Systembolaget stores in operation prior to and after the experiment.

carried out under similar conditions that would exist under free sale. Therefore wholesalers were able to order goods directly from foreign breweries.[18]

The original intention was to continue the experiment until the end of 1968, but soon after it was introduced reports of a sharp increase of alcohol consumption in the experimental counties, especially among youths, arrived. 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 counties during the experiment. In the first half of 1968 consumption increased from the 1967 level of 1.2 (0.32) million litres (gallons) to 10.5 (2.77) million litres (gallons) in Göteborgs- och Bohuslän. In Värmland the increase was even more drastic. In the first six months of 1967 0.2 (0.05) million litres (gallons) was sold compared to 3.0 (0.79) million litres (gallons) during the same months in 1968. Summarized over both counties consumption increased almost ten-fold. Per capita the consumption of strong beer increased from 1.8 (0.48) litres (gallons) during the first six months of 1967 to 15.3 (4.04) litres (gallons) in the same period in 1968 in Göteborgs- och Bohuslän. The corresponding figures for Värmland were 0.7 (0.18) litres (gallons) and 10.6 (2.8) litres (gallons) per capita for the two periods. From *Figure 1* we also see that 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 counties constitute a substantial share of the total population (12 % in 1968) and hence have a large impact on the national average. If we exclude the experimental counties, the rest of the country shows an increased consumption of 26 % from the first half of 1967 to the same period in 1968. From *Figure 1* we can also see that before the experiment the trends in consumption of strong beer in the two experimental counties followed the national average reasonably well. During the experiment consumption boomed and afterwards it fell back again. However, note that strong beer consumption in the experimental counties remained at an elevated level compared to the pre-experiment period even after the experiment had

[18] During the experiment all wholesalers were however obligated to report all transactions occurring with the amount of strong beer shipped to retailers.

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 cities. 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 neighbouring counties. 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.[19]

The possibility to evaluate the impact of the experiment on substitution between wine, spirits and strong beer is excellent. The Systembolaget stores kept exact records of the quantity sold per quarter in each county prior to, during and after the experiment. From the first half of 1967 to the first half of 1968 there was a decrease in liquor sales in the two experimental counties by ten and of five percent respectively, while the wine sales did not change notably. For the rest of the country the decrease in liquor sales was four percent while the wine sales increased with eight percent. These figures indicate that the experimental counties differ from the rest of the country by having larger decreases in liquor sales and no increase in wine sales. This suggests that in the experimental counties liquor and wine was substituted for strong beer. The changes in liquor and wine sales were however rather small and did not compensate the large increases in sales of strong beer.

A perhaps more important question is how the consumption of medium beer[20] was influenced. It is highly likely that the increased sales of strong beer lead to a decrease in the sales of medium beer, as these products are arguably closer substitutes.

[19] The reason is that while availability increased, prices (if anything) increased during the experiment (SOU 1971:77). In the empirical section we do however check whether the experiment generated any spill-over effects on children born in the neighboring counties.
[20] Medium beer may contain at most 3.6 % alcohol by weight.

Unfortunately there are no records of the quantity of medium beer sold at the county level. There are however data on aggregate monthly sales. The national consumption of medium beer increased with only 14 % during the first six months of 1968. This should be compared with the 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 rise of medium-strong beer with 10 percentage points, and that strong beer to some extent replaced medium beer in the experimental counties. During the first six months of 1967, 91 (24) million litres (gallons) of medium beer was sold, which means that the reduction should have been around 10 (2.6) million liters (gallons) overall. This quantity should be compared with the extra 11.8 (3.1) million litres of strong beer consumed in the experimental counties. Based on these calculations the total increase in the experimental counties in terms of liters of 100 % alcohol has previously been estimated to be at most five percent (SOU 1971:77). However, potential heterogeneous consumption responses to the increased availability between different sub-populations have not been considered.

The immediate impact on harms was only assessed in terms of number of persons taken into account 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 where 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 the implementing authority, the Alcohol Policy Commission, surveyed the local child welfare commissions (barnavårdsnämnder), the temperance commissions (nykter-hetsnämnder), the local education authorities and the police authorities in the experimental counties regarding their experiences of the free sales of strong beer so far. The main conclusion from this survey is that the temperance situation was negatively affected during the experiment. The police authorities underscored that the temperance situation was particularly worsened among youths. The main nuisances reported were an increased level of "disturbing behavior" and littering in connection with immense consumption of strong beer. An increase in drunk driving was also noted. Urban areas seem furthermore to have been more affected than rural areas (SOU 1971:77).

An explanation for the particularly detrimental effects on temperance among youths is probably that they experienced the largest increase in availability of alcohol during the experiment.[21] At the time the age limit at the Systembolaget stores was set to 21, and prior to the experiment this was the only place where strong beer could be bought. 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).

The main problem for estimating the effect of the experiment on consumption is the lack of data on alcohol use among sub-populations in the experimental counties. However, we know from a nationwide survey among youths aged 15 through 25 conducted in the summer of 1968, that beer consumption as the share of total amount of alcohol consumed was 44 % higher among youths than in the population on average. This suggests that the average increase in consumption among youths likely exceeds the previously estimated average increase of five percent. The survey also reveal that in 1968, 90 percent of the females and 97 percent of the males reported that their alcohol début occurred before turning 21 and that the abstainer rates in these age categories was low[22] (SOU 1971:77).

Considering the type of weekend binge drinking pattern common in Sweden[23], the report of a sharply worsened temperance among youths and the particularly damaging effects on the fetus from binge drinking, we believe that there is a clear scope for negative long term effects on children exposed to the experiment in utero. The following section describes the data and the empirical strategy we employ to identify the prevalence and importance of any such effects.

[21] 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 Norstrom and Skog (2005) for Sweden. For studies focusing on youths see e.g. Moore and Cook (1995).

[22] 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).

[23] 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. Kühlhorn et al. (1999).

3 Data and empirical strategy

3.1 Data and sample selection

The main hypothesis we aim to test in this paper is whether the exogenous increase in alcohol consumption during the experiment resulted in worse 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 interval 16 to 65 living or working in Sweden between 1990 and 2004. The LOUISE data are register-based and, besides information on year and month of birth, gender and county 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 furthermore linked each individual in the data to his/her biological parents.

