IFAU – INSTITUTE FOR
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EVALUATION

Essays on social interactions and the long-term effects of early-life conditions

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Abstract

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This thesis consists of four self-contained essays.

Essay 1: From the late 1970s through mid 1990s blood-lead levels decreased drastically in Swedish children due to the sharp phase-out of leaded gasoline. Exploiting the distinct geographical variation in early childhood lead exposure induced by the regulations together with micro data on all children in nine birth cohorts I show that reduced lead exposure early in life improves scholastic performance, cognitive ability, and labor market outcomes among young adults. At the relatively low levels of exposure considered, the analysis reveals a nonlinear relationship between local air lead levels in early childhood and adult outcomes, indicating the existence of a threshold below which further reductions no longer improve adult outcomes. Importantly, the effect is greater for children of lower socioeconomic status (SES), suggesting that pollution is one mechanism through which SES affects long-term economic outcomes and that environmental policies potentially can reduce the intergenerational correlation in economic outcomes.

Essay 2: 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.

Essay 3: We utilize a large-scale randomized social experiment to identify how co-workers affect each other's effort as measured by work absence. The experiment altered the work absence incentives for half of all employees living in Göteborg, Sweden. Using administrative data we are able to recover the treatment status of all workers in more than 3,000 workplaces. We first document that employees in workplaces with a high proportion treated co-workers increase their own absence level significantly. We then examine the heterogeneity of the treatment effect in order to explore what mechanisms are underlying the peer effect. While a strong effect of having a high proportion of treated co-workers is found for the non-treated workers, no similar effects are found for the treated workers. These results suggest that pure altruistic social preferences can be ruled out as the main motivator for the behavior of a non-negligible proportion of the employees in our sample.

Essay 4: We examine the influence that co-workers' have on each other's fertility decisions. Using linked employer-employee panel data for Sweden we show that female individual fertility increases if a co-worker recently had a child. The timing of births among co-workers of the same sex, educational level and co-workers who are close in age are even more influential. Consistent with models of social learning we find that the peer effect for first time mothers is similar irrespective of the birth order of the co-worker's child, while for higher order births within-parity peer effects are strong but cross-parity peer effects are entirely absent. A causal interpretation of our estimates is strengthened by several falsification tests showing that neither unobserved common shocks at the workplace level, nor sorting of workers between workplaces are likely to explain the observed peer effect. We also provide evidence suggesting that peers not only affect timing of births but potentially also completed fertility, and that fertility peer influences spills over across multiple networks. Our results suggest that social interactions could be an important factor behind the strong inter-temporal fluctuations in total fertility rates observed in many countries.

To my beloved family:
Dagny, Åke and Caroline

Acknowledgments

A doctor is worth several men (Homer, *The Iliad*).

In many languages an *odyssey* has come to define a long trip or period involving a lot of different and exciting activities, especially while in search for something. An equally condensed but less pretentious description of my years spent as a Ph.D.-student at the Department of Economics in Uppsala is hard to come up with in the final hours before this manuscript is supposed to be handed in. It has indeed been an incredibly exciting period containing both times of hope and despair.

A legion of friends, colleagues and relatives has contributed to this thesis. I am sure that not all of them (potentially nor I) are fully aware of their individual importance for the completion of this work. Since so many have contributed in one way or another, giving *all* of you the proper recognition surely deserved would require another full chapter. In this section I try to acknowledge the most influential ones. If you expect to, but do not find your name below, rest assured that you are not alone.

First and foremost I would like to thank my main advisor Per Johansson for his continuous support and encouragement from the first day I stepped into his office during my undergraduate studies. I first met Per when I was about to write my second essay in Economics. Per's natural, kind, clear and inspiring way of communicating his view of economics and econometrics was the reason why I decided to apply for the Ph.D. program. Without a doubt this thesis would not be in your hands without all the effort and time Per has invested in me (suitably already from an early "academic" age). During a brief venture into Statistics Per suggested that I should contact Patrik Hesselius, who at the time was a post doc at the Department of Economics in Uppsala. Patrik has also considerably influenced the way I have approached economics and his clear and constructive suggestions has always been of great help many times when not knowing how to proceed. I strongly suspect that it was mainly Per's and Patrik's recommendations that convinced the committee to give me the chance to start working on this thesis. Thank you for all your support over the years, for patiently listening to all crazy research ideas, and for giving me the opportunity!

Secondly, I would like to thank my close friends and colleagues Hans Grönqvist and Olle Folke. Hans for his brilliance, hilarious madness, and for always loosing on *the real* tennis court (and letting me win *one* (1) table

tennis match) during the last 8 years. Thanks for all the excellent suggestions and discussions on every imaginable topic we ever considered, for always pushing things one step further and finally for being a great friend. Olle, a.k.a. *Olof the altruist* or *Count Mörner*, much kudos to you for support in times of need, for promoting my case in Cambridge, for subletting your apartment on Åsögatan and the penthouse in Barcelona, and for everything else you have helped out with during the last years.

Many other colleagues at both the Department of Economics in Uppsala, at the Institute for Labor Market Policy Evaluation (IFAU), elsewhere in Sweden and abroad have also been of great help to me. IFAU has proved to be an excellent place to conduct the type of research contained in this thesis. Not only because of all of the skilled researchers active there and their openness to interaction with Ph.D. students, but also because of the natural and friendly workplace atmosphere. Thank you *Everyone* for always answering all my questions (and for all the memorable discussions on various important sporting events ;). In particular I would like to thank Erik Grönqvist and Björn Öckert for always keeping your doors open, P-A Edin, Peter Fredriksson, Bertil Holmlund for your support and Per-Petterson Lidbom for carefully reading and commenting on two of the manuscripts. The seamless infrastructure available at IFAU and at the Department in terms of data support and management and administration has also been very valuable; thank you Jörgen Moen, Cecilia Andersson, Anahid Zakinian, Maria Karlsson, Annette Olsson, Margareta Wicklander and Lena Blom, Åke Qvarfort, Katarina Grövall, Ann-Sofie Wettergren-Djerf, Monica Ekström and Monica Waller.

I would furthermore in particular like to thank the super Ph.D. students belonging to the same cohort as I do: Niklas Bengtsson, Kajsa Hanspers, Anders Norberg and Johan Vikström. This thesis by itself is direct evidence on the importance of peer effects in higher education: without you it would have been in much worse shape than it is. I think that we all should be proud over what we have accomplished so far academically, and I assume much more is to come. I have also enjoyed the company of several others at the Department and at IFAU; Pia Fromlet, Mickael Elinder, Mikael Lindahl, Caroline Hall, Matz Dahlberg, Eva Mörk, Patric Tirmén, Christian Andersson, Nikolay Angelov, Per Engström et alia.

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paisano in Manhattan, for taking me to all the great dive bars, the punk-rock karaoke places, and excellent hamburger joints.

In the spring before my longer stay in the US, while on a shorter visit to NYC, another of my Ph.D. program colleagues in Uppsala, Robin Douhan, his wife and I spent an absolutely fabulous night out at a Mexican diner celebrating *Cinco de Mayo* until eventually the brain freeze induced by all the Frozen Margaritas forced us to go home. Little more than a year later, in August 2009, Robin sadly, unexpectedly and much too early passed away. Robin was a brilliant, kind and low-key man, whom I was particularly proud to be a colleague of, and whom I really looked forward to spend more time with in the future, as a friend and as a colleague. It is truly depressing to know that this will not happen.

Some of the most influential factor inputs in the production of this dissertation are likely also least aware about their key role in the initiation and/or completion of it. Dan S., Daniel S., Erik B., Elin H., Henrik O., Karin J., Linda M., Mårten & Emma, Marcus F. (and his beautiful family) and Tobias B.; thanks for from time to time reminding me that the doctor Homer was referring to above was most likely not someone with a doctoral degree in Economics. Thanks for all the good times and for all the non-economic stimulus (90% of the time in form of puns, beautiful dance moves, *the* music, infected debates on politics, or just pure silliness). Without you guys stepping on to the scene, writing this thesis would probably gone faster but would also have been infinitely much duller; and like them others I would probably still be petrified.

A co-author of one of the studies in this thesis is Lena Hensvik, whom I would like to direct a special thank to for all the clear thinking, effort and efficiency which was *absolutely* necessary in order to complete "the fertility paper" in time for the deadline of this thesis. Thank you, *Darlin' companion*, for all the comments and suggestion on the other essays in the dissertation and for all inspiring discussion we have had over the last years.

Finally to my family: my mother, my father and my sister. It is of course impossible to with only a few sentences capture the full influence that you have had on me and how grateful I am to have you around. Needless to say, your loving support throughout my whole life is *the* key ingredient behind every word on each page of this dissertation. Thank you so much for always believing in me and for always being there.

J Peter Nilsson, October 30, 2009, Stockholm

Contents

Introduction

The four self-contained essays in this dissertation focus on two broad questions: How important are early-life conditions for future educational attainments and labor market outcomes? And how important is the influence of social networks in the workplace in determining behavior of the employees?

The essays are all empirical and all make use of the extensive population micro data available in Sweden. An additional common feature is the emphasis on identification and estimation of policy relevant parameters and to uncover behavioral aspects of these estimated "average" policy parameters. Below I put the four studies contained in thesis into context, give a basic intuition behind the empirical strategies employed and summarize the main findings.

Part 1: How important are early-life health for future educational attainments and labor market outcomes?

Beginning with the Colman report in the 1960s, a large literature has provided evidence suggesting that socioeconomic status (SES) gaps in children's cognitive and noncognitive skills have its' roots mainly in family background. The SES gap in cognitive skills tends to emerge *before* formal schooling begins and persists throughout childhood. Figure 1 gives a representative picture of the results from these studies. It shows the trends in children's cognitive test scores by maternal educational attainments (from top to bottom; college graduated, some college, high school graduate and less than high school) as the child ages. Since several studies at the same time has documented that cognitive and noncognitive skills are powerful predictors of social and economic success, it has been suggested that socioeconomic inequalities in future outcomes to a large extent has its origins in family background. Moreover, genes and environment interact and are both involved in the production of these skills.

Cunha and Heckman (2007) summarize the evidence from this literature and propose a theoretical framework as to why early childhood conditions are likely to play such a crucial role in determining long-term outcomes. In a nutshell, their idea is that skill formation are partly determined by the previous acquired skills since the accumulation of new skills is facilitated by

higher levels of previous skills; that is *skills beget skills* and *abilities beget abilities*. While remedying investments can reduce the impact of early disadvantages, the dynamic nature of skill formation provides a rational for why early investments in disadvantaged children typically are found to be more productive and less costly than interventions later on.

Besides the strong policy implications of these findings regarding the optimal allocation of resources across the child's life-cycle (c.f. Cunha and Heckman, 2007), the question naturally arises of *what* it is in the family that matter for children's long-term social and economic success.

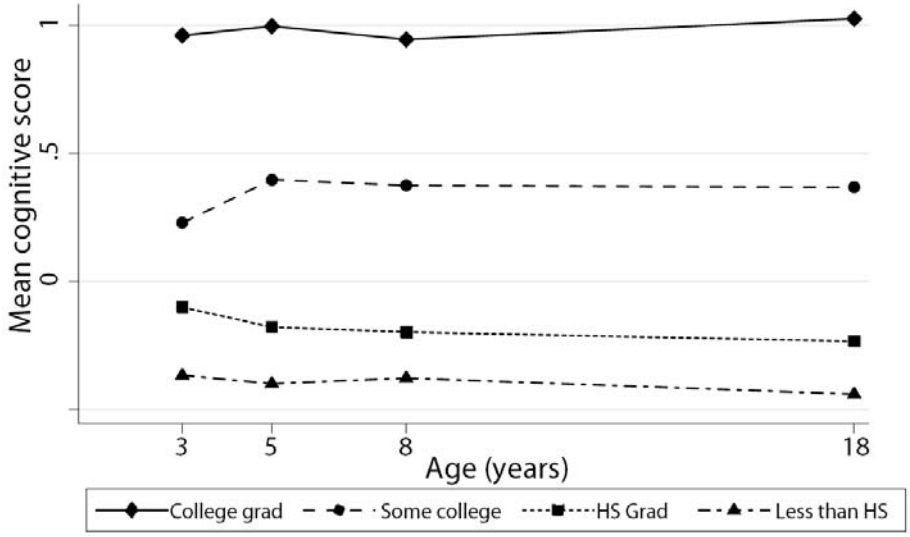

Figure 1 Trend in mean cognitive score by maternal education. (c.f. Cunha and Heckman, 2009, for details).

Recent work has demonstrated that a similar gap for child health as for cognitive skills opens up between socioeconomic groups already at birth and also persists or even increase as the child ages (see Figure 2). The strong coherence between these two patterns indicates that an important part of the intergenerational transmission of socioeconomic status may work through the impact of parents' socioeconomic status on children's health (c.f. Currie, 2009). Poor child health is likely to affect future health and also reduce the accumulation of skills, which potentially puts disadvantaged children on a lower trajectory already from the start. Hence, early life health fits very well into the dynamic framework suggested by Cunha and Heckman.

Figure 2 Children's health and parent's income. *Source*: Case, Lubotsky and Paxon (2002).

However, since e.g. parent's income is most likely an imperfect measure of the actual resources allocated to the child, an important research priority becomes to identify the ways through which parental SES affects child health, and further to determine how sensitive later life outcomes are to health chocks at different stages of development.

These stylized observations broadly summarize the outset of the first two studies in this thesis. The aim of these studies is first of all to provide insights into *how* sensitive future educational attainments and labor market outcomes are to adverse health conditions in early life. These insights are attained by investigating the long-term effects of two major policies which rapidly and unexpectedly changed the early life environment. One of the policies examined improved the early life environment, while the other resulted in worsened prenatal environment. As described further below, these rapid policy induced changes outside the control of the parents facilitates the identification of the effects of early life conditions on later life outcomes.

Essay 1: *Environmental Policy as Social Policy?*

The first study in this thesis examines the long-term effects of an environmental policy intended to protect the environment and public health. In the late 1960s the enormous dispersion of lead into the environment through automobile exhausts became a public health concern. Beginning in 1922 tetraethyl lead started to be added to gasoline since it had been found to improve engine performance. With an increasing number of automobiles, particularly during the 1960s air lead levels increased sharply in Sweden and peaked in 1970. This pattern is reflected in Figure 3 which displays lead

levels in Swedish lake sediments over 4000 years (2000 BC to 2000 AD) (Renberg et al., 2009). The first thing to note in Figure 3 is that although man made lead pollution was not a new phenomenon, leaded gasoline took atmospheric lead depositions to new heights.

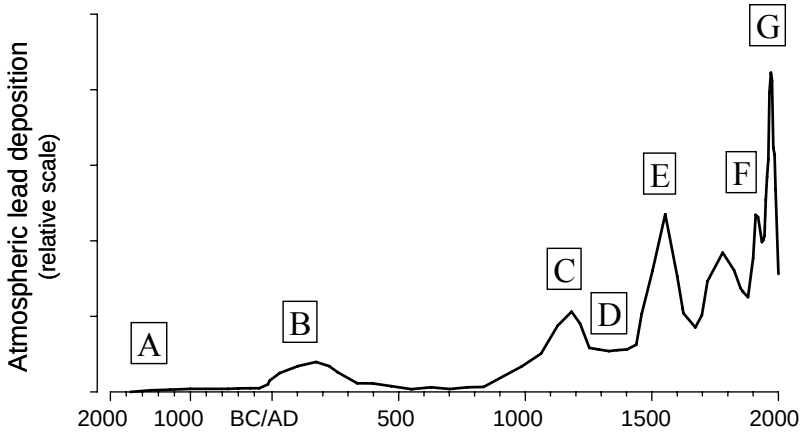

Figure 3 The history of atmospheric deposition of lead in Sweden over 4000 years. The history is based on lead analysis sediment sequences from a large number of lakes and peat bogs. A= first indication of lead pollution, B=Greek-Roman period, C=medieval time, D=the Black Death, E=the Discovery of America, F=industrial revolution, G= 1970's peak in lead pollution. *Source:* Renberg et al. (2009).

From the point of view of my study, another important pattern in Figure 3 is the tremendous drop in lead depositions that took place between 1970 and 2000. This drop is a result of the policy changes that rapidly reduced the maximum amount of lead allowed to be added to gasoline. In Sweden the main reductions of the allowed lead gasoline level took place between 1973 and 1980, and in 1995 there was a total ban on leaded gasoline. The current lead deposition in Sweden is on par with that prevailing during the 16th century.

While the toxicity of lead exposure have been known for a long time the impact of lead exposure in humans was believed to be either fatal or fully reversible. With accumulating evidence on lingering health effects among children that survived lead poisoning, more and more researchers began to study the health effects of ever lower levels of exposure. Today, although there is a consensus on the impact of high levels of lead exposure on cognitive development, there is still a debate on the effects of low levels of exposure on child health.

In the first study I make use of the sharp differences in changes across localities in air lead levels induced by the gasoline lead policy changes to estimate the effects of early childhood lead exposure on long term outcomes. By exploiting variation in lead exposure induced by the gasoline lead policy changes many of the methodological problems of previous studies are mitigated. The study is made possible by linking administrative data on compulsory school grades, military enrollment cognitive ability test scores, educational attainments and early labor market outcomes to a previously unexplored measure of early childhood lead exposure.

The results suggest that even the relatively low (in an international context) levels of lead exposure that the majority of Swedish children experienced in the early 1970s were high enough to affect their future educational attainments and early labor market outcomes. The results further indicate that low socioeconomic status children have gained most by the government mandated reductions in gasoline lead. Hence, environmental policies have the potential to function as social policy and as a redistributive instrument. Suggestive evidence of a lower "threshold" below which further reductions in lead exposure no longer seem to affect adult outcomes is also provided. Importantly, this level is estimated to be located at a considerably *lower* level than the current limit of concern suggested by the US Center for Disease Control and Prevention (CDC). Given that it has been estimated that 40% of all children world-wide have a blood lead level above the limit of concern identified in this study, global lead-reduction programs could potentially yield large benefits in terms of improved future economic outcomes.

Essay 2: *Does a Pint a Day Affect Your Child's Pay?*

The second study focuses on a policy intervention in the late 1960s that intended to shift consumption away from spirits towards lower alcohol content beverages. The policy shift was in part fueled by a contemporaneous discourse promoting a more continental European style alcohol culture, with low liquor consumption and higher consumption of lower alcohol content beverages. The background to this policy intervention was a concern about the believed particularly negative public health effects of liquor consumption. Since 1919 alcohol sales in Sweden has been strictly regulated by means of an off-premise retail monopoly (*Systembolaget*). Between the 1920s and 1955 alcohol sales were further restricted through a ration book system ("Motboken"). After the abandonment of the ration system, alcohol related problems primarily among heavy drinkers increased rapidly. To counter the increasing spirit consumption problems, between the 1960s and 1980s several policies were introduced in order to shift consumption away from stronger spirits towards lower alcohol content beverages.

An example of such a policy initiative was the experiment with free sales of strong-beer in grocery stores in two regions in western Sweden. During the policy experiment the availability of a low alcohol content beverage (strong beer) increased sharply. By increasing the relative availability of a lower alcohol content beverage, the consumption of higher alcohol content beverages was supposed to decrease. However, for those aged 16-20 the policy experiment had a sharply different effect on alcohol availability. Before the policy experiment this age group had no legal possibility to buy strong beer (or other spirits), since in the Systembolaget monopoly stores a minimum age restriction of age 21 was strongly enforced. In grocery stores on the other hand the age limit for purchases of low alcohol content beer was at the time set to age 16; and even that limit was poorly enforced. So for the youngest age group the policy shift implied that the relative availability of *higher* alcohol content beverage *increased* dramatically, rather than the other way around.

The consequences of this shift in availability soon became clear. In the experimental regions the strong beer consumption increased by around 1,000%, while at the same time the consumption of wine and spirits only decreased marginally. Finally, due to worrying reports about increased drunkenness among young people the policy experiment was ended after only 8.5 months out of the originally planned 14 months. On July 16, 1968, the day after the policy experiment ended, Systembolaget, as before, regained its monopoly on off-premise sales of strong beer.

In the second study of this thesis I examine the long-term consequences of the temporary policy experiment on those who were exposed to it while still *in utero*. At the time of the policy experiment relatively little was known about the potential consequences of alcohol consumption during pregnancy. In 1973 Jones and Smith published their seminal work on "the fetal alcohol syndrome", and in 1980 Socialstyrelsen (the Swedish equivalent of Center for Disease Control and Prevention) issued the first recommendations to abstain from alcohol consumption during pregnancy.

The temporary brief upsurge in availability and consumption allows me to compare children born before and after the affected cohort, and to use children born in other regions as additional controls. The results suggest that exposure to the experiment during the first half of the pregnancy resulted in substantially worse future outcomes for the affected cohort in comparison with the surrounding cohorts. In their 30s the children *in utero* during the experiment have completed fewer years of schooling, have lower earnings and higher welfare dependence rates. These results clearly illustrate the importance of *in utero* conditions for future educational and economic outcomes.

Part 2: Social influences of peers at work

The second part of the thesis examines to what extent our decisions and actions are influenced by people whom we interact with on a regular basis. There are many studies on the role of social networks on economic activity, including the transmission of job vacancies, new products, technology, opinions and behaviors; c.f. Jackson (2009) for a review. One reason why such studies are of interest from a public policy perspective is that better knowledge about how behavior and information is transmitted may enable more efficiently designed policies. For example, if transmission of behaviors within a social network is strong enough, it may be sufficient to target only a minor part of the network in order to change the behavior of the whole group. Moreover, an understanding of social interaction effects is important since it gives policy makers and idea of potential long-run equilibrium effects from policy reforms.

The basic level interest in any such study is first of all to establish a reliable answer on *if* behavior is influenced by social contacts. Establishing such a relationship is in fact not an easy task. To see this clearly, consider if we observe that two friends buy the same type of sun-glasses, one after the other. From this observation we can not conclude that one's purchase has affected the other, since there could be many other things that also influence the purchasing decisions of the two friends. For example, they could both have been exposed to the same advertising, be of the same age, have the same education, same style, face the same budget constraints etc. Only when we can observe *all* relevant factors influencing the purchasing decision, then we can construct a test of the influences of the purchase of one of the friend on the purchasing behavior of the other, after taking all the other contributing factors into account. The problem is that we do typically not observe all those other factors, or the timing of the friend's actions. In addition in most settings we do not even know what the relevant social network is.

The last two essays in this thesis focus on social effects within a particular well defined social network that most adults interact with on a regular basis: co-workers. The first study examines the influences that co-workers' work absence decisions have on individual absence. The second focuses on if and how the timing of childbearing among co-workers affects individual childbearing decisions. Both studies use linked employer-employee data to provide answers to these questions. One of the main contributions of these two studies to the current literature is the use of the population wide data on workers and workplaces. To put this into perspective, previous studies focusing on the influence of social networks in the workplace have typically been case studies, where only co-worker interactions within one firm, workplace or occupation have been considered. Naturally, the use of representative population data increases the possibility to extrapolate the results to other settings. Both studies also exploit variation in co-workers' behavior that is

either partly or completely determined by forces outside the control of the employees. This mitigates the two methodological problems discussed above. The individual contributions of each study are further described next.

Essay 3: *Sick of Your Colleagues' Absence?* (with Patrik Hesselius and Per Johansson)

During the 1990s Sweden experienced a sharp increase in the sickness absence rate. The increase in the sickness absence came unexpected and it was suggested that a too stressful work environment following the cut-backs during the economic crisis in the early 1990s had caused the increase in absence. An alternative explanation put forth was that a combination of lenient monitoring by doctors and a high replacement rate in the sickness insurance had caused a shift in the relative utility of staying home vs. being on the job. This had led to a deterioration of work norms and a higher acceptance of "overuse" of the sickness insurance.

The third paper in this dissertation exploits a unique randomized social experiment to find out if, to what extent, and why social interactions, or social norms, affect work absence decisions. The experiment was conducted in the late 1980s and in two regions the entire working age population was assigned into either a treatment group or a control group based on their date of birth. The treatment group was allowed to be absent from work due to illness without needing to provide a medical certificate until the 15th day of absence. The control group needed as usual to provide the same type of certificate on the 8th day of absence.

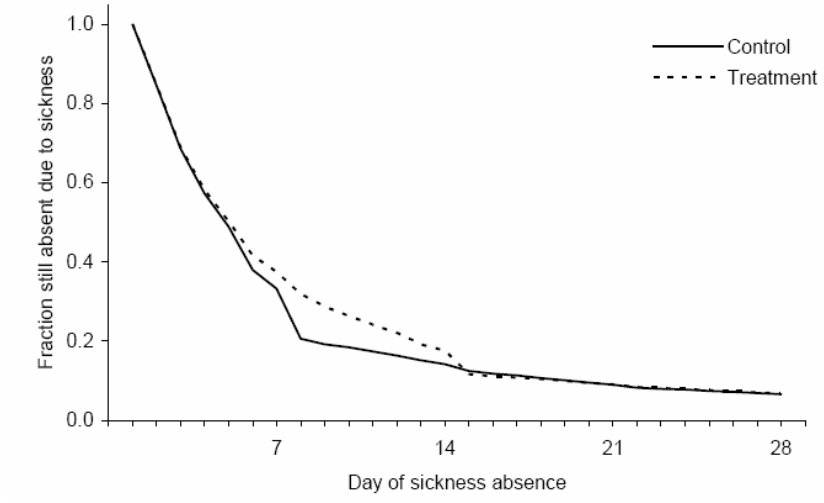

Figure 4 Survival function of treated and control individuals in Göteborg. *Source:* Hesselius et al. (2005).

Figure 4, summarize the evidence in Hesselius et al. (2005) who examine the impact of the experiment on sick leave by showing how the share of individuals that are still absent at a given day evolves during the first 30 days of absence in the control (solid line) and treatment group (dashed line) respectively. As is clear from the figure during the experiment the treatment group prolonged their absence spells considerably compared to the control group.

In the study in this thesis we link each individual employed in workplaces in Göteborg to her workplace and co-workers and thereby we can find out the number of co-workers that were assigned to the treatment group. We then show that having many co-workers assigned to the treatment group increases the worker's absence. Since the number of workers assigned to the treatment groups was randomly determined and assignment to treatment increases work absence, our result provides compelling evidence that co-workers influence individual behavior. We further find that the number of co-workers assigned to the treatment group only influenced those who themselves were assigned to the control group, but *not* the treated workers. This sharp difference in the response to the share of treated co-workers in the treated and the control group allows us to distinguish between various ways through which social interactions at work may affect work absence; ruling out motives such as enjoying joint leisure time, and being more consistent with a fairness effect or related social effect on preferences.

Essay 4: *Businesses, Buddies and Babies: Fertility and Social Interactions at Work* (with Lena Hensvik)

During the 20th century Sweden experienced several large baby-booms and baby-busts (see Figure 5). Several studies have examined the roots of these fluctuations, and the current consensus suggests that the main reason for these sharp fluctuations is that the parental leave system is earnings related. It has been argued that the system gives rise to the following optimal female fertility pattern: get an education, get a permanent job, have your first child, work part time, have your second child within three years (Björklund, 2006). This pattern gives rises to sharp pro-cyclical variation in fertility rate mainly due to the third component; get a permanent job. In bad times the number of permanent positions, and hence employment security if having a child, is reduced and hence women tend to postpone the timing of childbearing until more permanent positions are available.

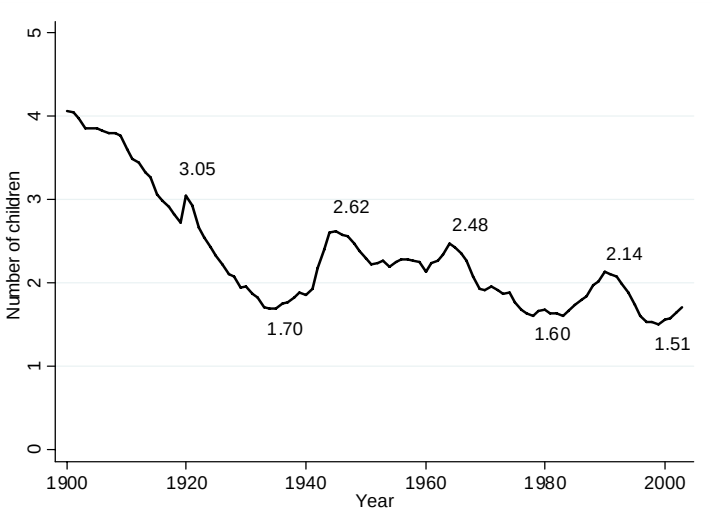

Figure 5 The yearly total fertility rate in Sweden (1900-2004), *Source:* Socialstyrelsen (2005)

In the fourth and final study of this thesis we look at to what extent the fluctuations in the timing of childbearing decisions induced by the parental leave and employment protection rules also are reinforced by social interaction effects between co-workers. That is, part of the effect believed to be caused by the parental leave legislations is potentially explained by spill-over effects in fertility in social networks.

To be able to assess the importance of peer effect in the timing of fertility we summon a seven year long panel on monthly fertility of half of all women in childbearing age and all of their co-workers. In particular we investigate whether childbearing in the recent past among co-workers increases the probability of having a child. Our results suggest that co-workers social influences are important in fertility decisions. We also provide insights of who is likely to affect and be affected. For example, co-workers who are of the same parity are particularly influential for each others childbearing decision, while across-parity peer effects are entirely absent. Co-workers with the *same or higher* education are particular influential, while childbearing among co-workers with *lower* educational attainments are not, suggesting that the relative status of the co-worker is an important component of the peer effect at work.

The results are interesting from a behavioral perspective but also from a policy perspective. Our results suggest first of all that policy evaluations of the effectiveness of a particular program designed to increase fertility are likely to underestimate the full effect of the program if spillover effects are not taken into account. Second, the strong heterogeneity of the spillover

effect suggest that caution is warranted before assuming that a policy which has been found to be effective in one setting will be effective in another setting too. The net effect of the same policy in a different setting partly hinges on the number and strength of the social ties within the targeted groups.

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Essay 1: The Long-term Effects of Early Childhood Lead Exposure: Evidence from the Phase-out of Leaded Gasoline♠

1 Introduction

From the end of the 1960s government air pollution regulations have become increasingly stricter throughout the developed world, which have lead to sharply improved air quality in many countries and regions.[1] Recently, the air pollution reductions following some of these regulations have been shown to improve neonatal health and to reduce infant mortality (Chay and Greenstone, 2003a; Currie and Neidell, 2005; Lüchinger, 2009). However, these and previous studies have not been able to asses the potential long run effects of exposure to poor air quality in early childhood on the surviving infants and children.[2] Since children who are on the life/death margin at birth only constitute a small fraction of all children, the total cost of air pollution in terms of its impact on child health could potentially be much higher if also the sub-clinical effects on the general population of children are taken into account. For example, many of the pollutants released are neurotoxicants that potentially impair children's development in early life even at low levels

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[1] See e.g. Giussani (1994) for a thorough report of the impact and history of pollution regulations in the UK. In the developing world along with increased economic growth pollution levels are still increasing rapidly. China stands out as a stark example where air pollution levels as measured between 1983 and 1993 were up to 5 times higher than what was observed in the US *before* the passage of the 1970 Clean Air Act Amendments (Almond et al., 2009).

[2] A notable exception is Reyes (2007) who focuses on the relationship between lead exposure in childhood and violent crime rates. I describe her study (and the differences to this study) in detail below.

of exposure. Such effects, even if not apparent at birth or in early childhood, can cause psychological or behavioral problems that first become apparent later on. Moreover, if skills beget skills, as suggested by Cunha and Heckman (2007) even if the direct health damage inflicted by pollutants early in life is fully reversible over time, long-run outcomes could still be affected through dynamic complementarities in human capital accumulation.

In order to shed light on how exposure to poor air quality early in life affects adult outcomes this study focuses on the causal impact of early childhood air lead exposure on subsequent cognitive skills, educational attainments, and labor market outcomes among young adults in Sweden. By merging unique data on local air lead levels in early childhood with comprehensive population micro data, it is possible to follow all children in nine birth cohorts from birth throughout school and examine their experiences on the labor market as young adults. The outcomes considered include scholastic performance (Grade 9 GPA), cognitive ability test scores (males, age 18), educational attainments and early social and labor market outcomes. These outcomes have previously been shown to be predictive of subsequent outcomes throughout the life cycle, and should therefore be particularly interesting from a public policy perspective.

The local lead pollution data stems from a previously unexplored data source. Since the early 1970s the Swedish environmental protection agency has used moss (bryophyte) samples covering the whole of Sweden to examine regional differences and trends in heavy metal air pollution levels. Mosses are particularly useful as air pollution biomonitors since they lack roots and therefore solely absorb heavy metal depositions from the air. The use of moss as biomonitors for ambient heavy metal air pollution is well established and the program has gradually been expanded; first to the rest of the Scandinavian countries, and since 1995 most other parts of Europe. In a companion study Nilsson et al. (2009) show that moss lead levels is a good predictor of blood lead levels in children.

Although a consensus exists on the health impact of high levels of lead exposure on adult health, the association between lower levels of lead exposure in childhood and cognitive development is still under debate (Canfield et al., 2003; Lanphear et al., 2000). The main reason is that lead exposure is not randomly distributed across locations, and hence confounding is a serious concern as highlighted by e.g. Bellinger (2004a). For example, parents with higher incomes or preferences for cleaner air are likely to sort into areas with better air quality and hence their children are less likely to be exposed to high levels of lead pollution. Failing to account for residential sorting of this kind can result in an upwardly biased estimate of the effect of lead exposure on children's subsequent outcomes. On the other hand, pollution tends to be higher in densely populated areas and at the same time metropolitan areas often attract highly educated parents with more resources, contain bet-

ter access to quality child care, schools, health care and other amenities that are positively associated with adult outcomes. Such local amenities could in turn result in an underestimated role of childhood lead exposure if not properly taken into account.

To mitigate these and similar concerns this study focuses on children born from the early 1970s until the mid 1980s. The reason is that during the 1970s, along with many other developed countries, Sweden initiated a gradual phase-out of leaded gasoline in order to protect the environment and public health. In Sweden, the main reduction in gasoline-lead levels occurred between 1973 and 1981 when the maximum allowed lead level per litre of gasoline dropped by 79% (Table 1). Since gasoline lead was the main source of lead exposure in the general population[3], as shown in Figure 1, children's blood-lead levels (B-Pb) decreased drastically from the 1970s until the mid 1990s when leaded gasoline finally was banned.[4]

Table 1 Changes in maximum allowed gasoline lead levels

Date of policy change:	Maximum lead content:
1 Jan 1970	Max 0.7 g/L (2.65 g/gal)
1 Jan 1973	Max 0.4 g/L (1.51g/gal)
1 Jan 1980	Max 0.15 g/L (0.56g/gal) for regular
1 Jan 1981	Max 0.15 g/L (0.56g/gal) for premium
1 Jan 1986	Leaded regular gasoline is prohibited
1 March 1995	Total ban on lead for all gasoline grades

Source: The Swedish Petroleum Institute.

Due to large differences in initial lead levels the phase-out of leaded gasoline induced substantial variation across localities in the reduction of lead exposure. In the main analysis I exploit the differential changes in early childhood lead exposure for the cohorts born between 1972 and 1984. I compare changes in outcomes for children born in municipalities experiencing large reductions in lead exposure with changes in outcomes of children born in municipalities with only minor changes in air lead levels. By exploiting these differential changes in exposure across birth cohorts within the same

[3] 80% of the air lead levels in the late 1980s where due to traffic (MOENR, 1994).

[4] Similar large reductions in blood lead levels associated with the phase-out of lead from gasoline have been documented in many countries (c.f. Thomas et al., 1999). Other sources of lead exposure such as leaded paint was banned in the early 1920's and are therefore believed not to have caused the reductions in blood lead levels during the 70's and 80's. The costs associated with phasing out lead have been shown to be low, c.f. OECD (1999). After 1995 children's blood lead first seemed to stabilize at around 2µg/dL, but since 2000 it has continued to decrease, albeit at a slower speed in absolute terms, c.f. Strömberg et al. (1995, 2001, 2008).

municipalities unobserved time-invariant differences between the municipalities is taken into account.

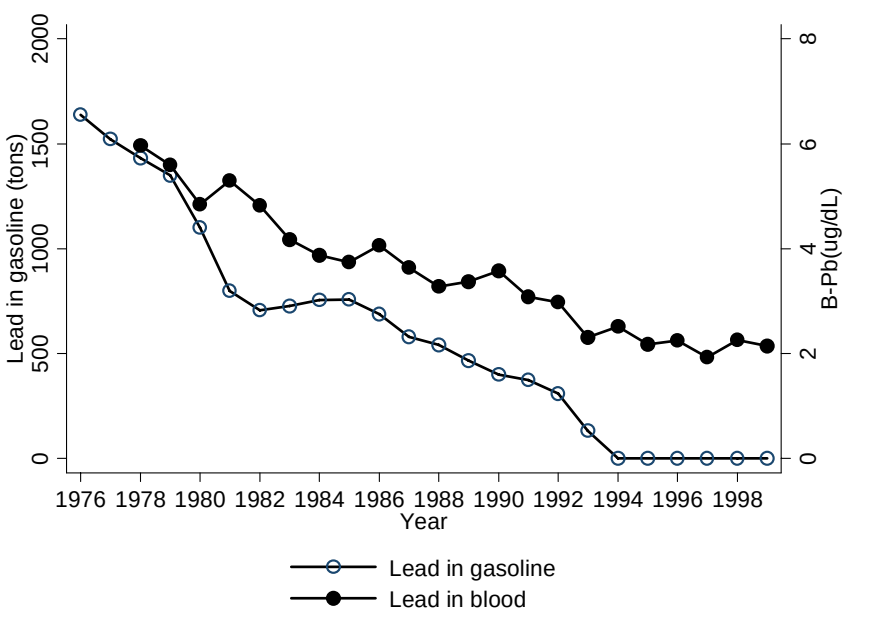

Figure 1 Mean blood-lead levels among primary school children and tons of lead added to gasoline 1976-99. *Source*: Strömberg et al. (1995) and Strömberg et al. (2003).

The importance of taking such unobserved characteristics into account is highlighted by a cross-sectional analysis showing that several *predetermined* parental characteristics that are strongly correlated with children's adult outcomes also are strongly correlated with their children's lead exposure. That is, higher lead exposure for the *child* is associated with lower educational attainments among the *parents*. This result suggests that cross-sectional estimates of the role of early childhood lead exposure are likely to overestimate the relationship between early lead exposure and subsequent outcomes due to omitted variable bias. In contrast, the within municipality variation in lead exposure induced by the gasoline lead level regulations is *not* significantly correlated with the predetermined parental characteristics, which provides support for the validity of the main identification strategy. In addition, besides several important individual, parental and municipality of birth control variables the data also contain unique family identifiers which enable a comparison of outcomes of full siblings with different early childhood lead exposure levels. By comparing differences in adult outcomes among siblings

it is possible to take into account additional unobserved characteristics which the siblings have in common and that also influence adult outcomes.

The World Health Organization estimates that globally 20% the urban children have blood lead levels exceeding 10μg/dL (Fewtrell et al.,2003); the level above which the Center for Disease Control and Prevention (CDC) recommends that actions to reduce lead exposure should be initiated.[5] Naturally, the relevance of the current limit of concern hinges on the relative effects of lead exposure above and below the limit. Since the average blood lead levels of Swedish children at its peak in the early 1970s on average already were *lower* than 10μg/dL, the Swedish experience is particularly interesting since it provides a direct test of the relevance of the concurrent limit. The combination of population micro data, relatively low initial exposure levels and considerable differences in changes in exposure induced by government regulations provides a compelling setting to search for a threshold of the relationship between early childhood lead exposure and adult outcomes.