In the main analysis we retain all first-born individuals alive in 2000 and born in Sweden between 1964 and 1972. We exclude children born in the 5 counties neighbouring the experimental counties to avoid diluting the estimates due to potential spill-over effects from the experiment. As the experiment was conducted at the county level this paper uses panel data for counties. However, for the reason discussed above, we also allow for potential differential effects of the experiment on children of young (below age 21 at delivery) and older mothers.

We divide the children born in the treatment counties in the selected cohorts into four groups depending on their exposure status: (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 who was conceived during the course of the experiment; and (4) those who where conceived after the end of the experiment and who was hence not exposed either during pregnancy or after birth.[24]

[24] 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.

This study mainly focuses on children belonging to group (2). The main reason is that we can be fairly certain that the experiment did not affect the timing of conception of this group of children. This is important as several studies have found an association between alcohol consumption and risky behaviour among youths (Kaestner and Joyce, 2001; Carpenter, 2005; Grossman and Markowitz, 2005). Hence, by focusing on children conceived prior to the experiment started, we 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).

To allow for heterogeneous effects of the experiment depending on duration and/or timing of exposure during gestation, we split the children of group (2) into two more groups: those who's mothers where in the first half of the pregnancy (month 1-4), and those in the second part (month 5-9) at the start of the experiment. The reason is that the first group (month 1-4) likely experienced a particularly high risk of being exposed to alcohol due to the experiment. Partly so due to the long duration of exposure in utero, but also because mothers in early pregnancy likely responded more strongly to an increase in alcohol availability compared to mothers in late pregnancy. A substantial share of the early pregnancy mothers did moreover probably not even realize that they were pregnant for some time during the experiment.[25] This may also have increased the probability of changing their consumption pattern due to the increase in availability. One should furthermore keep in mind that the knowledge about the risks associated with alcohol consumption during pregnancy was very low at the time.

3.2 A first look at the data

Table 1 presents descriptive statistics on the adult outcomes of children born in the control and treatment counties for the cohorts in utero prior to, during and after the experiment. All means are calculated using data aggregated to the county-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.

[25] The average pregnancy is not recognized until 5-6 weeks after conception (Floyd et al., 1999).

The first panel of *Table 1* reports the mean of the outcome variables for children born in the treatment counties and the control counties. Column 1 through 6 reports averages for children born in the experimental counties (column 1-3) and the control counties (column 4-6). Columns 7 through 12 reports the corresponding characteristics for children of mothers under age 21 at birth. The statistics in *Table 1* are calculated for the cohorts born during the first two quarters of each year. The table also presents the fathers and mothers ages at birth, the fraction of mothers with a high school diploma (measured in 1990), and the average number of children in each cell. From these (few) background characteristics one can note an increasing age trend among mothers, and that the number of young mothers decreases over time in both the treatment and the control counties. Looking at the average outcomes it seems like the children of the young mothers exposed to the experiment (i.e. born in 1968) in general 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 by mothers under age 21 in the treatment and control counties born between 1966:Q1 to 1970:Q4. Clearly the average years of schooling of the treatment county children conceived just prior to the experiment (born during the second quarter of 1968) deviate from the pattern displayed by the adjacent cohorts and the control county cohorts. A similar pattern is found in *Figure 3* where the comparison group now is children born in the treatment counties, but by mothers older than 20 at birth.

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

	Treated			Control			Treated			Control		
	All Mothers (I)			All Mothers (II)			Young Mothers (III)			Young Mothers (IV)		
Outcomes:	Born <1968	Born 1968	Born >1968	Born <1968	Born 1968	Born >1968	Born <1968	Born 1968	Born >1968	Born <1968	Born 1968	Born >1968
Education (years)	12.28	12.36	12.52	12.26	12.35	12.50	11.48	11.40	11.55	11.52	11.59	11.49
Fraction high school graduates	0.917	0.927	0.927	0.915	0.930	0.928	0.867	0.853	0.860	0.861	0.882	0.857
Fraction college graduates	0.162	0.163	0.173	0.160	0.156	0.174	0.064	0.052	0.064	0.068	0.068	0.064
Average log (yearly earnings) at age 32	7.206	7.341	7.402	7.213	7.333	7.406	7.091	7.143	7.302	7.1378	7.254	7.287
Percent w. zero earnings (age 32)	0.115	0.095	0.100	0.109	0.093	0.093	0.142	0.143	0.127	0.129	0.098	0.118
Percent on welfare in 2000	0.047	0.037	0.053	0.040	0.038	0.044	0.085	0.081	0.094	0.064	0.062	0.088
Fraction males	0.510	0.516	0.513	0.512	0.515	0.520	0.498	0.491	0.529	0.513	0.517	0.522
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 education (high school)	0.216	0.236	0.290	0.216	0.235	0.288	0.107	0.106	0.115	0.129	0.113	0.104
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. There are 1, 748 county-by-quarter-by-young/old mother-cells.

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 are clearly in line with the police reports suggesting that youths' alcohol consumption increased most during the experiment. The timing also corresponds well with the estimated duration of exposure as given in *Table A 1*.

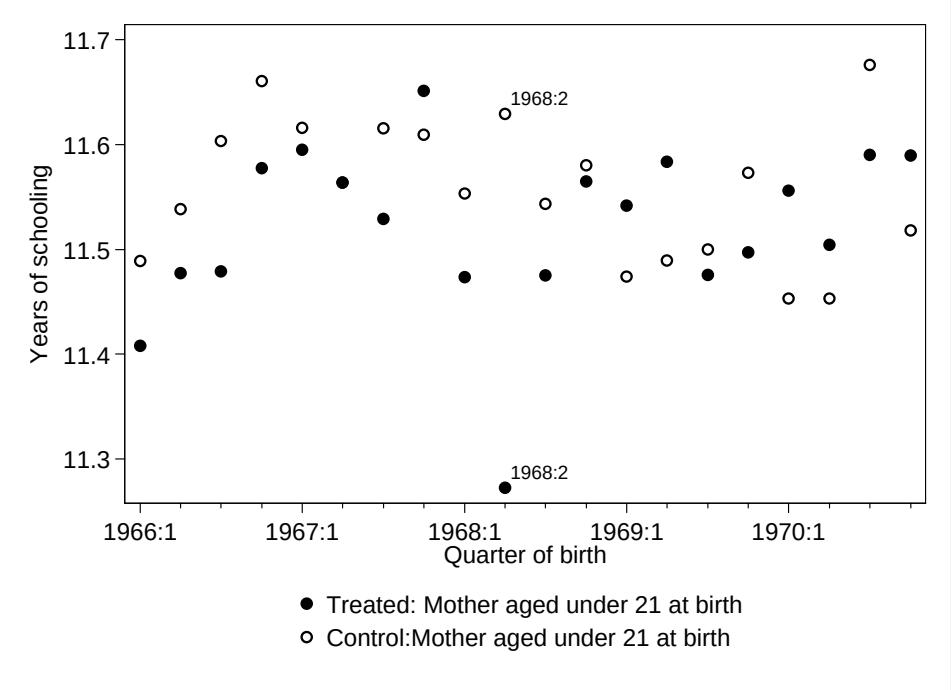

Figure 2 Average years of schooling, treated vs. control.