The main results suggest that low levels of lead exposure early in life have both statistically significant and economically important effects on future educational attainments and labor market outcomes. A key finding is a clear nonlinear relationship between local air lead levels in childhood and long-term outcomes at the relatively low levels of exposures considered. Above an estimated municipality average early childhood blood lead level of 5μg/dL, reductions in lead exposure have a consistently positive and significant impact on long-term outcomes. Below this level reductions no longer seems to affect adult outcomes in a consistent or significant direction. Importantly, the results are insensitive to a number of specification changes, such as the inclusion of family fixed effects, measures of other pollutants, measures of lead exposure later on in childhood, and various sample restrictions.

Further analysis reveals that children from poorer families seem to have benefited most from the gasoline lead reductions. Although data constraints prohibit a full differentiation of the mechanisms behind the socioeconomic status (SES) differences, a key finding is that residential segregation within municipalities (and thereby potentially differential neighborhood lead exposure levels) *does not* seem to be able to entirely explain the SES-gradient in the effects of lead. Instead differential avoidance behavior, differences in sensitivity to the same levels of exposure or differences in the ability to

[5] In the US, approximately 310,000 children aged 1-5 years have higher blood lead levels than the level of concern (CDC, 2005), and average childhood blood lead levels in the adult US population will have decreased from 10μg/dL in 2002 to below 3μg/dL in 2018 (Reyes, 2007). The acceptable limit has been revised downwards several times since the 1970 level of 60μg/dL as a result of increasing evidence of an association between lower lead levels and health.

compensate for the effects of early lead exposure seems to be more plausible explanations for the SES-gradient. Whichever of these pathways that matters most, these results indicate that environmental policies may be able to reduce the intergenerational correlation in economic outcomes and potentially function as a redistributive instrument, since it seems to disproportionally improve long-term outcomes among low SES children.

The remainder of the paper is structured as follows: section 2 gives a brief summary of previous studies linking childhood lead exposure to adult outcomes. Section 3 describes the data. In section 4 the empirical strategy is explained and section 5 presents the results. Sections 6 and 7 discuss the policy implications and conclude.

2 Early childhood lead exposure and adult outcomes

Exposure to lead has previously been linked to a number of adverse effects on health. Prospective cohort and cross-sectional studies of children have demonstrated associations of lead exposure, measured by various indices, and cognitive skills. In a series of meta-analyses, using data from some of the cross-sectional studies of school-age children (Skerfving and Bergdahl, 2007), it was concluded, that a decrease of one (1) IQ point was seen for every 2-4μg/dL increase in concurrent blood-lead levels (B-Pb).

There are however good reasons to suspect that lead exposure *in utero* or in early childhood could have a stronger effect compared to the effect of blood lead levels later on. First, the developing nervous system is more vulnerable to the toxic substances than the mature brain (Dobbing 1968; Schwartz, 1994; Lidsky and Schneider, 2003). Secondly, this sensitive period in human development coincides with a period of particularly high uptake of lead. B-Pb levels typically follow an inverted u-shaped pattern between ages 6 and 60 months, reaching its peak around age 24 months due to the intense hand-mouth activity common at these ages (Canfield et al., 2003). In a recent study a 10μg/dL decrease in B-Pb was estimated to increased cognitive ability at age 3 by 7.4 IQ points (cf. Lanphear et al., 2005).

Besides the effects on cognitive development, an association between early lead exposure and anti-social behavior has also been found. For example, using time-series data from early to late 20th century Nevine (2000) find that the consumption of lead in the general population in the first year of life co-varies with teenage pregnancy (18 years later) and crime rates (20 years later). Finally, lead exposure is also associated with poorer pre- and postnatal

growth, hearing impairment, reduced effectiveness of the kidneys, and lower skeletal growth among children.[6]

However, all of these estimates stem from observational studies, and while many of the studies try to account for important potential confounders e.g. maternal education, home environment etc., it is important to realize that unless *all* factors correlated with both lead exposure and e.g. cognitive test scores are accounted for, the estimated impact of lead will be biased; most likely upwards.[7] [8] The bias is furthermore likely to become more important when studying the relationship between lower levels of exposure and less obvious non-clinical outcomes, such as cognitive development. While many randomized control studies on animals supports a causal link between lead exposure and cognitive ability, it is not evident that the results from such studies are easily generalized to human subjects.[9]

A notable exception is Reyes (2007) that addresses the omitted variables problem by focusing on the impact of exogenous state-year specific changes in gasoline lead levels in the US on state level violent crime rates around 20 years later. The panel data employed allow for controls of fixed unobserved state-specific characteristics correlated with both childhood lead exposure and crime. Reyes finds a strong relationship between state level lead exposure in early childhood and state level violent crime rates, suggesting that the sharp reduction in lead in gasoline following the Clean Air Act Amendments in the early 1970s could explain as much as 50% of the sharp drop in violent crime that occurred in the US during the 1990s.

Although compelling, Reyes' analysis suffers from the use of aggregated data since it is not known whether the individuals exposed in early childhood are actually still living in the state where they were born 20 years later when the outcomes (also measured at the state level) are realized. Since in the US between 25-40% of the children migrate from the state of birth before age 22, this is clearly a source of concern. Reyes attempts to account for interstate migration rates, however, since it is not obvious how early childhood lead exposure affects migration propensities it is not clear to what extent correcting for general migration patterns solves this problem. Moreover, the mechanisms through which early childhood lead exposure affect crime remains unexplored in Reyes' study.

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[6] Bellinger (2004b) provides a thorough review of the literature on the association between childhood lead exposure and childhood outcomes.

[7] The importance of omitted variable bias has lately been recognized also in the epidemiological literature (cf. Bellinger, 2004b).

[8] Similarly the main problem with using time-series data is that there are many things which possibly co-vary with both lead consumption during childhood and unsocial behavior later in life.

[9] For example they do not take avoidance behavior into account such as staying indoors on days with high pollution (see Neidell, 2004).

This study distinguishes itself from and complements Reyes study in at least two important ways. First and foremost this study focuses on children with blood lead levels *below* the concurrent 10µg/dL level of concern. The subjects in Reyes' study on violent crime were estimated to have a blood lead level between 10 and 20µg/dL. Hence, the results from this study are potentially more informative about the long-term effects of early childhood lead exposure at levels that are still common in the US and in many other countries today.

Secondly, an improvement of this study compared to previous work using aggregate data is that the data employed follow individuals rather than states/counties/cities across time, but still makes use of the plausibly exogenous changes in local air lead levels induced by government regulations. The comprehensive population micro data derived from administrative registers enable me to follow children from birth throughout school, and to examine their early experiences on the labor market virtually without any attrition, which is typically a common problem in prospective studies.

3 The data

3.1 Measuring lead exposure in childhood

The measure of local lead exposure levels used in this study has not previously been explored in the literature. With a bi-decennial interval since 1975 the Swedish Environmental protection agency has monitored heavy metal air pollution using a nationwide grid of moss (bryophytes) samples. The use of mosses as biomonitors of heavy metal pollution was developed in Sweden at the end of the 1960s in pioneering work by Rühling and Tyler (1968, 1969) and is by now well established.[10] On a national scale, the use of moss as air pollution monitors expanded to Norway and Finland in 1985, and since 1995 28 countries participate in a bi-decennial moss survey designed to study regional differences and time trends in heavy metal deposition using around 7,000 sample locations throughout Europe in each round.[11]

Moss is particularly suitable for biomonitoring of air pollution levels for several reasons. (1) The lack of roots implies that moss solely depend on surface absorption of pollution through precipitation or dry deposition of airborne particles.[12] (2) The absorption and retention of metals is high, and

[10] See Onianwa (2001) for a recent and comprehensive review of this literature.
[11] The European biomonitoring program is described in greater detail in Rühling, ed., (1994).
[12] The close-set leaves of the carpet-forming moss species enable them to filter the air efficiently. The contact with the underlying mor layer and soil is negligible for most species, and

(3) it can be found in abundance in nearly all environments. (4) The annual growth of the moss species included in the surveys is easily distinguishable and, since the transportation of metal between the yearly growth segments is minimal, it is possible to distinguish temporal patterns in pollution levels.

Biomonitors also have several advantages over regular pollution monitors; the main being its simplicity, accuracy and low cost which allows a large number of sites to be included in the surveys. In the Swedish moss survey samples from around 1,000 locations are collected. Additionally, unlike regular pollution monitors which often go in and out of operation as a response to prevailing changes in local pollution levels, the moss samples are collected all over Sweden using a systematic procedure. The sampling sites are chosen carefully; they should be located at least 300 m away from bigger roads and closed residential areas, or at least 100 m from smaller roads and single houses. At each site 5 to 10 subsamples are collected in an area of approximately 100 m^2. From each sampling site the growth over the last three years of all sub-samples is pooled and analyzed and hence reflects the average air lead level during the three years preceding the date of sampling.

This study focuses on the samples collected in 1975, 1980 and 1985, which reflects the average lead deposition levels during the years 1972-1974, 1977-1979, and 1982-1984. The selection of these years is made for two reasons. First, between these years the maximum allowed grams of lead per litre of gasoline decreased particularly sharply. Second, since the main outcomes focused on are educational attainments and labor market outcomes it is necessary to restrict the sample to those cohorts that have completed their compulsory education and for whom the exposure level in early childhood is known.

Although the principles for choosing the location of the specific sampling sites and how to collect the samples is well defined it should be made clear that the sampling locations are not always identical across the survey years.[13] Hence, in order to construct a measure of municipality lead exposure I fol-

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the risk of contamination by metals from the substrate is thus insignificant. A non-negligible part of the lead deposition levels has its origin in other regions or even further away (Rühling and Tyler, 1973). While the analysis in this paper takes into account the fixed characteristics of localities (such as the yearly precipitation rate, distance from the contributing pollution sources in other parts of Europe etc.), it is still likely that parts of the variation in lead exposure are due to the phase-out of leaded gasoline in other parts of continental Europe. This implies that part of the effects provided here reflect the total impact of phasing-out lead from gasoline, not only in Sweden, but in other parts of Europe as well. For ease of exposition the total deposition level, that is the sum of local air lead levels and deposition from precipitation is simply denoted air pollution levels.

[13] However, it should be remembered this is not unique to the moss biomonitoring of pollutants. Traditional pollution monitors also go in and out of operation. Presumably to a higher extent due to changes in pollution than in the case of moss biomonitors.

low a similar approach as Neidell (2004) and Neidell and Currie (2004): first I calculate the centroid of each municipality. Then I measure the distance between the sampling site and the center of the municipality. Finally I calculate a weighted average air lead exposure level using the lead levels at the five closest sampling points (i.e. altogether between 25 and 50 samples), with the inverse of the distance to the sampling point as weight. This is done for each time period and municipality.

Figures 2 and Figure 3 display the lead concentrations in the municipalities in 1975 and 1985 using this definition of exposure. Figure 4 displays a kernel density plot of the distribution of the municipality lead exposure levels as measured in 1975, 1980 and 1985. From these figures it is clear that entire lead exposure distribution shifted drastically in between the years. Similarly a *within* municipality comparison of the lead levels clearly display the tremendous differences across municipalities in the reduction of lead exposure that took place between 1975 and 1985 (Figure 5). These sharp within municipality differences in the reduction of early childhood lead exposure across the cohorts is a key feature of the main identification strategy in this study as discussed further below.

Three important questions regarding the local lead exposure definition should be addressed before proceeding with the empirical analysis. The first concerns the arbitrary choice of using the five nearest sampling sites to define municipality of birth lead exposure. To test the sensitivity of the analysis to this assumption I have also used the 3 nearest sample points instead. The differences between these definitions are small and they are highly correlated (corr. coeff.> 0.9). [14]

Secondly, to get an idea on how accurate the five nearest sample approach is in predicting the actual exposure level, I estimate the level of lead at each sampling point, as opposed to municipality, pretending as if the sampling point of interest was not there. That is, I estimate the air lead level at a given sampling point based on the air lead levels at the five nearest sampling points. I do this for all sampling points in the data, and then calculate the correlation between the actual and the estimated air lead levels. The correlation between these two measures is high (corr. coeff.=0.80), which clearly indicates that the pollution assignment method employed provides reasonably accurate predictions of actual air pollution levels, and suggests that it does not seem to be a major concern for the analysis.

[14] The results on adult outcomes when using the 3 nearest samples definition rather than the 5 nearest sample definition are qualitatively similar.

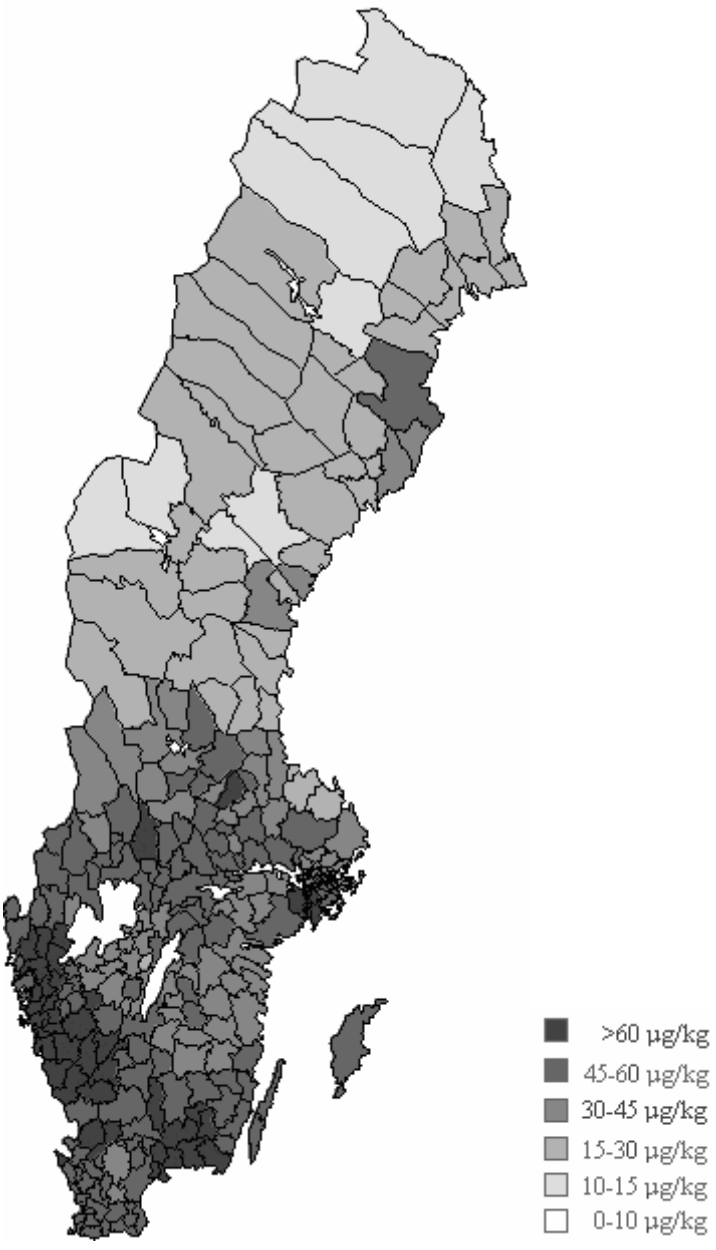

Figure 2 Moss lead levels (µg/Kg) in Swedish municipalities in 1975.Source: Authors calculation using data from the Swedish Environmental Research Institute (IVL).

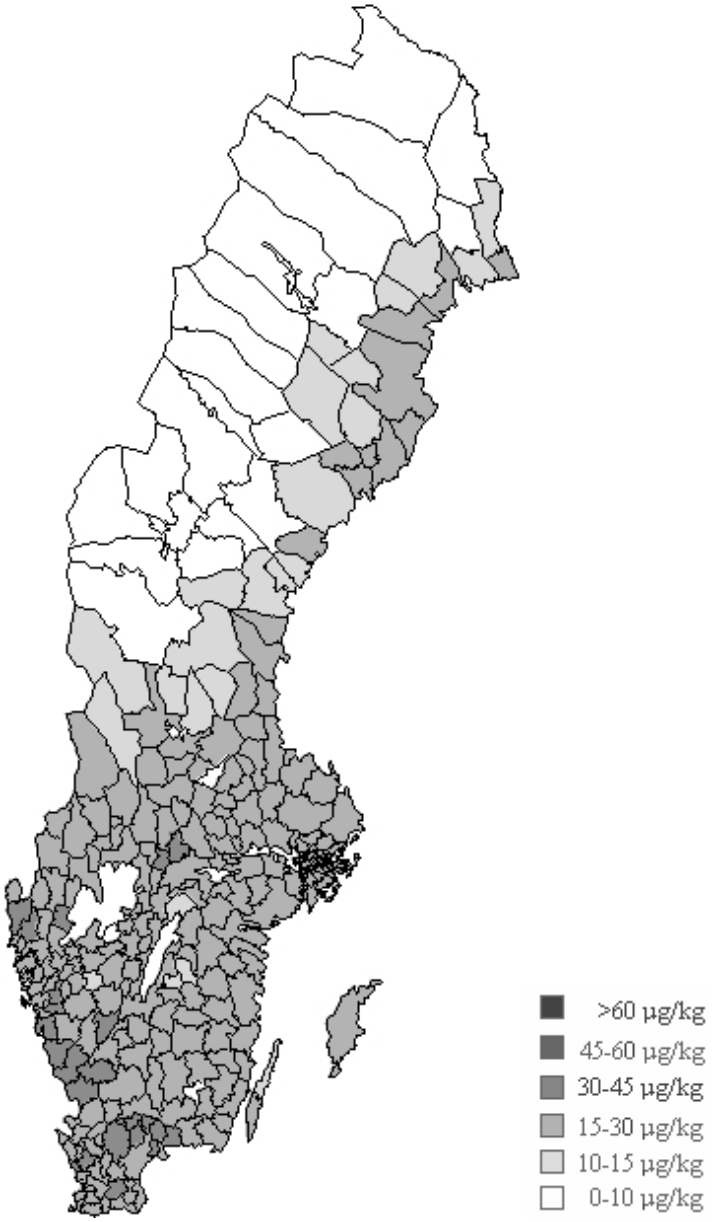

Figure 3 Moss lead levels (µg/Kg) in Swedish municipalities in 1985.Source: Authors calculation using data from the Swedish Environmental Research Institute (IVL).