It is also of interest to investigate to what extent the effects carry over to labor market outcomes. *Figure 4* plots the average earnings[26] at age 32 for the children whose mothers was under age 21 at delivery in the control and treatment counties.

[26] The data used in the figure have been trimmed as to leave out those individuals with yearly earnings below the 1st percentile (SEK 1400) and above the 99th percentile (SEK 563,700).

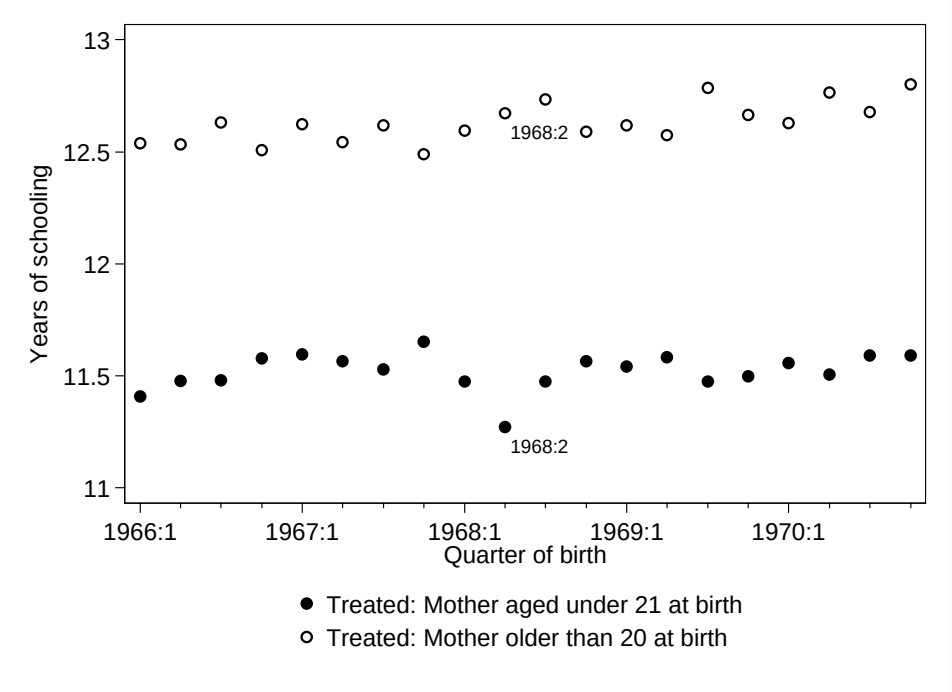

Figure 3 Average years of schooling, young vs. old mother.

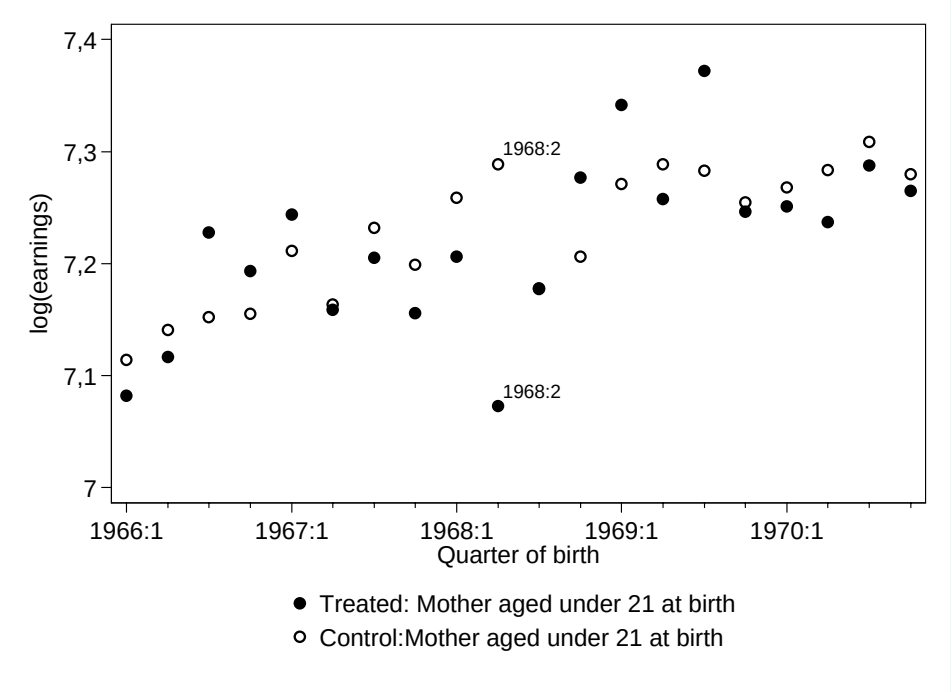

Figure 4 Average ln(earnings) at age 32, treated vs. control.

As in the case of education there is a distinct decrease in the relative earnings between treatment and control county children coinciding with the timing of the

experiment. To get a better picture of where this variation stems from, in the left hand side of *Figure 5* the cumulative earnings distribution of men and women born during the second quarter of 1968 are shown. These cumulative earnings distributions suggests that men in the lower end of the earnings distribution seems to have been 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 counties earning above the 50th percentile are relatively small.

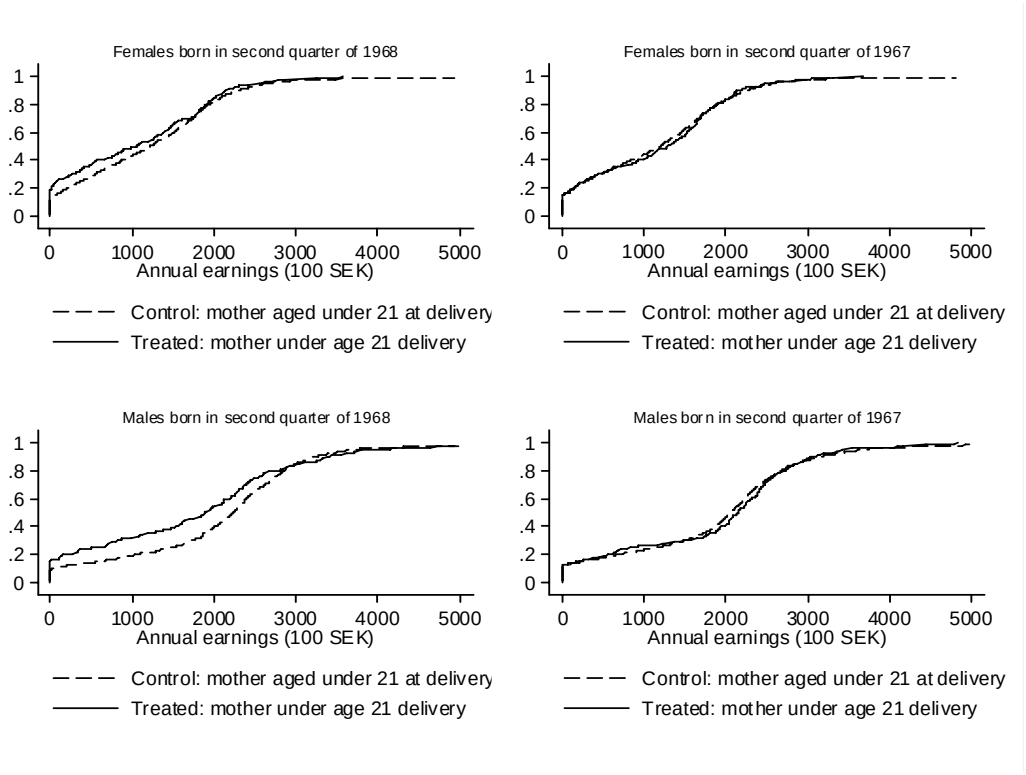

Figure 5 Cumulative distribution of earnings at age 32.

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.[27] For comparison on the right hands side of *Figure 5*

[27] The invariant rank assumption may however be a strong assumption in this context. A survey of youths aged between 15 and 25 conducted in the spring of 1968 reveals a clear 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 even have been those with the highest exposure.

the same distributions are shown for individuals born one year prior to the experiment. Again, the difference in distribution between the control and treatment county for this cohort is minimal.

Finally, *Figure 6* plots the fraction males in corresponding cohorts. Clearly the variance is higher in this case; but still there is a distinct drop in the fraction males coinciding with timing of the experiment and the changes in the other outcomes.

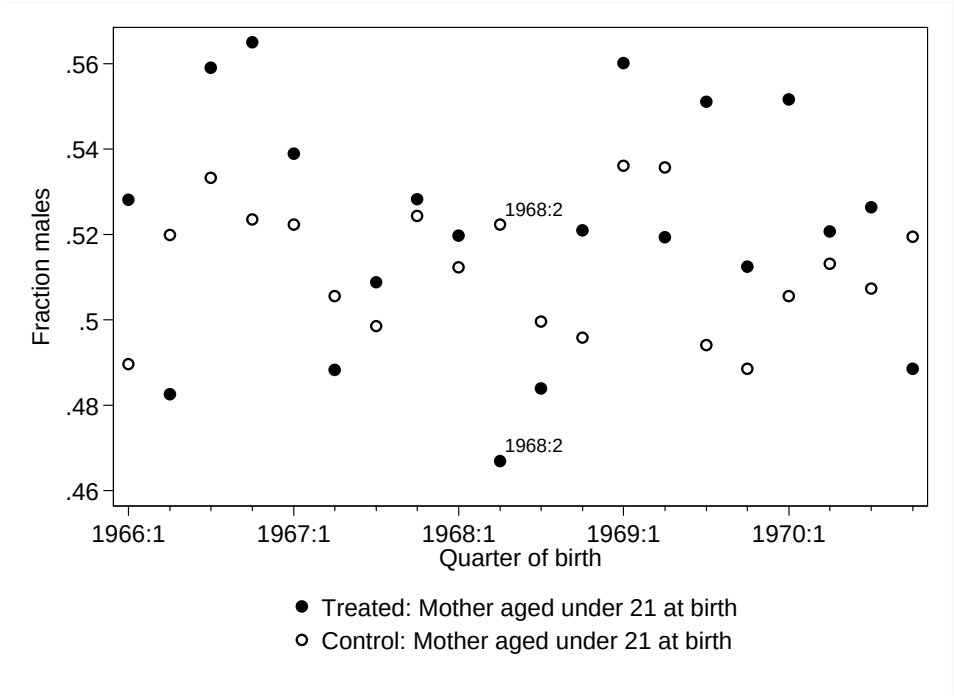

Figure 6 Sex-ratio in 2000

Previous research has 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). We explore this finding in more detail below.

3.3 Estimation strategy

The descriptive analysis above indeed suggests 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 employ a difference-in-difference-in-differences approach and estimate the following reduced form model:

$$\text{OUTCOME}_{c,t,mom<21} = \alpha_0 + \beta_1 \text{TREATED}_{c,t,mom<21} + \eta_c + \delta_t + \varphi_{mom<21} + \gamma_{c,t} + \lambda_{c,mom<21} + \mu_{t,mom<21} + \varepsilon_{c,t,mom<21} \quad (1)$$

using data aggregated by quarter of birth, age of mother (below/above 21) and county of birth[28]. In equation (1) *OUTCOME* is the outcome of interest (average years of schooling, share of high school graduates, share on welfare, average earnings etc.). *TREATED* is equal to 1 if the child is born by a mother under age 21 at delivery in the treatment counties and conceived between July and October 1967, and 0 otherwise.[29] 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 county of birth effects respectively. $\phi_{mom<21}$ is a parameter indicating if the child was born by a mother under age 21 at birth. The time (δ_t) and county (η_c) parameters respectively control for county and quarter of birth specific effects affecting the outcomes.[30] The $\phi_{mom<21}$ parameter accounts for fixed differences in outcomes between children born by mothers under age 21 and those above. The interaction terms $\gamma_{c,t}$, $\lambda_{c,mom<21}$ and $\mu_{t,mom<21}$ accounts for many other factors also related to the outcomes of interest. For example, as seen in *Table 1* over the observation period the number of under age 21 mothers decreased somewhat and hence the composition of these mothers may have deteriorated in terms of e.g. parental ability. The quarter*youngmom effect ($\mu_{t,mom<21}$) account for such compositional changes throughout the observation period. The county*young mom effects ($\lambda_{c,mom<21}$) in turn controls for fixed cross county

[28] 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-county correlations in the error term which may otherwise result in seriously underestimated standard errors as Donald and Lang (2007) shows. Using raw aggregated data as is done here 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).