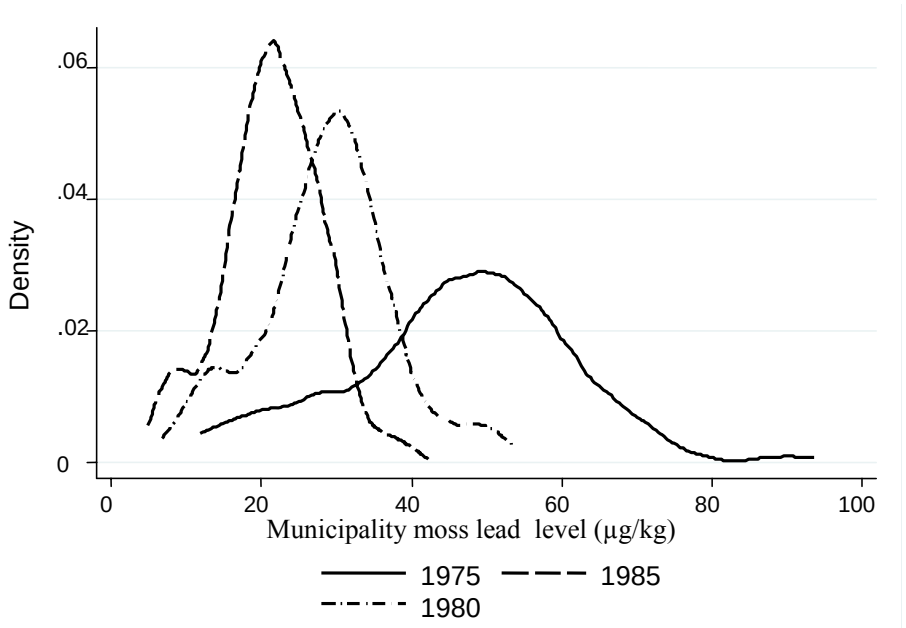

Figure 4 Kernel density distributions of moss lead levels in Swedish municipalities

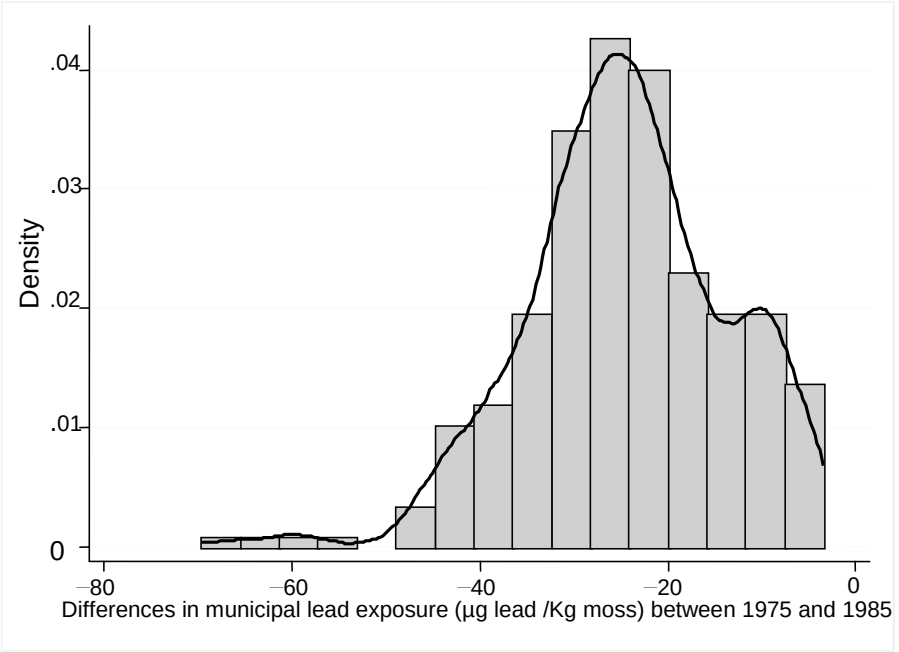

Figure 5 The distribution of within municipality lead level changes between 1975 and 1985

Finally, as in any study using data on local exposure levels rather than individual exposure an important question is how well the lead levels in moss predicts the actual blood lead levels in children. Unfortunately there exist no data that monitors the trends in blood lead levels among young children or the population in general in Sweden during this time period. However, since 1978 in two municipalities in southern Sweden, blood samples have been collected with a two year interval from about 120 primary school children (age 7-10) per annum. The results from these studies on the trends in childhood lead exposure are described in detail elsewhere (Strömberg et al., 1994, 2003). At the same time the department of environment (Miljöförvaltningen) in one of these municipalities (Landskrona) has at three time points (1984, 1995 and 2006) collected around 50 moss samples all over the municipality following the same procedure as the national monitoring program.[15]

Most previous studies using aggregate data on pollution have been forced to assume that local air pollution exposure is a valid proxy for actual exposure. However, the two datasets in Landskrona provide a unique opportunity to assess the strength of the relationship between local air lead exposure and children's lead exposure. Nilsson et al. (2009) do precisely this and link the average lead level of the five nearest moss samples to the children using their home addresses and estimate the elasticity between lead in moss and lead in children. Controlling for important individual characteristic, time and locality fixed effects they establish a Blood-Pb/Moss-Pb elasticity for the pre-gasoline lead free period (i.e. before 1995) of 0.44. This elasticity implies that a 10% reduction in Moss-Pb corresponds to a 4.4% decrease in primary school children's B-Pb. This estimate implies that the drop in air lead exposure between e.g. 1982 and 1994 can account for as much as 50% of the change in children's blood lead levels. Appendix A, gives a further review of the main findings in Nilsson et al. (2009).[16]

However, it is important to remember, as found in many previous studies, that the relationship between environmental lead exposure and very young children's blood lead levels is significantly higher. For example, Reyes (2007) finds that the elasticity between lead in gasoline and blood lead in

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[15] I am great full to Olle Nordell Landskrona miljöförvaltning for collecting and providing me with the data on lead in moss in Landskrona municipality. See also Nordell (2007) for (a Swedish) description of the sampling procedure and description of the moss data from Landskrona. To attain the moss lead levels which are comparable to those from the National survey it is necessary to calibrate the lead levels with a factor of 0.44 as described by Folkeson (1979), since they are measured in two different moss species.

[16] Assuming that this estimate functions as a valid proxy for the relationship between lead in moss and lead in children for the general population of children, in the last part of this paper I back out the elasticity between the adult outcomes focused on here and the children's blood lead levels.

children aged 0-6 is around 30% higher than among children aged 6-12, which is important to remember later on when trying to estimate the relationship between the adult outcomes and early childhood blood lead levels.

3.2 Outcome measures

The individual outcome data stems from two data sources in the Educational database at the Institute for Labor Market Policy Evaluation (IFAU) in Uppsala.[17] In the main analysis I use all individuals born in Sweden in the three years prior to the year the moss samples were collected; that is all those born in 1972-1974, 1977-1979 and 1982-1984. Again the reason for the 1972 and 1984 constraint is that many of the individuals born after 1985 are less likely to have finished schooling in 2004 and that the first lead exposure measure available reflects the situation in 1972-1974 (i.e. mosses collected in 1975). As explained above the lead levels in the mosses measure the local lead deposition when they were between 0-3 years old. As discussed above, this age interval corresponds to a particularly sensitive period in human development and a period with particularly high lead uptake rates.

It is important to recognize that this assignment of exposure does not reflect an exact definition of timing of exposure for the cohorts. For example, taking the measure of air lead in moss literarily, for children born in June 1972 the lead exposure levels approximately reflect average lead exposure from the second trimester (starting January 1972) until about age 30 months (December 1974). For children born in June 1974, the moss lead exposure level reflects the exposure from conception until age 6 months. To check whether the results are sensitive to this deviation of exposure within cohorts, separate regressions including only the children born in the middle of each exposure measure period, i.e. those born in 1973, 1977 and 1983, where also tested, which yielded very similar results.[18] For these children the exposure levels reflect the average exposure level from conception until age 2. Finally, I focus on children who were living in Sweden in 2004, who have completed compulsory schooling (9 years education) and who were born in Sweden, so that their municipality of birth (and hence childhood lead exposure) is known.

The outcome variables considered are grade point averages in grade 9 (GPA at end of the 9 year compulsory school), whether the GPA was below (above) the 25%-tile (75%-tile) of the GPA distribution, the cognitive test

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[17] I am grateful to Björn Öckert for assembling the data and for sharing it with me.

[18] While the estimates of the parameters are essentially unchanged the precision also decreases since the sample is reduced by 2/3. These results are not reported but available upon request from the author.

score as measured for all men at military enrollment, whether the score was below (above) the 25%-tile (75%-tile) of the cognitive test score distribution, whether having completed high school, ever enrolled in college education, the number of years of schooling completed, (ln) labor market earnings, welfare dependency and finally whether or not having become a teenage mother. The labor market and educational outcomes outcome variables are measured in 2004 (at ages 20-32). All of these outcomes have previously been shown to be predictive of other outcomes throughout life.

The military enrollment test scores are Stanine (Standard Nine) test scores which is similar to the AFQT in the US. The score is an evaluation of cognitive ability based on several subtests of logical, verbal and spatial abilities and a test of the draftees' technical understanding. The results on these subtests are combined to produce a general cognitive ability ranking on a 1-9 scale. All men were obliged by law to go through the military draft. However, due to reforms in the military enrollment procedures affecting the latest cohorts (i.e. those born during the 80s) the cognitive outcomes are only used for those born before 1980. Before that about 90 percent of all men in each cohort went through the draft procedure almost exclusively (99%) at age 18 or 19.[19] The test score is percentile ranked within each cohort of draftees to account for any minor changes in the tests over time.[20] Teenage motherhood is included as an outcome since it has previously been shown to be correlated with early childhood lead exposure using time-series data (see Nevin, 2000). Table B1 provides the definitions of the outcome and control variables and descriptive statistics for the outcome variables, individual and parental characteristics as well as some municipality of birth background characteristics.

4 Empirical method

4.1 Empirical model

As discussed above a number of factors complicate the estimation of causal effect of early childhood lead exposure on adult outcomes. Under the assumption that the effects of the covariates are additive and linear it is possible to remove the influences of many potential confounding factors by estimating a linear regression model that accounts for unobserved differences in municipalities and cohorts,

[19] In principle only the physically and mentally handicapped was exempted.
[20] The test has been subject to evaluation by psychologists and appears to be a good measure of general intelligence (Carlstedt, 2000).

$$y_{ijtc} = \alpha + \beta_1(\text{Lead exposure})_{jc} + X_{ijtc}'\beta_x + \gamma_t + \phi_j + \varepsilon_{ijtc} \quad (1)$$

where y is either a continuous measure or an indicator variable of the adult outcome of individual i, born in municipality j in year t and belonging to cohort c where $c \in \{1975,1980,1985\}$. Lead exposure is the continuous early childhood lead exposure measure (µg/kg moss) as described above; X is a vector children's own, parental and municipality of birth characteristics. They are indicators for child sex, month of birth, number of siblings, year of compulsory school graduation, maternal educational attainments (7 levels), maternal age at birth, indicators for parental earnings (quartiles) for sum of parental earnings in 1990, the average income of the parents in the municipality, share of parents that have completed high school/ university, the share with missing paternal indicator, the share of boys in the same cohort, cohort size, and the average family size. Finally γ_t and ϕ_j are nine year of birth and 287 municipality of birth specific effects respectively. ε_{ijtc} is the error term.

The inclusion of X controls for many of the important background characteristics that varies across cohorts and municipalities and the municipality of birth specific fixed effects ϕ_j accounts for persistent differences between municipalities that could be correlated with the children's future outcomes and childhood lead exposure. The month of birth dummies is included since both adult outcomes, but potentially also early childhood lead exposure can be influence by the season of birth. The nine year of birth dummies γ_t control for all general trends in the outcomes of interest.

The main parameter of interest is β_1 and the main hypothesis to test is whether $\beta_1 = 0$, that is if early childhood lead exposure has no effect on adult outcomes. Under the identifying assumption that the error term is uncorrelated with the lead exposure, $\widehat{\beta}_1^{OLS}$ reflects the causal impact of the local air lead level (an additional gram of lead per Kg moss) has on subsequent adult outcomes. That is after conditioning on individual, parental, observable and fixed unobservable municipality characteristics, the main identifying assumption requires that there are no unobserved characteristic that are correlated with both childhood lead exposure and adult outcomes.

4.2 An indirect test of the main identifying assumption

Although this identifying assumption is fundamentally untestable, it is possible to indirectly assess the plausibility of this assumption by looking at the correlation between factors that are expected to be correlated with adult outcomes of the child but not with the child's lead exposure if the identifying assumption is valid. It is particularly informing to contrast this correlation in a traditional cross-sectional analysis with the within-municipality analysis, since this may reveal how well the within municipality analysis can reduce the potential bias induced by omitted variables.

Candidate factors qualifying for such a test directly available in the data are predetermined parental characteristics; such as parents educational attainments. These parental characteristics can be considered to be predetermined in the present context since the lion share of the parents (>95 percent) were born before 1960, i.e. before environmental lead exposure became a serious environmental problem in Sweden.[21] Therefore assessing whether predetermined *parental characteristics* are correlated with municipality lead levels during their *children's early childhood* should give a hint of whether omitted variables (associated with parents characteristics) is a major concern in the within municipality analysis.

The first panel of Table 2 first provides estimates of β_1 from a regression of the parents' educational attainments (or earnings) on their children's early childhood lead exposure using data on all cohorts but without controlling for municipality fixed effects.[22] The columns present the estimated impact on whether at least one of the parents had completed high school, university, the total parental earnings and finally the same outcomes for the mother and father separately, and an indicator for if the father is not known/missing. The model only controls for year of birth and cohort size of the child. In this cross-sectional analysis many of the parental predetermined characteristics

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[21] Using mosses collected from 1860 until 1968 Rühling and Taylor (1968) show that in the southern part of Sweden (the most highly exposed in the present sample) the increase in lead concentrations in moss were restricted to two distinct periods: a first increase towards the end of the nineteenth century, and a second increase during the 1960s (80-90 µg/kg in 1968). Before that the average lead level in Skåne (the southernmost regions in Sweden with the highest lead level in the data used in this study) was around 40-50 µg/kg moss. They conclude that the first rise is probably due to industrial pollution, possibly due to the increase use of coal, and that the second rise is *more than likely* caused by the rapid increased use of lead gasoline. The exact same pattern is found in a study by Rehnberg et al. (2000) who use extraordinary data on lead levels in lake sediments to examine regional trends in lead depositions in Sweden over a period of *4,000 years*. In particular the lead concentrations in the lake sediments increased by 50% between 1960 and the peak year of 1970. Hence the parents of the children were exposed to relatively low levels during their own childhood and therefore the lead levels in during the parents childhood is not expected to be able to influence the children's adult outcomes to any large extent.

[22] The parent's outcomes are measured in 1990 when the average mother was 40 years old.

are statistically significant and generally indicate that poorer educational and labor market outcomes of the parents are strongly correlated with their children's childhood lead exposure levels.

In the second panel of Table 2 the same set of estimates is presented when only using the within municipality variation in childhood lead exposure. After controlling for municipality specific effects, for the majority of the outcomes, the magnitude of the relationship between lead and predetermined parental characteristics decreases typically by at least an order of magnitude, sometimes changes sign and are no longer statistically significant. The exception is father's high school completion which switches sign compared to the cross-sectional analysis and now indicates that higher exposure is correlated with higher probability of having a father that has completed high school education. However, it is only marginally statistically significant, and given the number of outcomes considered it is not surprising that at least one coefficient is significant at the 10% level.

This exercise highlights the problems with using cross-sectional research designs to make causal inferences. It furthermore provides supports for the validity of the main identifying assumption since the within municipality analysis seems to be able to reduce the importance of observed and hence also most likely unobserved omitted variables considerably.

Table 2 Parents predetermined characteristics and their childrens' lead exposure

PANEL A:	Parents Earnings	Parents High school	Parent College	Mom's College	Mom's High school	Father College	Father High School	Father earnings	Young mother
Lead exposure (µg/Kg)	.0004	-.0011**	-.00035	-.00023	-.0011***	-.0002	-.0009**	-.0001	-.0005*
	(.0005)	(.00029)	(.0004)	(.0003)	(.0003)	(.0004)	(.0004)	(.0004)	(.0003)
R-squared	0.02	0.01	0.02	0.01	0.01	0.03	0.02	0.02	0.03
Municipality fixed effects?	No	No	No	No	No	No	No	No	No
PANEL B:	Parents Earnings	Parents High school	Parent College	Mom's College	Mom's High school	Father College	Father High School	Father earnings	Young mother
Lead exposure (µg/Kg)	.0002	.00012	-.00003	0.00001	.00012	-.00005	.00046*	.00008	-.00017
	(.0004)	(.00025)	(.00029)	(.0002)	(.00025)	(.0002)	(.00025)	(.0003)	(.0002)
R-squared	0.04	0.03	0.06	0.06	0.02	0.05	0.03	0.04	0.03
Municipality fixed effects?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
# observations	797,889	797,889	797,889	797,889	797,889	768,644	768,644	768,644	797,889

Notes: Standard errors are clustered at the municipality level. Controls for year of birth of the child and cohort size.

5 Results

In the following sections I look at the impact of childhood lead exposure levels on future outcomes. To preview the central results, in the baseline specification early childhood lead exposure have a negative impact on virtually all future outcomes considered. The number of years of schooling, having a low GPA at the end of the compulsory school, high school graduation, and being on welfare are all statistically significantly correlated with early childhood lead exposure. The estimated impact on the remainder of the outcomes is too imprecise to draw definite conclusions. However, further analysis reveals that the poor precision of the baseline estimates seems to be due to that the relationship with long-term outcomes are nonlinear. Reductions in lead exposure from high initial levels have consistently significant effects on virtually all of the outcomes, but similarly sized reductions from initially low levels of exposure only yield inconsistent and insignificant effects on the outcomes considered. A number of specification checks reveals that the estimated effects of exposure reduction from the highest levels is robust and that children from disadvantaged families seem to have benefited most from the reductions in lead exposure.

5.1 Baseline results

5.1.1 Cross-sectional estimates

Before proceeding with the main fixed effects analysis it is useful to replicate the results from a conventional cross-sectional analysis. For each cross section (1975, 1980, 1985) the results from estimations of equation (1) (but without the municipality fixed effects) on all outcomes considered in the main analysis is presented in Column (1)-(3) of appendix Table B2. These cross-sectional estimates in general points in the expected direction, although there is considerable variability in the magnitude of the estimates both within a given year for different outcomes but also across years for a given outcome. The estimates are furthermore only occasionally statistically significant at conventional significance levels. After pooling the data (column 4), except for earnings, all the estimates indicated that reduction in early lead exposure improves long-term outcomes.

If considering the statistically significant estimates from the pooled cross-sectional model it seems as if lead exposure particularly impairs development among children in the lower tail of the ability distribution. Both the risk of ending up in the lower quartile of the GPA and IQ-test score dis-

tribution are significantly affected, although only at the 10% significance level. The estimated effects on these outcomes suggest that the average reduction in air lead exposure (60%) that occurred between the early 1970's and early 1980s reduced the incidence of ending up in the lower tail of the grade and IQ distribution by around 1 percentage point or by about 4%. Although, as shown above in Table 2, these cross-sectional estimates are likely biased by unobserved characteristics correlated with both high levels of lead exposure and the adult outcomes of the children. Overall the cross-sectional results in Table B2 provide little evidence of a significant relationship between adult outcomes and early childhood lead exposure. However, as will become clear, the pattern with stronger effects in the lower part of the ability distribution remains throughout the empirical analysis, even after unobserved heterogeneity has been taken into account.

5.1.2 Municipality of birth fixed effects

Next the analysis proceeds by focusing on the fixed effects estimates which under the present conditions potentially give a more accurate picture of the relationship of interest. The first column in Table 3 presents the results from the estimates of equation (1) for the percentile ranked GPA, now including the municipality of birth fixed effects. The estimate for GPA presented in column (1) implies that when the average lead exposure during early childhood increases with 1 μg/kg the grade point average decreases with 0.017 percentiles. Similarly, the probability of ending up in the lower quartile of the grade distribution increases with 0.024 percentage points per 1μg/kg increase in lead exposure. For males the average IQ level also decreases with 0.010 percentiles. An inverse relationship between lead exposure and the probability of ending up in the higher end of the grade distribution is also found. The probabilities of ending up in the top or lower part of the IQ distribution are also affected as expected. Table 4 present the estimated impact on educational attainments, early labor market and social outcomes. Again all point estimates suggest that higher levels of lead exposure are detrimental for subsequent outcomes.