[29] Hence in the estimations the "quarter" of birth is defined as Q1=Jan.-March, Q2=April-July, Q3=Aug.-Sept., Q4=Oct-Dec, as to better be able to capture the full effect on those conceived just prior to the experiment.

[30] See Costa and Lahey (2005) and Dobelhammer and Vaupel (2001) for the importance of season of birth effects on adult health.

differences in the composition of mothers giving birth to children under age 21. The error term $\varepsilon_{c,t,mom<21}$ are assumed to be IID and potentially heteroskedastic.

We estimate the model in equation (1) by OLS. The identifying assumption needed for a consistent estimate of β_1 is rather weak. There can be no change in unobserved factors coinciding with the timing of the experiment, only affecting the adult labor market outcomes of children born by mothers under age 21 at birth in the experimental counties. While we can never test this assumption directly, in the following section we report a number of robustness checks besides the baseline difference-in--difference-in-differences estimates in order to validate our estimation strategy. All regressions are weighted by the number of children in each cell. The reported standard errors are robust with respect to heteroscedasticity.

4 Results

To preview the results we find that children 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. Moreover, we find that males seems to be particularly affected by adverse conditions in utero as for most outcomes the effects of the experiment are more pronounced for males than for females. The cohort most highly exposed in utero is furthermore significantly more female. We also find that while there is no significant effect on the month of birth of females in the highest exposed cohorts, there is a negative effect on the month of birth of males. These two findings indicate that those most heavily exposed to the experiment were more likely to be either spontaneously aborted or born prematurely. The results are furthermore robust to a number of specifications changes.

4.1 Baseline results for educational outcomes

This section reports baseline results from regression analysis based on the specification in equation (1) focusing on the educational attainments of the exposed cohorts. Panel A, B and C of *Table 2* reports estimates of β_1 using the average years of schooling, the fraction high school graduates and the fraction with at least 3 years of higher education

as the dependent variable respectively. Columns (1)-(3) in each panel provide the estimate using the full sample, the male sample, and finally the female sample. The educational attainment is measured in 2000 when the children in the sample are aged between 28 and 36.

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 but not statistically distinguishable from zero at any conventional significance level. Turning to the fraction who graduated from high school it seems 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 reduced high school completion rate by 10 percent with respect to the mean among males (-0.09/0.9). The effect on the fraction who have graduated from higher education is only significant among males, although the point estimate is in this case similar in magnitude but again not significant for females. The effect on the fraction 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: Years of schooling	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	-0.266***	-0.473***	-0.101
	(0.049)	(0.124)	(0.151)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1393	1388	1387
R-squared	0.98	0.96	0.95
Mean	12.33	12.18	12.49
	Sample		
B. DEPENDENT VARIABLE: Fraction high school graduates	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	-0.039***	-0.092***	0.015
	(0.009)	(0.017)	(0.014)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1393	1388	1387
R-squared	0.90	0.85	0.82
Mean	0.921	0.910	0.934
	Sample		
C. DEPENDENT VARIABLE: Fraction graduated from higher education	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	-0.025**	-0.039***	-0.021
	(0.012)	(0.013)	(0.014)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1393	1388	1387
R-squared	0.95	0.92	0.92
Mean	0.159	0.138	0.181

Note: 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 county. "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, county 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.

4.2 Baseline results for labor market outcomes

Moving on to the impact on labor market outcomes presented In *Table 3*, we find that males and females in this case are similarly affected, although the estimates are more precisely estimated for the male sample.

On average the exposed cohort has close to 24 percent lower earnings at age 32. [31] Again males seem to have been somewhat more strongly affected than females. However, the assumption that women's earnings at age 32 are accurate measures of their permanent earnings is questionable. Böhlmark and Lindqvist (2006) estimates of life-cycle biases shows that for Swedish women one would ideally like to use earning after age 40 in order to get a decent proxy for permanent earnings.

Panel B in *Table 3* present the result from a regression using the fraction with zero earnings as the dependent variable. In this case there is a similar pattern for both men and women, the experiment increases the risk of having no labor income at all at age 32 with 7 percentage points for the children in utero. The last panel in *Table 3* reveals that the fraction on welfare among the exposed males is 5 percentage points higher in the exposed cohort. The fraction females on welfare are also higher but the impact is not statistically different from zero.

[31] The size of the earnings effect is big. One potential reason could be that the main part of the action takes places at the lower end of the earnings distribution as shown in figure 5. Hence, while the log transformation of the earnings variable simplifies interpretation it also emphasizes differences in the lower end of the earnings distribution. Running the same regression on the non-logged earnings (still excluding the zeros) indeed reduces the point estimate significantly to around 15 % , which is still a sizeable effect.

Table 3 The impact of the experiment on labor market outcomes

	Sample		
A.DEPENDENT VARIABLE: Average ln(earnings)	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	-0.241*** (0.053)	-0.228*** (0.081)	-0.177** (0.097)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1391	1387	1388
R-squared	0.88	0.87	0.79
Mean	7.26	7.57	6.93
	Sample		
B.DEPENDENT VARIABLE: Fraction with zero earnings	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	0.071*** (0.012)	0.069*** (0.017)	0.069*** (0.013)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1393	1388	1387
R-squared	0.76	0.71	0.67
Mean	0.10	0.09	0.11
	Sample		
C.DEPENDENT VARIABLE: Fraction welfare participants	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	0.036*** (0.009)	0.051*** (0.016)	0.021 (0.021)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1386	1386	1386
R-squared	0.84	0.74	0.76
Mean	0.042	0.039	0.046

Note: Each column and panel represents a separate regression. The dependent variable is average log earnings, fraction with zero income or fraction on welfare. The unit of observation in each regression is all children alive in 2000 born either by mothers aged>=21 or below in a given year, quarter and county. "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, county of birth, mother under age 21 at delivery dummies and the corresponding interaction terms. Earning outcomes are measured at age 32 while fraction welfare recipients are measured in 2000. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. The earnings coefficient presented is given by the transformation (exp(beta)-1). Heteroscedasticity robust standard errors are reported in parenthesis.

4.3 Further results and robustness checks

The pattern from *Table 2* and *3* is clear. The alcohol experiment seems to have resulted in significantly worse adult outcomes for the children in utero during the experiment. Males seem to have been particularly strongly affected. Why then should males be so much more affected by an increased prenatal exposure to alcohol than women? The results in *Table 4* provide some guidance. The table reports coefficients from regressions on three health related outcomes, potentially yielding some insights into the underlying mechanism explaining the differences in outcomes between women and men. Column (1) presents the point estimate from a regression using the standard model from equation (1) on the full sample with the fraction males in each cell as the dependent variable. The coefficient suggests that the fraction males are 7.2 percentage points lower in the exposed cohort. Column (2) & (4) presents the results from a regression where the dependent variable is the average month of birth for children born between January through 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 a large medical and biological literature suggesting that male fetuses are more sensitive to adverse conditions in early life than females, see e.g. Lee et al (1998) and Wells (2000) and the references cited therein.[32]

[32] The results are also consistent with differences in sensitivity to binge alcohol exposure displayed among male and female rats found by Goodlett and Peterson (1995).

Table 4 The impact of the experiment on health related outcomes

	DEPENDENT VARIABLES:				
	Fraction males (1)	Month of birth (2)	ln (cohort size) (3)	Month of birth (4)	ln (cohort size) (5)
	ALL	MEN	MEN	WOMEN	WOMEN
In utero	-0.072*** (0.024)	-0.240** (0.122)	-0.166*** (0.055)	0.042 (0.146)	0.134* (0.070)
Year/Quarter dummies	YES	YES(YEAR)	YES	YES(YEAR)	YES
C.O.B dummies	YES	YES	YES	YES	YES
Mom age<21 dummy	YES	YES	YES	YES	YES
Observations	1393	359	1385	354	1386
R-squared	0.56	0.65	0.98	0.63	0.98
Mean(not logs)	0.515	4.00	124.97	4.00	117.85

Note: Each column and panel represents a separate regression. Except for when the dependent variable is "month of birth" the outcomes are measured within each county 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 county/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, county 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.

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 counties. In *Table 5* we examine to what extent such cross-border shopping also resulted in adverse outcomes for the children born in these counties. Remember that in the previous regressions these children were excluded from the sample. *Table 5* reports coefficients from the same specifications as reported in *Table 2* through 4 but now the "in utero" dummy is equal to 1 for the cohort of children born between April and July 1968 by mothers under age 21 in one of the five counties neighboring the experiment area[33]. The results from this exercise suggest that cross-border shopping did not affect the outcomes of the children in the neighbouring counties to any major extent. None of the coefficients is significantly different from zero at any conventional level of significance. Given that the neighbouring counties and the treatment counties today are

[33] We exclude the experiment county children from these regressions.

Table 5 The impact of the experiment on neighboring counties: labor market, educational and health outcomes

	DEPENDENT VARIABLES: Labor and education											
A.	Years of schooling		High school graduates		Higher education		Earnings		Zero earnings		Welfare	
Sample	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
In utero (month 1-4)	-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)
Number of observations	1598	1598	1598	1598	1598	1598	1598	1598	1598	1598	1598	1598

	DEPENDENT VARIABLES: Health				
B.	Fraction males	Month of birth		ln(cohort size)	
Sample	ALL	Men	Women	Men	Women
In utero	-0.006	0.119	-0.037 (0.123)	0.022	0.037
	(0.025)	(0.085)		(0.097)	(0.074)
Number of observations	1598	413	408	1598	1598

Note: Each column and panel represents a separate regression. Except for when the dependent variable is “month of birth” the outcomes are measured within each county 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 county/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.

highly interdependent and constitute a local labor market this exercise also strengthens the case for our estimation strategy.

Table 6 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 we also include the original "in utero" dummy to see 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. We interpret this finding as evidence that it is indeed prenatal exposure to alcohol rather than an increased incidence of detrimental post-natal events caused by the experiment that drives the main results. Moreover, the respecified model yields qualitatively similar results as the baseline model which is reassuring.

Table 7 a reports the impact of the experiment on children of mothers in late pregnancy (5th to 9th month) at the start of the experiment vs. the original treatment cohort. Only the probability to have graduated from high school seems to have been significantly affected in the earlier cohort, whereas the estimates of the impact on the original cohort are virtually identical to the main results. One might be tempted to interpret the result from 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. The estimation strategy we employ here does unfortunately not allow us to distinguish between these two mechanisms.

Table 6 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:					
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)
In utero (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	1394	1394	1394	1394	1394	1394
	DEPENDENT VARIABLES:					
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)
In utero (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	1394	1394	1394	1394	1394	1394

Note: Each column and panel (A & B) represents a separate regression. The outcomes are measured within each county 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 of 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 7 a 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
In utero (month 5-9)	0.036	-0.019**	0.014	0.032	-0.005	0.004
	(0.097)	(0.009)	(0.021)	(0.075)	(0.016)	(0.006)
In utero (month 1-4)	-0.256***	-0.043***	-0.023*	-0.242***	0.070***	0.033***
	(0.063)	(0.010)	(0.013)	(0.053)	(0.013)	(0.010)
Number of observations	1394	1394	1394	1394	1394	1394

Note: Each column represents a separate regression. The outcomes are measured within each county 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.

In order to attain a clearer picture of the dynamics of the impact of the experiment *Table 7 b* report estimates from regressions using monthly rather than quarterly data.[34] Specifically we now let the treatment window slide 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 we now start with those born between November 1967 and February 1968, continuing with December 1967 through March 1968, up until 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 follows a clear pattern. While there are no significant differences for the children with the least amount of exposure (reported in column I, II) there is an increasingly worsening trend in outcomes as the treatment window is rolled towards the most exposed cohorts. For the educational outcomes the strongest negative effect is reached somewhere between March and August 1968 (columns V through VII), as is the case for earnings.

For years of education and earnings we 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 1 cohort exposed to the experiment. From this figure we more clearly see that the timing in the dip in relative outcomes among the highest exposed cohorts are unusually large and fits very well with the number of weeks of exposure.