However, in general the precision of the estimates presented in Tables 3 and 4 is poor. Only the probability of ending up in the lower tail of the grade distribution, high school completion rates, the number of years of schooling and the welfare dependency rates are significant at conventional significance levels. At first examination the estimated effects may seem small but it is important to recall that these reduced form estimates imply that the reductions in lead exposure during the observation period implies that the probability of ending up in the lower end of the GPA distribution decreased by 3.3 percent, increased high school completion increased by 0.9 percent, years of schooling completed in 2004 increased by 0.05 years and the prob-

Table 3 Grade point averages and cognitive test scores

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	GPA	Low GPA	High GPA	IQ	Low IQ	High IQ
Sample	ALL	ALL	ALL	Males	Males	Males
Lead exposure (µg/Kg)	-.0171	.00024*	-.00017	-.0109	.00016	.00002
	(.0104)	(.00014)	(.00013)	(.0135)	(.00019)	(.00018)
R-squared	0.22	0.12	0.13	0.17	0.09	0.1
Mean of dep. var.	50	0.25	0.25	50	0.25	0.25
Individual characteristics	yes	yes	yes	yes	yes	yes
Year of birth	yes	yes	yes	yes	yes	yes
Fixed municipality	yes	yes	yes	yes	yes	yes
Mean of dependent variable	50	0.25	0.25	50	0.25	0.25
Observations	797,889	797,889	797,889	262,283	262,283	262,283

Notes: The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. In addition the estimated model includes controls for parental characteristics, and municipality characteristics (see section 4.1 for details). Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

Table 4 Alternative long-run outcomes

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	High school	Ever in College	Yrs. in School	Log earnings	Welfare	Teen mother
Sample	ALL	ALL	ALL	ALL	ALL	Women
Lead exposure (µg/Kg)	-.00022**	-.00029	-.00142*	-.00021	.00016***	0.00001
	(.00010)	(.00020)	(.00084)	(.00036)	(.00005)	(.00005)
R-squared	0.06	0.18	0.2032	0.1301	0.03	0.03
Mean dep. var.	0.89	0.33	12.7	176,400	0.04	0.04
Individual characteristics	yes	yes	yes	yes	yes	yes
Year of birth	yes	yes	yes	yes	yes	yes
Fixed municipality	yes	yes	yes	yes	yes	yes
Observations	797,889	797,889	797,889	718,843	797,889	387,576

Notes: In addition the estimated model includes controls for parental characteristics, and municipality characteristics (see section 4.1 for details). Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

ability of welfare dependency in 2004 decreased by 0.6 percentage points. Again, the effects seem to be stronger in the lower tail of the ability/skill distribution.

5.2 Nonlinear effects in lead exposure

Since most neurotoxins follow a hockey stick shaped effect, with a much lower marginal effect below some threshold, one potentially important reason for the relatively poor precision of the estimates in Table 3 and 4 could be that the relationship is nonlinear or discontinuous at the levels of exposure considered. As discussed above identifying the threshold of such nonlinear effects are, of course, highly interesting from a public policy perspective. However, neither the biological nor the epidemiological literature provides a strong theory and only very limited evidence that could give any guidance in the search for a threshold when it concerns lead (c.f. Needleman, 2004). Indeed most studies have failed to identify a lower threshold for effects on cognitive skills, although an important reason is presumably that the sample sizes at the lowest exposure levels have been relatively small, and that confounding most likely becomes even more acute when studying the sub-clinical effects of low exposure levels.

In order to examine the presence and influence of nonlinearities in this case the same model as in equation (1) is estimated, but now the single continuous linear lead exposure measure is replaced by linear splines with breakpoints at each quartile of lead exposure. This setup mimics the approach taken by Reyes (2007), who find no/only weak nonlinearities in the lead exposure-violent crime relationship. However, again the average blood lead levels in her sample were considerably higher than in this context. By using splines it is possible to examine if the effect of a similar sized reduction in lead exposure within the different quartiles of exposure has heterogeneous impacts on adult outcomes.

Table 5 presents the results from this specification for the GPA and the cognitive test scores, and Table 6 for the other outcomes. In contrast to the analysis using a single linear measure of exposure, the estimates based on changes within the different quartiles of exposure show a strikingly consistent pattern. A 1 µg/kg reduction in moss lead in the municipality of birth in early childhood has a highly significant and consistent adverse effect on basically all outcomes considered; but only within the highest quartile of exposure (i.e. >48µg/kg). Below this level similarly sized reductions in lead exposure has inconsistent and generally insignificant effects on long-term outcomes.

Table 5 Nonlinear effects of early childhood lead exposure: GPA and cognitive test scores

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	GPA	Low GPA	High GPA	IQ	Low IQ	High IQ
Sample	ALL	ALL	ALL	Males	Males	Males
Lead in 1st quartile	-.0463	.00006	-.0008	-.0166	-.0010	-.0010
	(.0426)	(.0005)	(.0006)	(.0529)	(.0006)	(.0008)
Lead in 2nd quartile	-.0421	.00024	-.0010*	-.0269	.0013**	.0002
	(.0452)	(.00059)	(.0006)	(.0404)	(.0006)	(.0005)
Lead in 3rd quartile	.0128	-.00008	.00015	.0216	-.0003	.00045*
	(.0301)	(.00041)	(.0003)	(.0175)	(.0003)	(.00025)
Lead in 4th quartile	-.0350**	.00046***	-.00025	-.0283***	.00025	-.0003**
	(.0136)	(.00018)	(.0002)	(.0106)	(.00016)	(.0001)
R-squared	0.22	0.12	0.13	0.17	0.09	0.10
Mean of dep. var.	50	0.25	0.25	50	0.25	0.25
Individual characteristics	yes	yes	yes	yes	yes	yes
Year of birth	yes	yes	yes	yes	yes	yes
Fixed municipality	yes	yes	yes	yes	yes	yes
Observations	797,889	797,889	797,889	262,283	262,283	262,283

Notes: The coefficients shown reflect the average effect of a 1µg/kg increase within each quartile. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels. The sample for the IQ test scores are reduced and only include children born before 1980 in order to reduce the impact of changes in the enrollment procedures for men born after 1980.

Table 6 Nonlinear effects of early childhood lead exposure: Alternative long-run outcomes

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	High school	Ever in College	Yrs. in School	Log earnings	Welfare	Teen mother
Sample	ALL	ALL	ALL	ALL	ALL	Women
Lead in 1st quartile	-.0002	-.0006	-.0043	-.0004	-.00039	.00015
	(.0005)	(.0011)	(.0039)	(.0020)	(.0003)	(.00025)
Lead in 2nd quartile	.0004	-.0005	.0003	.0044**	.00035	.00023
	(.0005)	(.0011)	(.0040)	(.0021)	(.00025)	(.00027)
Lead in 3rd quartile	-.0003	-.0001	-.0001	.0004	.00004	-.00025*
	(.0003)	(.0005)	(.0023)	(.0007)	(.00013)	(.00014)
Lead in 4th quartile	-.00026**	-.0005	-.0027**	-.0016***	.00019***	.00017*
	(.00013)	(.0003)	(.0013)	(.0005)	(.00006)	(.00009)
R-squared	0.06	0.18	0.20	0.1302	0.03	0.03
Mean of dep. var.	0.89	0.33	12.7	176,400	0.04	0.04
Individual characteristics	yes	yes	yes	Yes	yes	yes
Year of birth	yes	yes	yes	Yes	yes	yes
Fixed municipality	yes	yes	yes	Yes	yes	yes
Observations	797,889	797,889	797,889	718,843	797,889	387,576

Notes: The coefficients shown reflect the average effect of a 1μg/kg increase within each quartile. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

This analysis reveals a clear nonlinear effect of reduction in early childhood lead exposure on long-run outcomes. Since the average blood-lead level in the cohorts considered already initially were lower than the limit of concern, these results also suggest that if anything the threshold of the adverse effects of lead exposure on the long-term outcomes lie at a level that is significantly lower than a blood lead level of 10µg/dL.[23] In section 6 I use the elasticity provided by Nilsson et al. (2009), to estimate the childhood blood lead level corresponding to the moss lead level found in the analysis in this section.[24]

5.3 Robustness checks

Next, since the previous estimates primarily show an effect at the highest quartile of lead exposure, in order to increase precision, the remainder of the analysis concentrates on children born in municipalities with a moss lead

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[23] The earliest systematic blood lead levels sampled among children in Sweden was conducted in 1978 by Strömberg et al. (1995). At that time the blood lead level was just below 6 µg/dL on average in two locations in southern Sweden (where the air lead levels were among the highest in Sweden at the time). Needleman report blood lead levels in the US in the same year of on average around 14 µg/dL. Before this point in time in order to assess the lead levels of the children I use the estimates of previous work by Reyes (2007). She finds that an increase of 1 gram lead per gallon of gasoline increases blood lead level with approximately 3.3 µg/dL in the general population. Taking her baseline specification literally (B-Pb=9.316+3.325*GRAMSLEAD/GALLON) and combining it with the data in Table 1 suggest that at the year of birth of the first cohort born in 1972-74 the average blood lead level would have been on average 6µg/dL. A second way to assess the initial blood lead levels is to use the model developed in Nilsson et al. (2009) and use the average moss lead level in 1975 to predict the blood lead levels in primary school children at that time. This approach provides a predicted initial blood lead level of around 3µg/dL in primary school children. After adjusting the blood lead moss lead elasticity using the age specific blood lead blood gasoline elasticity estimated by Reyes (2007) (30% higher for children aged 0-6 than for children aged 6-12) and under the additional assumption that the additative separable specification used in the estimation hold for both populations, the relevant blood lead level in for children aged 0-6 would on average correspond to about 5µg/dL. A third way to estimate the initial blood lead level is to use the estimates in Strömberg et al., who based on repeated blood lead measurements find that individual blood lead levels in primary school children decreases on average by around 6% per year. Given the average level in 1978 this would imply that the average blood lead levels among 1-4 year olds in 1972 (since the samples are taken from children aged 7-10 in 1978) would be on average 8.5µg/dL. Since these samples were taken in a region with the highest lead exposure (based on the moss lead values) it seems reasonable to assume that this level represent a higher bound of blood lead levels in the general population of children in these cohorts. Hence all three approaches provide estimates that suggest that initial blood lead levels were below 10µg/dL for the cohorts born between 1972 and 1974.

[24] One concern with this analysis is that the apparent nonlinear effect could be due to that the precision of the estimates are poor at the lower quartiles of exposure simply because the changes in exposure within these quartiles are not large enough. However, the pattern in Table (6) and (7) is if anything reinforced if the children growing up in municipalities with the least (<10 %-tile) changes in exposure between 1975 and 1985 are excluded.

level above the 1st quartile of initial exposure (>37 µg/kg in 1975). This decreases the number of municipalities included in the analysis to 210 and the number of children to 670,000.

To make sure that excluding the lowest initial level municipalities in this way does not introduce any major bias it is informative to compare the baseline model estimates for the reduced sample with the baseline linear spline estimates. Column (1) of Table B4 in appendix B report the estimates from the original specification in equation (1) estimated on the reduced sample (Tables B2 and B3 report the full results). As expected, these estimates are higher and more precisely estimated compared to the baseline OLS estimates displayed in Tables 3 and 4, and are reasonably similar to the estimates for reductions in the highest quartile of exposure in Tables 5 and 6. For further comparison, in Table B4 I have also included the estimated effect when children in the highest quartile of initial exposure are excluded (column 3). After excluding the highest quartile of initial exposure the estimated parameters in virtually all cases are insignificant and when they are significant (earnings, teen pregnancy) they are always pointing in the unexpected direction. By comparing the results in columns (1) and (3) in Table B4 it again becomes clear that the initial level of exposure is important and that the relationship between early childhood lead exposure and long-term outcomes seem to be nonlinear. Columns (2) and (4) of Table B4 also report estimates after splitting the sample based on *changes* in exposure between 1975 and 1985. The resemblance between the high initial and high changes municipality estimates is striking.

Table 7 continues the analysis by testing the sensitivity of the reduced sample estimates. First, for ease of comparison, column (1) reviews the baseline estimates for the children under risk of being affected. In column (2) family fixed effects estimates are reported. This model accounts both for the fixed characteristics of the biological parents and the municipality of birth. The family identifier used is a combination of the unique mother and father identifiers and hence in the analysis the comparison is made between full biological siblings only (in total 123,324 families).

Table 7 Robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Specification/ Sample:	Baseline	+ Family fixed effects	+ Linear time trends	+ cad-mium exposure	Log exposure	Excl. 3 largest cities	+ Child-care enroll.	Munici-pality level data
EXPOSURE:	*Lead*	*Lead*	*Lead*	*Lead*	*ln (lead)*	*Lead*	*Lead*	*Lead*
OUTCOMES:								
GPA	-.0356**	-.0175*	-.0294	-.0453***	-1.290**	-.0246**	-.0356**	-.0289**
	(.0143)	(.0102)	(.0218)	(.0172)	(.5575)	(.0125)	(.0145)	(.0148)
Low GPA	.0006***	.0004**	.00034	.0006***	.0162**	.0004***	.0006***	.00045**
	(.0002)	(.0002)	(.0003)	(.0002)	(.0074)	(.0002)	(.0002)	(.0002)
High GPA	-.0003	-.00014	-.00043*	-.00044**	-.0154**	-.00018	-.00032*	-.00025
	(.0002)	(.00018)	(.00025)	(.0002)	(.0071)	(.00018)	(.00018)	(.00018)
IQ (Men)	-.0336***	-.0664**	-	-.0403***	-1.129***	-.0352***	-.0349***	-.0272**
	(.0128)	(.0290)		(.0105)	(.4004)	(.0106)	(.0092)	(.0128)
Low IQ (Men)	.0003**	.0009*	-	.00044**	.0141**	.00034**	.0003**	.00031
	(.00015)	(.0004)		(.00015)	(.0058)	(.00015)	(.00015)	(.00025)
High IQ (Men)	-.00026**	-.0009*	-	-.0003*	-.0064	-.00024*	-.00028**	-.00031*
	(.00012)	(.0005)		(.00015)	(.0059)	(.0001)	(.00012)	(.00017)

High school	-.00033*	-.0003**	-.0002	-.0003	-.0066	-.0001	-.0002	-.0002
	(.00017)	(.00014)	(.00028)	(.0002)	(.0067)	(.00015)	(.00014)	(.00015)
University	-.0002	-.0003	.0002	-.00033	-.0071	-.00024	-.0002	-.0002
	(.0002)	(.00019)	(.0003)	(.00028)	(.0093)	(.00028)	(.0002)	(.0002)
Years of schooling	-.0022*	-.0019**	-.0007	-.0022*	-.0644	-.0015	-.0015	-.0009
	(.0012)	(.0008)	(.0015)	(.0013)	(.0447)	(.0012)	(.0010)	(.0008)
Welfare	.00012	.00017**	.00004	.00005	.0017	.0002***	.0001	.00011
	(.0001)	(.00008)	(.0001)	(.0001)	(.0035)	(.00006)	(.0001)	(.00008)
Earnings	-.0009**	-.0011**	.0012**	-.0015**	-.0123	-.0009*	-.0007*	-.0007*
	(.00045)	(.0005)	(.0005)	(.0007)	(.0237)	(.0005)	(.0004)	(.0004)
Teenage mom	.0001	.0005**	.00013	.00014*	.0038	.0001	.0001	.00007
	(.0001)	(.0002)	(.0001)	(.00008)	(.0034)	(.0001)	(.0001)	(.00005)

Notes: Each row and column represent a separate regression. Column (2) presents the results estimates from a family fixed effects model; column (3) introduces municipality of birth specific time trends; column (4) replaces the linear exposure measure with log lead exposure, column (5) excludes the 3 largest cities; column (6) checks to what extent the estimated baseline effect of exposure to lead is confounded by effects of changes in other pollutants; column (7) adds controls for public childcare enrollment; finally column (8) reports estimates from a model where the individual data have been aggregated to the municipality level. In addition all specifications includes controls for municipality, cohort fixed effects and individual and parental controls. The reported estimates is the marginal effect of one (1) unit (1 μg/Kg) increase in municipality of birth lead exposure during early childhood, except in column (4) which report estimates for a lin-log specification. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. */**/*** indicate significance at the 10/5/1 percent levels. Standard errors are clustered at the municipality level (except for the IQ outcomes regressions that only uses two time periods and hence adjust standard errors at the time period-municipality level). The municipality level regressions (column (7)) are weighted by the number of children in each municipality/period cell. The IQ (GPA) outcomes also control for year of enlistment (Graduation) specific effects. The baseline sample estimates refers to the linear estimates after excluding the municipalities below the 1st quartile of initial (1975) lead exposure level (full results in appendix B). In the family fixed effect model only families with less than four children are included.

The municipality fixed effect in this model is identified by families that report differing municipality of birth for their children.[25] After including family fixed effects, the effect of childhood lead exposure is identified by differences in exposure between siblings. As the estimates in column (2) show, conditioning on parental fixed effects in addition to the municipality fixed effects produces estimates that are similar to the baseline estimates, but for cognitive test scores the point estimates are larger in absolute terms. The high similarity between the sibling fixed effects results and the main results are striking, and provides further evidence for the validity of the estimated relationship.

In column (3) the baseline specification is augmented with municipality of birth specific linear time trends. This model addresses the concern that the main effects could partly result from trends in outcomes at the local municipal level. After introducing these time trends, the majority of the point estimates decreases somewhat. For earnings the point estimate changes sign. At the same time the precision of almost all estimates decreases significantly. However, the sharply decreased precision of the estimates after controlling for municipality linear time trends should come as no surprise. The parameter of interest in this specification is identified using only the residual variation of each municipality around its own time trend. Since the effective panel only stretches over three years (1975, 1980, 1985), this specification is likely to reduce the signal-to-noise ratio considerably and increase attenuation bias.

When trying to identify the effects of a particular polluting element it is important to address the concern that any association found between lead exposure in childhood and outcomes later in life in principle could be due to that the observed air lead levels simply proxy for other unobserved pollutants correlated with lead. If higher (unobserved) pollution also leads to poorer subsequent outcomes, this may bias the estimated impact of lead upwards. The focus on the changes in air lead levels induced by government regulations targeting gasoline lead levels in particular should mitigate much of this problem. Still, since the moss sample data also hold information on seven of the other most common heavy metal pollutants (As, Cd, Cr, Cu, Ni, V, Zn) it is easy to do an initial assessment of the potential severity of this problem.[26] Of all the observable environmental pollutants in the data, the only other air pollutant which displays even nearly as large and widespread changes during the observation period as lead does is cadmium (Cd).

Cadmium has previously been found to be associated with adverse health outcomes (kidney damage, bone disease). Early exposure to Cd has been

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[25] In 19.8% of the two-child families the siblings have differing municipalities of birth.

[26] This is the data contained from the start of the moss survey. From 1985 the Iron (Fe) and mercury (Hg) levels also started to be assessed.

shown to be able to produce neurotoxic effects in laboratory experiments (Anderson et al., 1997; Peterson et al., 2004), and in a recent study cadmium air releases are shown to affect infant health in humans (Currie and Schmeider, 2009). Moreover, air cadmium and air lead levels display a fairly high correlation at the municipality level; both in the cross-section (corr. coeff.=0.8) and in changes between 1975 and 1985 (corr. coeff.=0.5). Hence, changes in air Cd levels could potentially at least partly explain the estimated relationship between lead and subsequent adult outcomes. However, in this context it is not likely that the baseline estimates for lead are driven by the changes in local air cadmium exposure rather than local air lead exposure. This is so since unlike lead, the primary exposure route of cadmium is dietary rather than respiratory (WHO, 1972; IPCS, 1992; Moon et al., 2003; Ohlsson et al., 2005).[27] Cadmium accumulates in crops, fish and livestock. But since only a small proportion of the food that children in Sweden (and elsewhere in most developed countries) eat is locally produced, *a priori* it is not expected that the intertemporal changes in *local* air cadmium levels in early childhood necessarily are associated with adverse future outcomes.

Nevertheless, to make sure that the impact of the changes in lead are not confounded by the changes in cadmium both the lead and cadmium exposure measures are entered into the same regression to assess to what extent controlling for cadmium has an effect on the precision and/or magnitude of the estimated effect of lead. The estimated lead coefficients are reported in column (4) of Table 7. For most outcomes the estimates effects of lead remain highly similar after the additional control for cadmium is included. These results clearly indicate that the effect of lead does not seem to be caused by the simultaneous changes in cadmium.[28]

In column (5) estimates are reported from a specification where the natural log of exposure has replaced the baseline linear exposure measure in or-

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[27] For example, Moon et al. (2003) calculate the ratio of the dietary route uptake over the sum of the uptake via dietary and respiratory routes in a sample of non-smoking non-occupationally exposed mothers and their children. Cadmium intake was almost exclusively from food (98%), both in children and mothers. Dietary cadmium intake of children significantly correlated with that of their mothers. Dietary lead intake in children, however, did not correlate with that of their mothers. Lead uptake from ambient air tended to be higher (50%) in children than in their mothers (35%).