[34] Some precision is however inevitably lost when calculating the outcomes variables as cell sizes decreases compared to when using quarterly data.

Table 7 b 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 November 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
Outcome:											
Years of Schooling	0.065	-0.063	-0.173**	-0.224***	-0.240***	**-0.266*** **	-0.300***	-0.220**	-0.110	-0.130	0.043
	(0.134)	(0.079)	(0.074)	(0.075)	(0.082)	**(0.083)**	(0.95)	(0.112)	(0.108)	(0.101)	(0.097)
High School grad.	-0.002	-0.014	-0.030*	-0.026*	-0.044***	**-0.037** **	-0.036*	-0.019	-0.013	-0.009	0.007
	(0.015)	(0.017)	(0.016)	(0.015)	(0.012)	**(0.016)**	(0.020)	(0.022)	(0.019)	(0.017)	(0.016)
University grad.	0.012	-0.011	-0.017	-0.018	-0.015	**-0.023** **	-0.036***	-0.030*	-0.010	-0.017	0.001
	(0.025)	(0.017)	(0.016)	(0.015)	(0.016)	**(0.012)**	(0.013)	(0.018)	(0.020)	(0.020)	(0.020)
Labor earnings	-0.012	0.026	-0.035	-0.163	-0.204*	**-0.290*** **	-0.203*	-0.081	-0.040	0.014	0.011
	(0.043)	(0.086)	(0.102)	(0.119)	(0.118)	**(0.092)**	(0.109)	(0.068)	(0.072)	(0.076)	(0.079)
Zero earnings	-0.008	0.016	0.051*	0.071***	0.076***	**0.072*** **	0.034**	0.011	-0.008	-0.016	-0.036***
	(0.021)	(0.018)	(0.029)	(0.024)	(0.024)	**(0.026)**	(0.017)	(0.023)	(0.021)	(0.018)	(0.013)
Welfare dependency	-0.001	0.005	0.017	0.012	0.022	**0.034** **	0.017*	0.013	0.007	0.003	0.002
	(0.016)	(0.013)	(0.018)	(0.017)	(0.016)	**(0.015)**	(0.010)	(0.013)	(0.013)	(0.013)	(0.013)
Fraction males	-0.002	0.004	-0.008	-0.058**	-0.064**	**-0.073** **	-0.039	-0.025	-0.015	-0.001	0.003
	(0.028)	(0.024)	(0.028)	(0.027)	(0.028)	**(0.029)**	(0.033)	(0.031)	(0.040)	(0.041)	(0.043)
Number of observations	4240	4240	4240	4240	4240	4240	4240	4240	4240	4240	4240

Note: Each column and panel represents a separate regression using the model in equation (1). The outcomes are averages/fractions within each county 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).

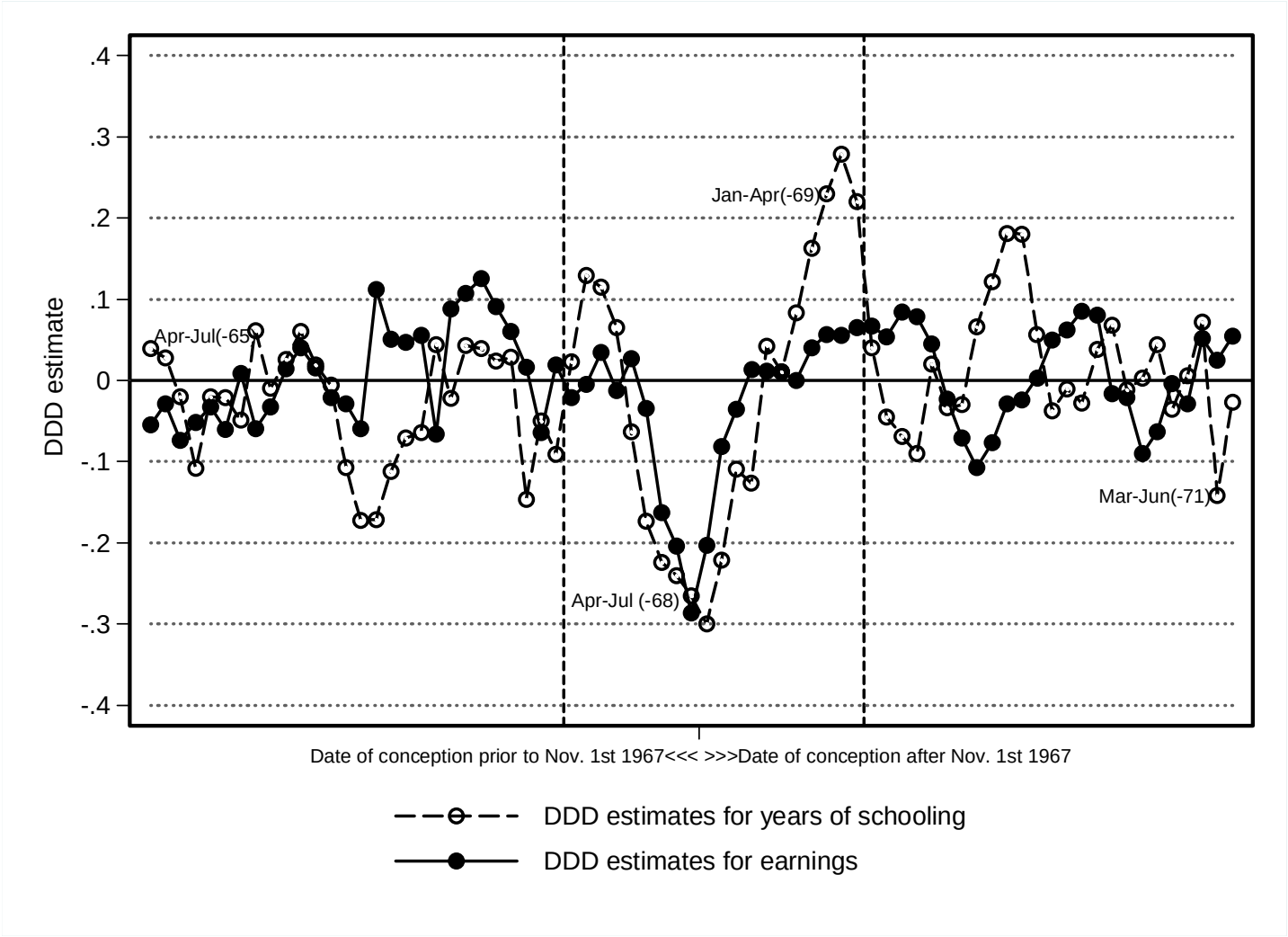

Figure 7 Difference-in-difference-in-differences estimates for years of schooling and earnings

Second, while there also exist dips for other cohorts for each one of the outcomes, during the experiment the change in both educational outcomes and earnings follows each other very well. Third, interestingly in the case of education the DDD 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. From previous research we know that alcohol consumption increase the frequency of risky behaviour among youths.[35] Hence, if the increase in alcohol consumption increased fertility relatively more among high ability parents this may explain the relative increase in educational attainments among cohort conceived at the end of the experiment period.[36] To be able to test this hypothesis directly we would ideally like to have some parental quality indicator measured *prior* to birth of the child. As such a measure is not available to us we look at whether the fraction of children born by a mother with a high school degree (measured in 1990) is higher among those conceived during last part of the experiment.[37] 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 high school graduated mothers increased by 4 percentage points (on a base of 18 %).[38] 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 rightmost vertical dashed lined in *Figure 7*).

[35] See e.g. Carpenter (2005), Grossman and Markowitz (2005).
[36] In the absence of legalized abortions (not freely available until 1975) the potential pathways for such effects to occur are several. One reason is that high skill 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 high skilled women than low skilled women.
[37] Note that for these children conception was potentially affected by the experiment, while the time in utero during the experiment was short.
[38] This effect is driven by 35 percent increase (est. 0.35, std.err 0.15) in the number of children born by high school educated mothers rather than a decrease in the number children born by less educated mothers. The estimates are attained by running the baseline regression with the fraction of mothers with high school diploma as the dependent variable. We use the last cohort where all children was conceived during the experiment (children born between January and April 1969) as the treatment group and also include a dummy for children born in the same months of 1968 in the specification. For the cohort size outcomes separate regressions are run for children of high school and non-high school educated mothers.

The pattern in *Figure 7* also suggests that in order to identify the effects of a given alcohol policy intervention on children's outcomes, it seems crucial to investigate whether parental composition and fertility rates have been affected too. Neglecting such effects may otherwise underestimate the true effect of the policy. However, for the cohorts for whom we in the present case can rule out direct effects on conception rates (i.e for those conceived before the experiment started), increased alcohol exposure do indeed seem to have significant and economically important effects on adult outcomes.

5 Summary and conclusions

We use a Swedish alcohol policy experiment conducted in the late 1960s to identify the impact of prenatal exposure to alcohol on adult labor market and educational outcomes. Under age 21 youths experienced the largest increase in alcohol availability during the experiment and reportedly increased their consumption of alcohol the most. In line with these reports we find that the cohort of children who was born by mothers under age 21 and exposed to the experiment while in utero have significantly reduced earnings, higher welfare dependence rates, and lower educational attainments compared to the surrounding cohorts. While we can not fully rule out that other unobserved factors correlated with alcohol consumption may explain at least parts of the relatively large economic effects of prenatal alcohol exposure found here (e.g. smoking and illicit drug use) the magnitude and timing of the effects suggest that it is indeed alcohol that drives the results.[39]

This paper is the first to address this issue within the field of economics. It is also the first paper to apply a quasi-experimental strategy to identify the effect of maternal alcohol consumption during pregnancy on the child's outcomes. The findings in this paper add to a growing body of research documenting the effects of prenatal insults on adult outcomes. Together these studies suggest that investments in prenatal health may have long term consequences not only for the individual but also generate multiplier effects beneficial to society in general.

[39] Attempts to asses the effects of alcohol use vs. other drugs suggest that prenatal alcohol exposure may have broader and more long lasting effects compared to other drugs, see e.g. Day and Richardson (1994).

Our study also provides evidence on an overlooked and potentially important mechanism explaining the linkage between teenage childbearing and child outcomes.[40] Given the findings in this paper, identifying effective policy tools to reduce binge drinking among youths may not only improve the health of the individual, but also the outcomes of children born by teenage mothers. [41] [42]

A final caveat is in order before generalizing our results to other settings. To what extent the results found here would emerge 30 years from now if a similar experiment was implemented is uncertain. For example, information regarding the health hazards of maternal drinking during pregnancy is arguably much higher now than it was 30 to 40 years ago. In some developing countries and among sub-populations in more developed countries reduced alcohol availability and/or increased information may however still prove to be an important tool for improving health and economic outcomes of future generations.

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

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

[42] 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.

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Appendix A

Table A 1 Estimated prenatal exposure to the experiment

Month of birth	Est. date of conception[†]	Est. gestational age at start of experiment (month)	Minimum/Maximum number of weeks in utero during experiment		Trimester under exposure:	Experiment may have affected conception rate?
Before November 1967	Before February 1967	born	0	0	-	NO
November 1967	February 1967	8-9	0	4	3rd	NO
December 1967	March 1967	7-8	4	8	3rd	NO
January 1968	April 1967	6-7	8	12	3rd	NO
February 1968	May 1967	5-6	12	16	2nd, 3rd	NO
March 1968	June 1967	4-5	16	20	2nd, 3rd	NO
April 1968	**July 1967**	**3-4**	**20**	**24**	**2nd, 3rd**	**NO**
May 1968	**August 1967**	**2-3**	**24**	**28**	**1st, 2nd, 3rd**	**NO**
June 1968	**September 1967**	**1-2**	**28**	**32**	**1st, 2nd, 3rd**	**NO**
July 1968	**October 1967**	**0-1**	**32**	**34**	**1st, 2nd, 3rd**	**NO**
August 1968	November 1967	-	30	34	1st, 2nd, 3rd	YES
September 1968	December 1967	-	26	30	1st, 2nd, 3rd	YES
October 1968	January 1968	-	22	26	1st, 2nd, 3rd	YES
November 1968	February 1968	-	18	22	1st, 2nd	YES
December 1968	March 1968	-	14	18	1st, 2nd	YES
January 1969	April 1968	-	10	14	1st, 2nd	YES
February 1969	May 1968	-	6	10	1st	YES
March 1969	June 1968	-	2	6	1st	YES
April 1969	July 1968	-	0	2	1st	YES
After April 1969	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.

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