[28] The parameter estimates for cadmium are almost exclusively insignificant, and almost always point in an unexpected direction; i.e. higher cadmium improves adult outcomes. The discrepancy between the impact of Cd found for infant health in Currie and Schmeider (2009) and the lack of effects on long run outcomes found here could be due to that Currie and Schmeider examine releases of large doses at critical periods *in utero*. The changes in cadmium exposure in this setting potentially reflect more subtle changes at low levels in Cd exposure. Therefore the evidence here should not be taken as evidence that a temporary large dose of air cadmium exposure does not have an effect on long-run outcomes. The cadmium exposure estimates are retained due to space limitations, but are available upon request from the author.

der to investigate to what extent the model is sensitive to changes in functional form. A somewhat counter intuitive feature of the lin-log model, given the suggested nonlinear effect with the strongest effect at the highest exposure, is that it imposes decreasing marginal effects at the highest levels of exposure. Still the effects on GPA and IQ remain highly significant while the impact on schooling outcomes no longer is significant. However, the point estimates suggest effects of similar size as the baseline estimates. This could potentially indicate that exposure at the highest levels are particularly important for the later adult outcomes, while the more subtle effects on IQ and GPA remain even at lower levels of exposure.

Column (6) assess to what extent the exclusion of children growing up in the three largest cities (Stockholm, Göteborg and Malmö) affects the parameter estimates. For various reasons one may suspect that the lead exposure measure in these areas is a less good predictor of the children's blood lead levels than in other less densely populated areas. For example, exposure to motor vehicle exhaust is likely higher and at the same time the moss sampling sites may differ significantly from other areas. However, as shown in column (6) restricting the sample in this way only has limited effects on the precision and the magnitude of most of the estimates.

Approximately simultaneous with the sharp phase-out of leaded gasoline there was also a strong expansion in the public day-care system in Sweden. Hence, a concern is that the impact of the reductions in lead exposure could partly be confounded by increased day-care enrollment (see e.g. Baker, Gruber and Milligan, 2009). However, first of all a regression of the day-care enrollment rates on childhood lead exposure suggests no significant association between the two variables. Still, to make sure that the increase in day-care enrollment rates is not biasing the baseline estimates, column (7) reports the results from a model where cohort-municipality specific day-care enrollment rates (averaged over ages 0-6) has been included as additional controls.[29] Again as seen in column (7) the baseline results are in general not sensitive to this change in specification.

Finally in column (8) I have aggregated the data to the municipality level in order to address the concerns of biased inference due to the regression of a municipality level explanatory variable (lead exposure) on individual outcome data.[30] This alternative and conservative method in general provides

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[29] Moreover, since left-wing local governments were more likely to expand public day-care, additional controls for the number of years during the index person's childhood that the municipal council had a left-wing majority is also added. Note that the ideological orientation of the municipality council potentially captures many different hard to observe characteristics of the parents and the municipalities besides childcare. The data on childcare exposure and municipality level political majorities were kindly provided by Per Pettersson-Lidbom.

[30] I follow Bertrand et al. (2004) and first regress all individual variables on the outcomes and then use the average residuals as the outcome variable in a regression on the municipality

fairly similar results as the baseline model does which is reassuring. The estimates for the outcomes which no longer remain significant after aggregating the data are typically not significantly different from the baseline estimates.

To summarize, the weight of the evidence presented in this section first of all clearly displays the robustness of the main results to various specification changes. Several tests of alternative explanations for the observed effect of lead on adult outcomes suggest that neither observed nor unobserved changes in other important factors such as other pollutants, unobserved parental characteristics, or municipality specific factors seem to be able to explain the main results.

5.4 Heterogeneity

The analysis now proceeds by investigating the heterogeneity of the main effects. In section 5.4.1 the potential redistributive role that environmental policies may play is examined by checking if low SES and high SES children are differentially affected by the phase-out of leaded gasoline. Section 5.4.2 assess to what the extent there exists any difference in the susceptibility or impact of lead exposure early in life between boys and girls. Finally, in section 5.4.3 the sharp inter-temporal differences in lead exposure is exploited in order to test the validity of the assumption that early childhood lead exposure is more harmful than lead exposure later on in childhood.

5.4.1 Can differences in pollution exposure early in life explain parts of the SES-gap in economic outcome later in life?

Parental resources may potentially help mitigate some of the negative effects of adverse conditions in early life (see e.g. Currie and Hyson, 1999; Case et al., 2002; Cunha and Heckman, 2007). Moreover, several studies have found that low SES children are under higher risks of being exposed to environmental hazards; either through residential segregation or by less care taken by polluters in reducing the risk of exposure in neighborhoods with families of low political and/or economic influence.[31] Information differentials about the health effects of pollution exposure between low and high SES households could also result in differential childhood lead exposure even within the same localities, since it can induce differential avoidance behavior across

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level controls and fixed effects. The reported standard errors are robust to within municipality correlation. The observations are weighted by the number of children in each municipality-period cell, rather than municipality year of birth cell which explains the differences between the individual level estimates and the estimates from the aggregated data.

[31] See e.g. Davidson and Anderton (2000) or Szasz and Meuser (1997) for a review of the environmental justice literature.

social groups (c.f. Neidell, 2004). A SES gradient in the long-term effects of early childhood lead exposure could also be expected if parents from different social groups have different preferences for or possibilities to compensate the impact of lead on subsequent outcomes. Finally, since children from poorer backgrounds more often suffer from other health problems, a SES gradient in the effect of early childhood lead exposure could result from interactive effects of lead exposure and other health problems (c.f. Currie et al. 2009). In either case environmental policy initiatives that improve air quality are bound to benefit children in the poorest household the most.[32]

To assess whether the phase-out of leaded gasoline improved long term outcomes particular for low SES children, Table 8 report results from separate regressions by parents earning (below/above the median in 1990), and by parental education attainments (at least one of the parents has completed high school or not). As expected the parameter estimates are systematically larger and more precisely estimated for low SES children suggesting that the benefits of the reductions in lead exposure are particularly beneficial for the relatively disadvantaged children.

Given the data at hand, it is not possible to fully differentiate between the relative importance of the competing underlying mechanism behind the SES gradient. However, since the data also contain information on the parish of birth it is possible to examine if the same SES-gradient persists even if fixed parish of birth effects (2500 parishes) are included as additional controls.[33] A parish on average corresponds to the size of a US census tract (≈ 4.500 individuals). Hence, the parish of birth fixed effects regression compares children growing up in the same "neighborhood" within the municipality, and thereby the importance of differential exposure between low and high SES children induced by residential segregation *within* municipalities should be reduced. While parishes are not an ideal measure of the relevant neighborhood, it is the finest locality of birth data available and can be assumed to work as a decent proxy for it.[34]

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[32] Chay and Greenstone (2003b) find suggestive evidence of a SES gradient in the impact of Total Suspended Particulates (TSP) on infant mortality.

[33] Unfortunately, I do not have access to polygons for parish of birth, and hence can not calculate exposure levels for the parishes. However, it should be noted that even if they were available it is not evident that parish exposure would be a preferable measure compared to municipality exposure since parish boarders are likely to be crossed in regular day to day activities to a higher extent than municipality boarders.

[34] A better neighborhood definition than the parish of birth would be to use the SAMS areas (9,000 locations), which are very well defined neighborhoods. However, unfortunately information on the SAMS of residence are not available before 1985, and therefore the parish of birth was preferred in order to minimize the risk of attaining biased estimates due to endogenous parental migration between the year of birth and 1985. However, I have also estimated the same models using SAMS fixed effects instead and the results were *highly* similar.

Table 8 Estimates by socioeconomic status

Specification	(1)	(2)	(3)	(4)
Sample:	Low education parents (no high school)	Educated parents	Below median earnings	Above median earnings
OUTCOMES	N=196,359	N=472,550	N=329,076	N=339, 847
GPA	-.0425**	-.0329**	-.0469***	-.0249*
	(.0166)	(.0144)	(.0152)	(.0151)
Low GPA	.0007***	.0005**	.0008***	.00034*
	(.00024)	(.0002)	(.0002)	(.0002)
High GPA	-.0003	-.00025	-.0004**	-.0002
	(.0002)	(.00020)	(.0002)	(.0002)
IQ (Men)	-.0613***	-.0156	-.0307	-.0378**
	(.0165)	(.0113)	(.0193)	(.0144)
Low IQ (Men)	.0006**	.0002	.00061*	.00006
	(.00026)	(.00016)	(.00034)	(.0002)
High IQ (Men)	-.00039**	-.00016	-.00008	-.00043*
	(.00017)	(.00015)	(.00022)	(.00023)
High School	-.00034	-.0003*	-.00038*	-.00017
	(.0002)	(.00016)	(.00019)	(.00014)
University	-.0004	-.00017	-.00041	-.00009
	(.00025)	(.00026)	(.00026)	(.00025)
Yrs of schooling	-.0028**	-.0016	-.0024**	-.0011
	(.0012)	(.0012)	(.0012)	(.0009)
Welfare	.00022	.00010	.00018	.00005
	(.00015)	(.00007)	(.00011)	(.00006)
Earnings	-.0018***	-.0009	-.00073*	-.0012**
	(.0006)	(.00065)	(.00040)	(.0005)
Teenage mother	-.00011	.00020	.00008	.00012*
	(.00014)	(.00007)	(.0001)	(.00007)
Individual char.	Yes	Yes	Yes	Yes
Parental char.	Yes	Yes	Yes	Yes
Year of birth FE	Yes	Yes	Yes	Yes
Mun. of birth FE	Yes	Yes	Yes	Yes

Notes: Each row and column represent a separate regression. The reported estimates is the marginal effect of a (1) unit (1 μg/Kg) increase in municipality of birth lead exposure during early childhood. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. */**/*** indicate significance at the 10/5/1 percent levels. Standard errors are clustered at the municipality level (except for the IQ outcomes regressions that only uses two timeperiods and hence adjust standard errors at the timperiod-municipality level). Parent's characteristics include maternal education (7-levels) and indicators for quintile of total parental earnings in 1990. The IQ (GPA) outcomes also control for year of enlistment (Graduation) specific effects.

The estimated differences in the impact of lead exposure early on between children of differential socioeconomic background persist even if they grew up in the same neighborhood. The point estimates generally decrease somewhat but not to the same extent as would have been expected if residential segregation would be the main cause of the SES-gradient (not reported). Next I limited the sample to children with parental earning more or less than the median earnings but, with and without having parents with a college/high school education. The parameter estimates from this matched comparison provide suggestive evidence that the income of parents seems to matter more than education of parents (not reported). The differences in the impact of lead on children in the two parental income groups are still large even after conditioning on parental educational attainments. Hence, although parental earnings seem to matter a lot for the influence of lead exposure on child outcomes an obvious candidate mediating mechanism for this income gradient, residential sorting within municipalities, does not seems to be the main mechanism at work. In addition, even after conditioning on parental education, the parental income gradient remains almost as strong.

Under the assumption that university education of the parents is indicative of a higher awareness of potential negative effects of air pollution (and thereby a higher degree of avoidance behavior), these two auxiliary results suggest either i) that children with parents with low earnings are more heavily affected by air lead levels, potentially due to heightened sensitivity (e.g. due to co-morbidities); ii) that poorer parents lack the resources needed to compensate for the initial insult to the child's development, or iii) that low and high SES parents have different preferences regarding the value of remedying investments in their children.

Whichever of the suggested reasons that are most valid, these results clearly indicate that environmental policies such as the ban of leaded gasoline not only has the ability to reduce the intergenerational transmission in economic outcomes. However, they also indicate that public and/or private resources/initiatives may potentially be effective in reducing the impact of early life insults on long-term outcomes, particularly among low SES children.

5.4.2 Effects of lead exposure by gender

Studies investigating gender differences in the impact of lead exposure has typically not detected differential effects of early childhood lead exposure on behavioral or cognitive outcomes among boys and girls (see e.g. Burns et al 1999). But again these studies typically focus on children with relatively high levels of exposure. It has been suggested that in general male fetuses and infants are more susceptible to damage from early insults to health.

Since boys tend to develop more slowly than girls, this could imply that exposure to lead may lead to greater damage in boys.[35]

To assess this notion Table 9 and 10 present the OLS estimates (except IQ and teenage pregnancies) on all outcomes from separate regressions on the male and the female samples respectively. In general the point estimates are highly similar for both boys and girls. The precision is somewhat better for the girls (particularly for the educational attainments) potentially reflecting the lower variance in these outcomes among girls.

Table 9 Women

Specification	(1)	(2)	(3)	(4)	(5)
	Women	*Women*	*Women*	*Women*	*Women*
Outcomes:	High school	Ever in College	Yrs. in School	Log earnings	Welfare
Sample	ALL	ALL	ALL	ALL	ALL
Lead exp. (µg/Kg)	-.00033**	-.0003	-.0023**	-.00085	.00014
	(.00014)	(.00026)	(.0011)	(.0006)	(.00009)
R-squared	0.06	0.17	0.19	0.06	0.03
Mean of dep. var.	.91	.38	12.9	141,437	.037
Outcomes:	(6) GPA	(7) LOW GPA	(8) HIGH GPA		
Lead exp. (µg/Kg)	-.0334**	.00043***	-.00038		
	(.0160)	(.00019)	(.00025)		
R-squared	0.19	0.09	0.13		
Mean of dep. var.	56	.18	.31		
Indiv. Char.	yes	yes	yes	yes	yes
Yr of birth FE	yes	yes	yes	yes	yes
Muni. F.E:	yes	yes	yes	yes	yes
# Obs.	324,694	324,694	324,694	291,002	324,694

Notes: The table reports results for separate regression on females. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

[35] It is also interesting to highlight the fact that studies that have investigated gender differences in blood lead levels has found that up until around age 10 the blood lead levels in boys and girls are generally highly similar, and then starts to diverge (see e.g. Strömberg et al., 1995).

Table 10 Men

Specification	(1)	(2)	(3)	(4)	(5)
	Men	*Men*	*Men*	*Men*	*Men*
Outcomes:	High school	Ever in College	Yrs. in School	Log earnings	Welfare
Sample	ALL	ALL	ALL	ALL	ALL
Lead exp. (µg/Kg)	-.0002	-.0002	-.0020	-.00098*	.00011
	(.0002)	(.00025)	(.0014)	(.00055)	(.00009)
R-squared	0.06	0.17	0.20	0.14	0.03
Mean of dep. var.	.87	.27	12.5	211,095	.037
Outcomes:	(6) GPA	(7) LOW GPA	(8) HIGH GPA		
Lead exp. (µg/Kg)	-.0380**	.00065***	-.00022		
	(.0161)	(.00023)	(.00019)		
R-squared	0.03	0.09	0.11		
Mean of dep. var.	44.8	.31	.18		
Indiv. Char.	yes	yes	yes	yes	yes
Yr of birth FE	yes	yes	yes	yes	yes
Muni. F.E:	yes	yes	yes	yes	yes
# Obs.	371,996	371,996	371,996	310,772	371,996

Notes: The table reports results for separate regression on females. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

5.4.3 Early childhood (age 0-2) vs. pre-primary school (age 5-7) lead exposure

In line with the epidemiological literature the analysis so far has assumed that children's development should be most strongly affected by early childhood lead exposure. As discussed above the motivation for the focus on this age period is that lead take up is higher and the rate of development is particularly rapid and critical in early life. Moreover, when it comes to insults to children's development it has been suggest that earlier insult should have a stronger effect than later insults on subsequent outcomes due to the potentially dynamic complementarities of human capital accumulation; that is if skills beget skills (Cunha and Heckman, 2007). Hence, disadvantages early on may induce children to fall behind and not catch-up to their healthier peers. If either of these notions is true then early exposure should play a greater role than exposure to lead later on.

On the other hand since the brain continues to develop until around age 20, and children potentially recover from early insults, more recent exposure to air pollutants might be more important (Currie et al., 2009). Indeed a few recent studies have suggested that lead exposure in ages 5-7 are more strongly correlated with IQ than early childhood exposure (c.f. Hornung et al., 2009) and the references cited therein).[36] Moreover, it has been suggested that for cognitive skills the most sensitive period is early childhood while the most sensitive period for noncognitive skills occur later in childhood (c.f. Heckman, 2007). Further evidence on the age of greatest vulnerability to lead is moreover of clear policy relevance. If later exposure is shown to produce the same effects as early exposure efforts to reduce blood lead levels should continue through out childhood (Hornung et al., 2009).

In order to differentiate between the impact of early and late childhood lead exposure one would ideally like to have measures of the lead exposure from birth until the outcome of interest is realized. But, since lead exposure at different ages will be highly correlated, a distinction between the impacts of early vs. late childhood exposure is difficult in most settings. With these caveats in mind with my data it is however still possible to estimate a horse race model between early childhood (ages 0-2) and the pre-school age (age 5-7) exposure since for the present cohorts the changes in lead exposure between the different ages are substantial.[37]

The estimates for the impact of lead at the different ages on adult outcomes are presented in Tables 11 and 12. From the results in these tables a clear pattern emerges. For all outcomes the baseline estimates for early exposure is highly similar to the baseline model estimates and in most cases significant. For virtually all outcomes the estimated impact for exposure later in childhood is smaller than the age 0-2 exposure, and not statistically significant. The only two exceptions to this rule is the estimated impact on welfare dependency and earnings where the point estimates is higher for later childhood exposure than early childhood exposure.

Moreover, in most cases the standard errors are not any larger for the later childhood estimates than for the early childhood estimates. Hence, it does not seem as if the later childhood exposure estimate is insignificant just because the precision decreases due to collinearity between the two measures of exposure. However, before concluding that early is much worse than later exposure, it should be kept in mind that there are at least two additional factors which potentially hamper the validity of this interpretation. First, the

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[36] However, again previous studies in general investigate children with much higher lead exposure than the children in this setting, use small samples, typically look at cognitive test administered only in childhood and are susceptible to omitted variable bias.

[37] In order to implement this exercise data on lead exposure from the 1990 moss survey was added to the last three cohorts.

Table 11 Age of greatest susceptibility, GPA and cognitive test scores

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	GPA	Low GPA	High GPA	IQ	Low IQ	High IQ
Sample	All	All	All	Males	Males	Males
Lead exp.	-.0461***	.0007***	-.0004*	-.0264*	.0002	-.00023
(age 0-2)	(.0164)	(.0002)	(.0002)	(.0150)	.0002)	(.00018)
Lead exp.	-.0162	.00018	-.00013	-.0020	-.0001	-.00019
(age 5-7)	(.0185)	(.0002)	(.0002)	(.0177)	(.0003)	(.00027)
R-squared	0.21	0.12	0.14	0.17	0.09	0.1
Mean of dep. var.	50	0.25	0.25	49.8	0.22	0.26
Individual & parental char..	yes	yes	yes	yes	yes	yes
Yr of birth FEs	yes	yes	yes	yes	yes	yes
Muni FEs	yes	yes	yes	yes	yes	yes
Observations	668,909	668,909	668,909	220,498	220,498	220,498

Note: The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate statistical significance at the 10/5/1 percent levels.

Table 12 Age of greatest susceptibility, alternative long-run outcomes

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	High school	Ever in College	Yrs. in School	Log earnings	Welfare	Teen mother
Sample	All	All	All	All	All	All
Lead exp.	-.00027*	-.0001	-.0015	-.0013***	.00016	.00014*
(age 0-2)	(.00015)	(.0003)	(.0010)	(.0005)	(.0001)	(.00008)
Lead exp.	.0002	.0004	.0021	-.0021***	.00027*	.00007
(age 5-7)	(.0002)	(.0003)	(.0016)	(.0008)	(.00015)	(.0001)
R-squared	0.06	0.18	0.21	0.13	0.03	0.03
Mean of dep. var.	0.89	0.32	12.7	177,283	0.037	0.042
Individual & parental char.	yes	yes	yes	yes	yes	yes
Yr of birth FEs	yes	yes	yes	yes	yes	yes
Muni FEs	yes	yes	yes	yes	yes	yes
Observations	668,909	668,909	668,909	601,774	668,909	325,010

Note: see Table 11

exposure measure used is local exposure rather than individual blood lead levels. Since lead uptake is higher in early childhood the differences in the impact on long run outcomes could be due to differential blood lead levels at the different ages. Second, the nonlinearity of the relationship between childhood lead exposure and adult outcomes suggested above could also provide a similar pattern since the lead exposure in the pre-primary school years typically has decreased below the relevant level of concern as estimated above.

Although definite conclusions regarding the most sensitive period is difficult to make with the data at hand at least the results in this section do not provide any direct support for the hypothesis that later childhood lead exposure should be more detrimental than earlier exposure. Instead, the weight of the evidence suggests that early childhood lead exposure is more influential than later exposure which is in line with most the theoretical mechanisms suggested in the literature and the previous empirical evidence.

6 Economic significance and policy implications

In order to attain a rough estimate of what blood lead level the critical moss lead levels correspond to, I use the model estimated in Nilsson et al. (2009) for the pre-ban of leaded gasoline period.[38] This yields an estimate suggesting that for the children aged 7-10 a local moss lead level of 50µg/kg (i.e. lower end of the 4th quartile of exposure used in section 5.2) correspond to a blood-lead level of around 3µg/dL under a log-normal distribution. After adjusting the blood-lead moss-lead elasticity using the age specific blood-lead gasoline-lead elasticity estimated in Reyes (2007) (30% higher for children aged 0-6 than for children aged 6-12) and under the additional assumption that the additive separable specification used in the estimation holds for both populations, the relevant blood lead level in this setting would correspond to about 4.8µg/dL.

This estimate suggests that the average early childhood blood lead level among children in more than 50% of the Swedish municipalities in the period 1972-1974 were high enough to affect their adult outcomes. Since these municipalities also are the most densely populated, a majority of the children in Sweden born in the late 1960s and early 1970s likely suffered from blood lead levels high enough to potentially affect their future adult outcomes. However, since this study use the average municipality lead levels as exposure measure, the average effects on cognitive ability are associated with a *municipality* average blood lead level above 4.8µg/dL. It is thus in principle possible that the entire effect could solely be caused by large effects on cog-

[38] Evaluated at the mean of the explanatory variables

nitive development among a few children with very high blood lead levels. However, since that the standard deviation in childhood blood lead levels is not extremely large this seems less likely. Still, the preciseness of this blood lead level "threshold" remains to be confirmed in future research using individual childhood blood lead levels *and* a credible empirical strategy that takes unobserved confounders into account. At the very minimum the evidence provided in this study gives a clear indication that while the current acceptable blood lead limit (10µg/dL) is set at a level above which acute effects of lead might be avoided, it is clearly not low enough to prevent more subtle damage on child development.

With these caveats in mind it is interesting to consider the effects on future GPA and earnings if early childhood blood lead levels would decrease from 10µg/dL to 5µg/dL. By combining the estimated average impact on GPA in the upper quartile (see Table 5 & 6) and assuming that the estimated elasticity between lead in moss and lead in children is constant in this interval (i.e. 0.57) a decrease in a child's blood lead level from 10 µg/dL to 5 µg/dL would imply an average increase in 9th grade GPA by 2.2 percentiles and an increase in the high school graduation rate by 2.3 %. In terms of labor market outcomes the same decrease would imply an estimated increase in earnings (average for ages 20-32) by 5.5%.

Although due to the strong life-cycle variations in income, concurrent earnings measured below age 30 is typically not a very accurate measure of life time earnings, and hence the earnings estimate should be interpreted with care (c.f. Haider and Solon, 2006; Lindqvist and Böhlmark, 2006). If instead regressing age 30-32 earnings (i.e. only for those born in 1972-1974) on a high school graduation dummy (or grade 9 GPA), gender, year of birth and family fixed effects, the Swedish high school premium is estimated to be about 17%, and a one (1) percentile rank increase in GPA is associated with on average 0.54% higher earnings at age 30-32.[39] If combining these estimates with the estimated effects of lead exposure on GPA and high school graduation rates, the effect of reducing early childhood blood lead levels from 10 to 5µg/dl implies that life time earnings would increase by 1.2% (2.2*0.54) using the GPA/earnings estimate and around 0.4% (17*0.023) using the high school graduation premium estimate.[40] Of course these alternative estimates only capture the part of the lead exposure effects on earnings that goes through educational attainments.

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[39] In Sweden the life cycle bias in earnings are found to be minimal after age 33 which is why I estimated the impact of the educational attainments on earnings for the earliest cohorts only; see further Böhlmark and Lindqvist, (2006).

[40] The lower estimate for high school likely reflects that part of the earnings effects which goes through the impact of increased university *completion* (which is too early to estimate).

With these estimates it is for example possible to calculate the hypothetical gains of reducing the blood lead levels of the 310,000 children in the US (c.f. CDC, 2005) with a blood lead above 10µg/dL to 5µg/dL. Since general equilibrium effects most likely is not an issue, under the assumption that the earnings effects are directly translatable to the US setting, and given an annual income of 30,000 USD, the benefits in terms increased labor market earnings from reducing the blood lead level in these children would hence be around USD $112 million annually after age 32 (30,000*310,000*0.012) using the GPA/earnings estimate, and around USD $37 million annually using the high school graduation/earnings estimate.[41] This reflect the effect on the average population of children, but since 60% of all children with blood lead levels above 10µg/dL are Medicaid eligible (see Currie, 2009) the expected effects on individual earnings could be larger.

7 Concluding remarks

This study use a new measure of early childhood lead exposure to estimate the long run effects of the rapid reductions in lead exposure following the phase out of leaded gasoline. The results are robust to a number of specification changes and suggest that in Sweden the reduction in children's blood lead levels that occurred between 1972 and 1984 has improved young adult outcomes for a majority of the population.

The main policy implication of the results concerns the evidence of the nonlinear effect of municipality air lead levels in early childhood on young adult outcomes. This nonlinear relationship provides suggestive evidence of the existence of a threshold below which further reductions in early childhood blood lead levels no longer improves long term outcomes. Given the wide use of heavy metal moss monitoring throughout Europe, the finding that reductions in lead exposure below 49µg/kg moss no longer seems to affect long-term outcomes is of clear policy relevance in itself. However, this study also provides an estimate suggesting that the critical moss lead level corresponds to an early childhood blood lead level of approximately 5µg/dL. This is well below the current blood lead limit of concern (10µg/dL) suggested by the US Center for Disease Control and Prevention (CDC). Since the CDC estimates that more than 310,000 children aged 1-5 in the US alone have blood lead levels exceeding 10µg/dL, and WHO estimate that globally 40% of the urban children suffer from blood lead levels that exceed

[41] Note that this calculation assumes that the lead levels in all the children are lowered from 10µg/dL to 5µg/dL, which implies that the gain is underestimated since a non-negligible share of the children has higher levels than 10µg/dL.

5μg/dL (Fewtrell et al., 2003), reductions in the recommended limit of concern and implementations of further programs designed to reduce lead exposure could potentially be cost effective

A second key result of this study is that while low SES children seem to suffer more heavily from lead exposure in early childhood, the SES differences *does not* seems to be solely caused by differences in pollution exposure due to residential segregation. The SES-gap persists even when comparing children growing up in the same neighborhood. This result indicates not only that environmental policy, such as the ban of leaded gasoline, may additionally function as social policy, but potentially also that public or private investments may potentially mitigate some of the detrimental effects of early life exposure to lead.

In planned future work the same strategy will be used to investigate if early childhood lead exposure even at the relatively low levels found in Sweden can yield similarly sized effects on violent crime rates as those found for the much higher exposure levels considered in Reyes (2007). Other relevant health outcomes such as birth outcomes will also be considered.

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Appendix A: The relationship between lead exposure and blood lead among children

This section briefly review the results from Nilsson, Skerfving, Stroh and Strömberg (2009) that provide the estimated elasticity between lead levels in moss and blood-lead levels in children. The interested reader is referred to that study for further details on the data and execution.

The moss samples Nilsson et al. (2009) use was collected at 55 sites in the municipality of Landskrona in 1983, 1995 and 2006, following the same principles as in the national bio-monitoring program. These data where then matched to the blood lead measurements from about 420 children aged between 7 and 10 collected by Strömberg et al. (1995, 2003) in the year prior to that during which the mosses where sampled. Using the coordinates of the children home address each child is assigned an average lead exposure level using the 5 nearest moss sampling sites.[42] The raw correlation between this lead exposure measure and children's blood lead level is 0.75, which compares very well with findings in previous studies linking ambient air pollution to actual population exposure.

Table A1 report the estimated elasticity between lead in moss and children's blood lead levels using six different versions of the following specification,

$$\ln(blood\_lead)_{it} = \alpha + \gamma \ln(exposure)_{it} + X'\beta + \theta_c + \theta_t + \varepsilon_{it} \qquad \text{(A1)}$$

In the first column of Table A1 the elasticity between B-Pb and M-Pb using the full sample is shown without any additional control variables added to the model. The estimated coefficient suggest that for an 10% increase in the lead level in moss the blood lead level increases with on average 3%. In column (2)-(5), individual characteristics, fixed community, year of sampling and finally year*community fixed effects is stepwise introduced. The year fixed effects seem to be the only control which influence on the estimated elasticity.

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[42] Following Currie and Neidell (2003), in order to assess the accuracy of the air pollution measure Nilsson et al., compare the actual level of pollution at each moss sample site with the level of pollution that they would have assigned using the implemented method (i.e. using the five closest measuring sites), if the actual moss sample was not in fact available. The correlation of the actual and estimated level is high for Pb (r=.88), suggesting that it is an accurate measure for the air pollution exposure for the children's home address. Also note that as long as the measurement errors in assigned and actual exposure are not systematic, the relationship between the children's blood-lead levels and our air pollution measure will be biased towards zero.

Table A1 The relationship between blood lead and moss lead levels

Dependent variable:	ln (B-Pb)	ln (B-Pb)	ln (B-Pb)	ln (B-Pb)	Ln (B-Pb)	ln (B-Pb)
Specification:	(1)	(2)	(3)	(4)	(5)	(6)
Time period:	ALL	ALL	ALL	ALL	ALL	Before 1995
(ln) Lead exposure	.303*** (.034)	.333*** (.035)	.383*** (.037)	.250** (.095)	.287*** (.099)	.440*** (.111)
Individual controls	no	yes	yes	yes	yes	yes
Community F.E.	no	no	yes	yes	yes	yes
Year F.E.	no	no	no	yes	yes	yes
Year*community FE	no	no	no	no	yes	yes
R-squared	0.55	0.69	0.74	0.92	0.92	0.92
Nr of children	410	410	410	410	410	249

Notes: The table reports regression results from of OLS estimations of equation (A1). All in all there are 410 children in 50 cells (249 children and 30 cells in column (6)). The dependent variable is the average blood-lead level at each monitoring point and is weighted with the number of children in each cell. The blood lead is measure in µg/L blood. The lead exposure is µg/kg of moss. The controls are gender, whether the child's practicing any lead exposing hobbies, and ln(hemoglobin) level. The data has been trimmed so to leave out children with blood lead values below the 1st and above the 99th percentiles in each year (7 children in total). Standard errors are reported in parenthesis and are robust with respect to heteroscedasticity. */**/*** reflects significance at the 10/5/1 percent levels respectively. Source: Nilsson, Skerfving, Stroh and Strömberg (2009)

The first five columns report the estimated elasticity using the full sample. However, from 1995 lead in gasoline was banned. Hence as the relative contribution of air lead for total body burden decreases, the predictive power of the moss samples is likely to decrease as mosses only take up lead from the air. This is mirrored in Figure 1 which show that while the moss in lead continued to decrease throughout the observation period, the children blood-lead levels leveled off at around 2 µg/dL after the ban on leaded gasoline. This is pattern is clearly in line with a shift away from air-borne sources as the major source of lead exposure in children after lead was phased out of gasoline. In column (6) we test this notion by estimating the full model, only on the two cohorts sampled before the ban on leaded gasoline, i.e. in 1984 and 1994. When using this restricted sample the estimated elasticity increases to 0.44 while the standard errors increase only marginally. This result suggests that the relative importance of air-lead exposure indeed was stronger in the period prior to the ban than after, as expected. The pre-ban period is also the one focused on in this paper. The final result also provides suggestive evidence on the validity of using mosses as monitors of *air* pollution.

Appendix B: Tables

Table B1: Descriptive statistics

Outcome variables	Definitions	Mean	Std. dev.
GPA	Grade point average (percentile ranked)	50.09	28.8
Low GPA	=1 if GPA in bottom 25%, 0 otherwise	.25	.43
High GPA	=1 if GPA in top 25%, 0 otherwise	.25	.43
IQ	IQ test score (percentile ranked)	50.0	28.5
Low IQ	=1 if IQ in top 25%, 0 otherwise	.25	.43
High IQ	=1 if IQ in bottom 25%, 0 otherwise	.25	.43
Schooling	Year of schooling (imputed)	12.7	1.9
High School	=1 if completed high school, 0 otherwise	.89	.31
University	=1 if ever attended Higher education, 0 otherwise	.33	.47
Earnings	Natural log Labor market earnings	7.2	1.14
Welfare	=1 if receiving welfare, 0 otherwise	.04	.19
Parental characteristics			
% with at least one parent graduated from High school		60	41
% with at least one parent graduated from College		32	47
Sum of parent earnings: SEK 100 (measured in 1990)		2584	1379
Family size		1.5	0.6
Mothers year of birth		1950	0.6
Municipality of birth characteristics:			
Lead exposure (µg/kg)		35	16
Cadmium exposure (µg/kg)		.54	.19
% in childcare average (age 0-6) share of cohort in daycare		13.5	6.9

Table B2: Cross-sectional estimates

Specification	(1)	(2)	(3)	(4)
Sample:	1975	1980	1985	All years
OUTCOMES	N=291,539	N=255,587	N=250,763	N=797,889
GPA	-.0094	-.0298	-.0591	-.0168
	(.0122)	(.0235)	(.0402)	(.0144)
Low GPA	.00023	.00055*	.00042	.0003*
	(.00016)	(.0003)	(.00047)	(.00018)
High GPA	.00006	-.00015	-.0009*	-.0001
	(.00015)	(.00027)	(.0005)	(.00017)
IQ (Men)	-.0258**	-.0395	-	-.0297*
	(.0131)	(.0257)		(.0153)
Low IQ (Men)	.00025	.00035	-	.00029*
	(.00016)	(.00025)		(.00017)
High IQ (Men)	-.00024	-.00043	-	-.00029
	(.00016)	(.00034)		(.00019)
High School	-.00034***	.00009	-.00004	-.00017
	(.0001)	(.00028)	(.00025)	(.00011)
University	-.0002	-.00035	-.0001	-.00024
	(.0002)	(.00045)	(.0004)	(.00023)
Years of schooling	-.0017	-.00106	-.00059	-.00136
	(.0011)	(.0022)	(.0015)	(.0009)
Welfare	-.00006	-.00006	-.0008	-.00012
	(.00004)	(.00014)	(.00019)	(.00008)
Earnings	.00024	.0014***	.00321	.00105
	(.0002)	(.00041)	(.00102)	(.0002)
Teenage mother	-.000016	-.0001	-.00004	-.00005
	(.00007)	(.0001)	(.00012)	(.00006)
Individual characteristics	Yes	Yes	Yes	Yes
Parental characteristics	Yes	Yes	Yes	Yes
Year of birth fixed effects	Yes	Yes	Yes	Yes
Municipality of birth F.E.	No	No	No	No
Mean lead level (μg/Kg)	49.41	30.81	22.77	35.08

Notes: Each row and column represent a separate regression. The reported estimates is the marginal effect of a (1) unit (1 μg/Kg) increase in municipality of birth lead exposure during early childhood. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. */**/*** indicate significance at the 10/5/1 percent levels. Standard errors are clustered at the municipality level (except for the IQ outcomes regressions that only uses two time-periods and hence adjust standard errors at the timperiod-municipality level). Parent's characteristics include maternal education (7-levels) and indicators for quintile of total parental earnings in 1990. The IQ (GPA) outcomes also control for year of enlistment (Graduation) specific effects.

Table B3 Baseline estimates for municipalities above 25%-tile initial (1975) lead exposure: Grade point averages and cognitive test scores.

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	GPA	Low GPA	High GPA	IQ	Low IQ	High IQ
Sample	ALL	ALL	ALL	Males	Males	Males
Lead exp. (µg/Kg)	-.0356**	.0006***	-.0003	-.0336***	.0003**	-.00026**
	(.0143)	(.0002)	(.0002)	(.0128)	(.00015)	(.00012)
R-squared	0.22	0.12	0.14	0.17	0.09	0.1
Mean of dep. var.	50	025	0.25	49.8	022	0..26
Individ. controls	Yes	Yes	Yes	Yes	Yes	Yes
Y. of birth F.E.	Yes	Yes	Yes	Yes	Yes	Yes
Muni. F.E.	Yes	Yes	Yes	Yes	Yes	Yes
# Obs.	668,909	668,909	668,909	220,324	220,324	220,324

Notes: The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

Table B4 Baseline estimates for municipalities above 25%-tile initial (1975) lead exposure: Educational attainments and other long-term outcomes

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	High School	Ever in College	Yrs. in School	Log earnings	Welfare	Teen mother
Sample	ALL	ALL	ALL	ALL	ALL	ALL
Lead exp. (µg/Kg)	-.00033*	-.0002	-.0022*	-.0009**	.00014	.0001
	(.00017)	(.0002)	(.0012)	(.0005)	(.00009)	(.0001)
R-squared	0.06	0.18	0.20	0.1301	0.03	0.03
Mean of dep. var.	0.89	0.32	12.7	177,283	0.037	0.042
Individ. charact.	yes	yes	yes	yes	yes	yes
Year of birth	yes	yes	yes	yes	yes	yes
FE muni.	yes	yes	yes	yes	yes	yes
# Obs.	696,690	696,690	696,690	601,774	696,690	325,010

Notes: The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

Table B5 Alternative sample restrictions

Specification	(1)	(2)	(3)	(4)
Sample:	Exclude <25%-tile *initial* lead exposure	Exclude <25%-tile *change* in lead exposure	Exclude >75%-tile *initial* lead exposure	Exclude >75%-tile *change* in lead exposure
OUTCOMES	N=696,690	N=665,116	N=572,019	N=565,758
GPA	-.0356**	-.0326**	.0120	.0121
	(.0143)	(.0143)	(.0171)	(.0157)
Low GPA	.0006***	.00052***	-.0003	-.0003
	(.0002)	(.0002)	(.0002)	(.0002)
High GPA	-.0003	-.00025	-.0001	-.00016
	(.0002)	(.0002)	(.0002)	(.0002)
IQ (Men)	-.0336***	-.0322**	.0265	.0248
	(.0128)	(.0129)	(.0190)	(.0186)
Low IQ (Men)	.0003**	.0003	-.0002	-.0001
	(.00015)	(.0002)	(.0003)	(.0003)
High IQ (Men)	-.00026**	-.0003	.0004	.0004
	(.00012)	(.0002)	(.0003)	(.0003)
High School	-.00033*	-.00025*	-.0001	-.0001
	(.00017)	(.00015)	(.0002)	(.0002)
University	-.0002	-.00028	.00005	-.0001
	(.0002)	(.00024)	(.00046)	(.0004)
Yrs. of schooling	-.0022*	-.0019*	.0011	.0002
	(.0012)	(.0010)	(.0020)	(.0018)
Welfare	.00012	.0001	.0001	.0001
	(.0001)	(.0001)	(.0001)	(.0001)
Earnings	-.0009**	-.0009***	.0023***	.0021***
	(.00045)	(.0005)	(.0007)	(.0007)
Teenage mother	.0001	.0001	-.0003***	-.00014
	(.0001)	(.0001)	(.00013)	(.0001)
Individ. char.	Yes	Yes	Yes	Yes
Parental & muni. characteristics	Yes	Yes	Yes	Yes
Y. of birth F.E.	Yes	Yes	Yes	Yes
Mun. of birth F.E.	Yes	Yes	Yes	Yes

Notes: Each row and column represent a separate regression. The reported estimates is the marginal effect of a (1) unit (1 μg/Kg) increase in municipality of birth lead exposure during early childhood. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. */**/*** indicate significance at the 10/5/1 percent levels. Standard errors are clustered at the municipality level (except for the IQ outcomes regressions that only uses two timeperiods and hence adjust standard errors at the timeperiod-municipality level). Parent's characteristics include maternal education (7-levels) and indicators for quintile of total parental earnings in 1990. The IQ (GPA) outcomes also control for year of enlistment (Graduation) specific effects.

Essay 2: Does a Pint a Day Affect Your Childs' Pay? Unintended and Permanent Consequences of a Temporary Alcohol Policy Experiment♠

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.

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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 beer[1]. 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 substitutes[2], 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 (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.

[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.

[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.

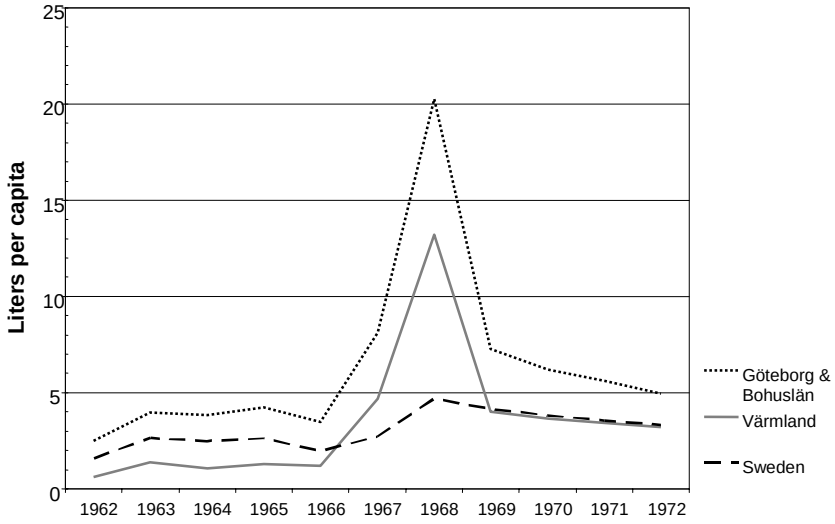

Figure 1 Yearly strong beer consumption per capita. *Source*: SCB 1962-72

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] 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

[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 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.

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 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.

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[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.

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

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 exceptions[9], 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.

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

[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.

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 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

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[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.

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]

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

[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.

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 beer[16] 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 the implementing authority, the Alcohol Policy Commission, surveyed the local child welfare commissions (barnavårdsnämnder), 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 im-

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

mense 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 % 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 début occurred before

[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.

[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.

turning 21 and that the abstainer rates in these age categories was low[20] (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 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,

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[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.

[24] See e.g. CDC (2004).

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 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

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[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.

weekend binge drinking pattern common in Sweden[28], 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 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

[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. Kühlhorn 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 asses 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.

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 where 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 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 substan-

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[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.

tial 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,

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

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 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

[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.

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. $\varepsilon_{c,t,mom<21}$ 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.

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, com-

[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 chil dren 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.

pared 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 earnings[39] 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 hands 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.

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[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 (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	12.4	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.

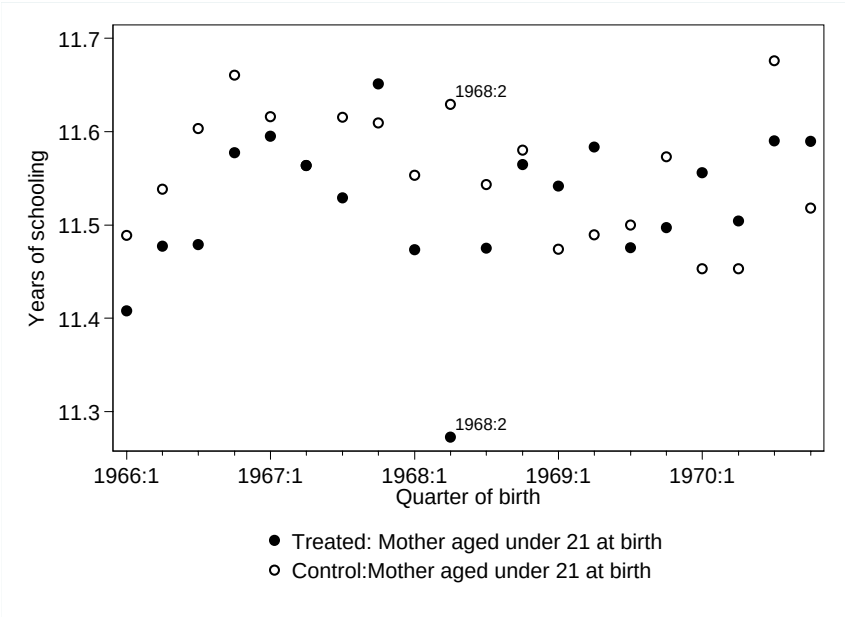

Figure 2 Average years of schooling, treated vs. control.

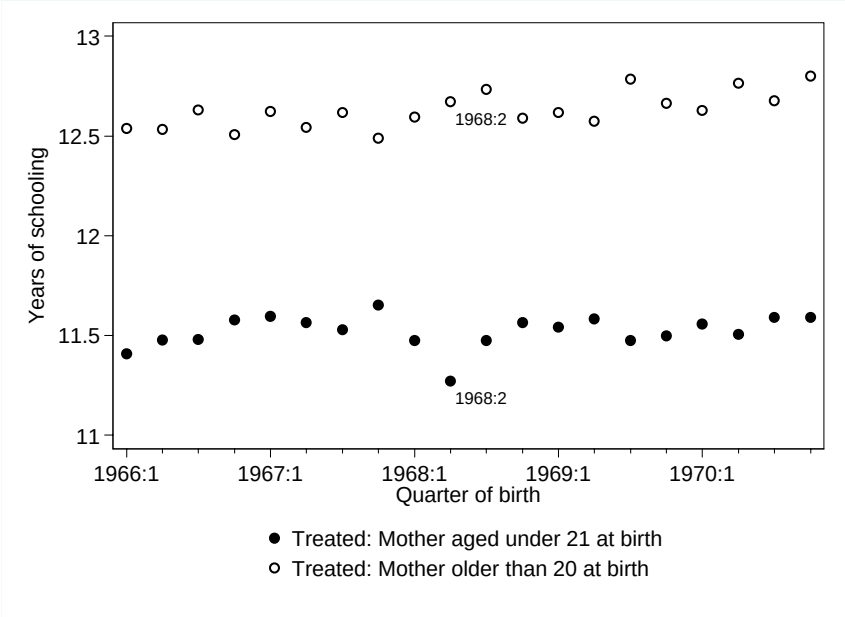

Figure 3 Average years of schooling, young vs. old mother.

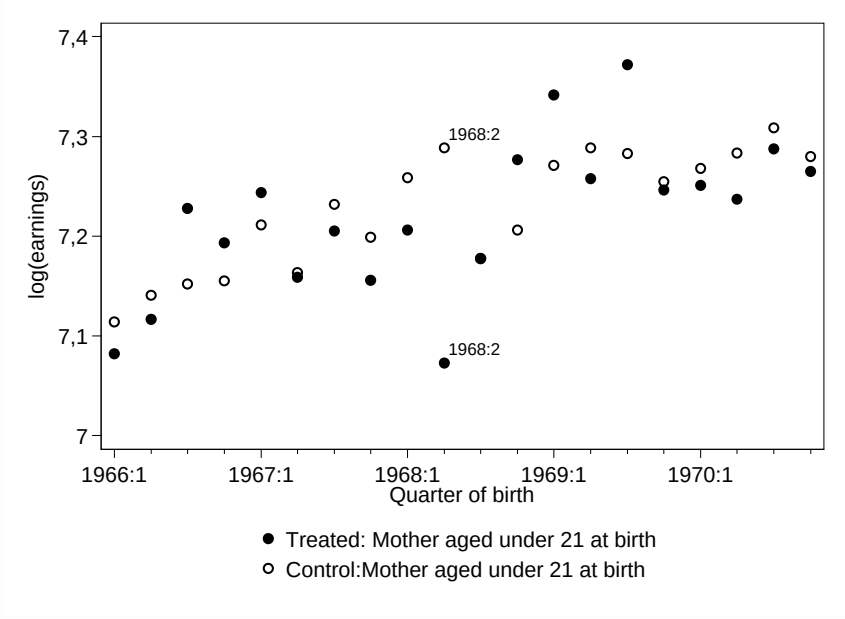

Figure 4 Average ln(earnings) at age 32, treated vs. control.

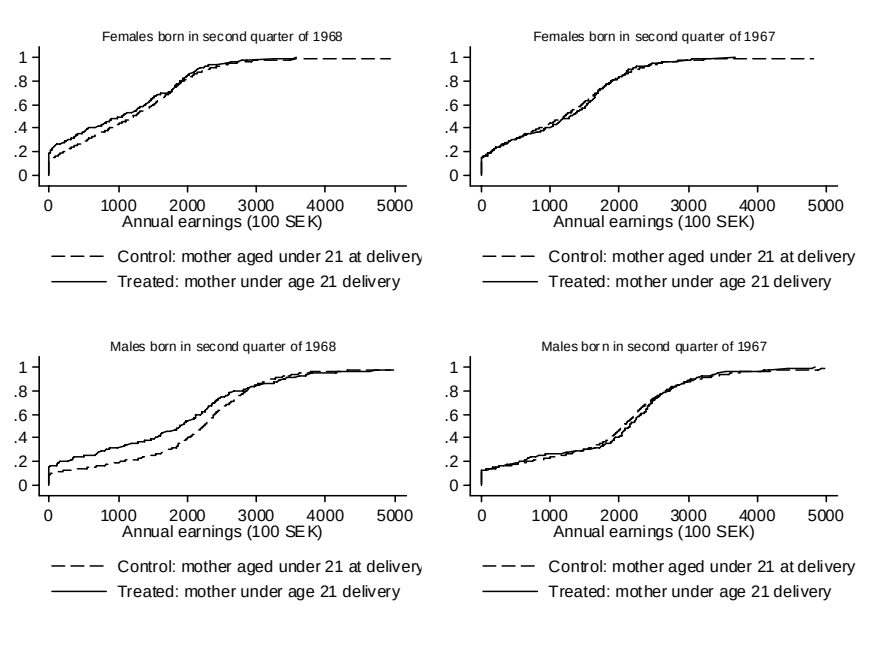

Figure 5 Cumulative earnings distribution at age 32. Left column presents earnings for women (top) and men (bottom) born during the second quarter of 1968. The right column shows the same distributions for children born during the second quarter of 1967 (i.e. before the experiment).

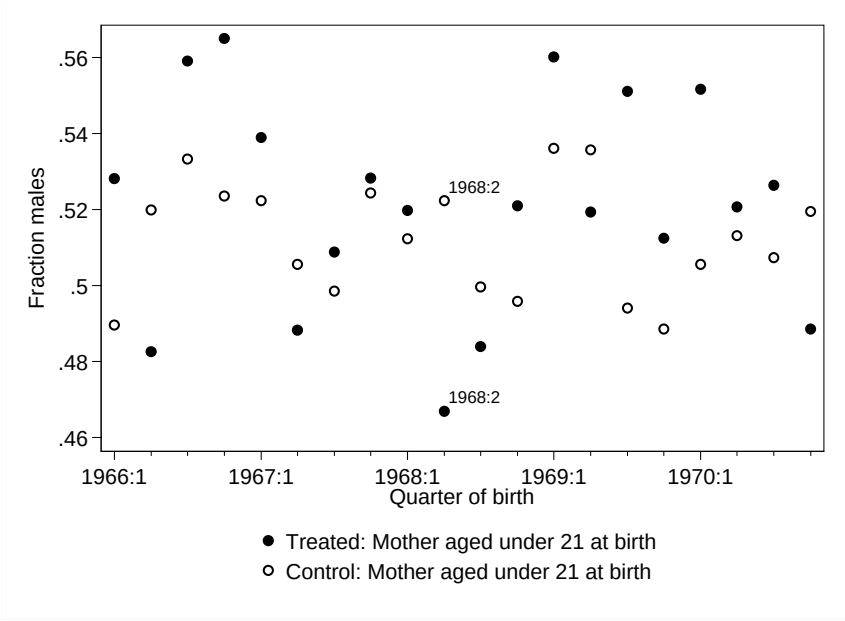

Figure 6 Proportion of males in 2000

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

A. Dependent variable: Years of schooling	Sample		
	All (1)	Men (2)	Women (3)
***In utero* (month 1-4)**	–0.266***	–0.473***	–0.101
	(0.049)	(0.124)	(0.151)
Observations	1350	1350	1350
R-squared	0.98	0.96	0.95
Mean	12.33	12.18	12.49
B. Dependent variable: Fraction high school graduates	Sample		
	All (1)	Men (2)	Women (3)
***In utero* (month 1-4)**	–0.039***	–0.092***	0.015
	(0.009)	(0.017)	(0.014)
Observations	1350	1350	1350
R-squared	0.90	0.85	0.82
Mean	0.92	0.91	0.93
C. Dependent variable: Fraction graduated from higher education	Sample		
	All (1)	Men (2)	Women (3)
***In utero* (month 1-4)**	–0.025**	–0.039***	–0.021
	(0.012)	(0.013)	(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

	Sample		
A. Dependent variable: 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)
Observations	1350	1350	1350
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)
Observations	1350	1350	1350
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)
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 earning 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

	Dependent variables											
Panel A.	Years of schooling		High school graduates		Higher education		Earnings		Zero earnings		Welfare	
Education of mother (1990)	High	Low	High	Low	High	Low	High	Low	High	Low	High	Low
***In utero* (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
***In utero* (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 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)	0.130* (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

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[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.

equal to 1 for the cohort of children born between April and July 1968 by 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 exluded from these regressions.

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

A.	Dependent variables: Labor and education											
	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	– 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

B.	Dependent variables: Health				
	Fraction males	Month of birth		ln(cohort size)	
Sample	ALL	Men	Women	Men	Women
In utero	– 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 exposed cohorts. For the educational outcomes, the strongest negative effect is reached some-

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)
***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	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)
***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	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 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 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
***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	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
Outcome:											
Yrs. 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)**	(0.095)	(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 dep.	–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)
# 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).

where 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]

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[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.

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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.

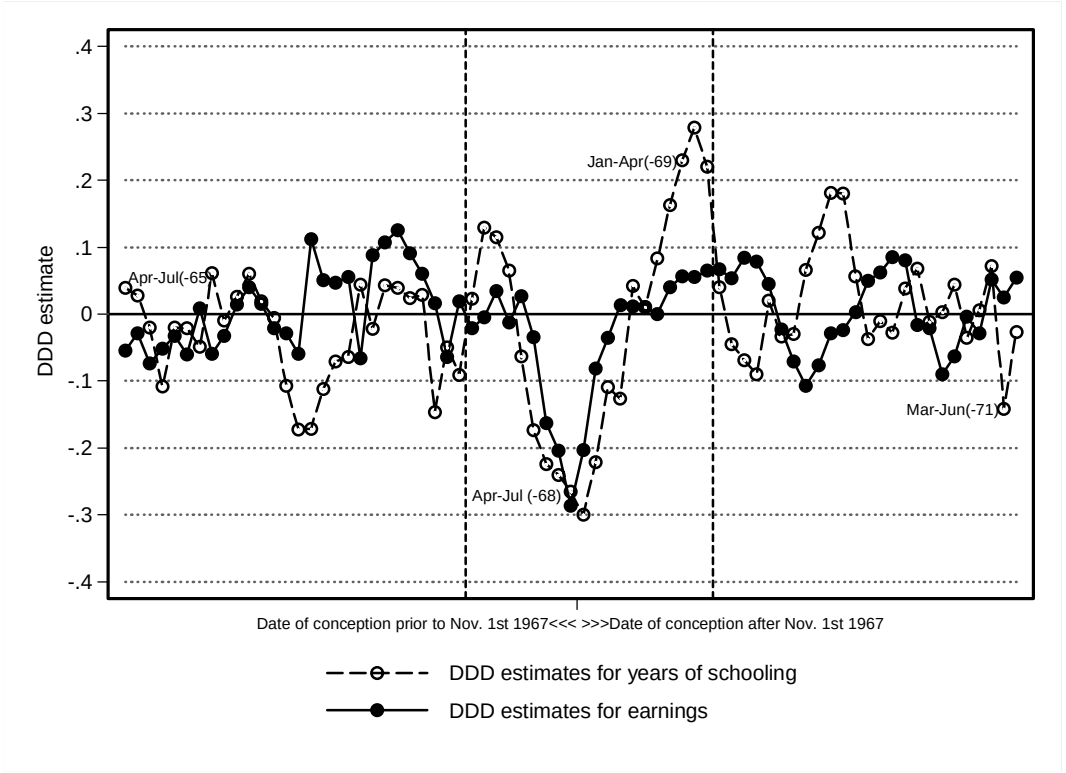

Figure 7 DDD estimates for years of schooling and earning

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***	–0.044	–0.037**	–0.151	0.063**	–0.013
	(0.137)	(0.032)	(0.019)	(0.121)	(0.031)	(0.025)
R-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 prental 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 differences in the impact

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

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.

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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.

Essay 3: Sick of Your Colleagues' Absence?[†]

Co-authored with Patrik Hesselius and Per Johansson

1 Introduction

A substantial amount of theoretical work has suggested that social interactions within the workplace are an important determinant of worker effort and firm productivity.[1] Recently a growing empirical literature, using matched employer-employee data, has aimed at identifying to what extent social preferences affect productivity in practice. Due to the lack of consistent and reliable measures of effort/productivity across firms, previous studies have been forced to examine social interactions between co-workers' effort using data from single firms or occupations.[2] While using high quality data from single firms sometimes enhances the possibility to tease out what type of mechanisms are underlying the social interaction effect, clearly, the evidence provided by case studies might be difficult to generalize to other populations.

Therefore, in order to shed further light on how co-workers affect each other's behavior we take an alternative approach compared to previous studies. We focus on how co-workers affect each other's effort in the form of

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[1] For example Lazear (1989), Kandel and Lazear (1992), and Rotemberg (1994) incorporate social concerns into the analysis of behavior within firms.

[2] For example in four recent, interesting and related studies Bandiera, Barankay and Rasul (2005); Mas and Moretti (forthcoming); Guryan, Kroft and Notowidigdo (2007); Kato and Shu (2008) use data from a fruit picking farm in the UK, 6 US supermarket stores, male professional golfers on the PGA tour, and a Chinese textile firm respectively.

work absence. Work absence is, of course, intimately related to the productivity of the firm. This relationship is perhaps most obvious in firms with just-in-time type of production technologies. In such firms the output loss associated with the unexpected absence of one worker is not simply equal to the loss of the single worker's output, but potentially also the lost total value of the entire downstream production line. Moreover, in many jobs both management and co-workers may experience difficulties in observing whether an employee shirks or not. The pattern and frequency of work absence is however arguably more easily monitored and may serve as a proxy for the worker's effort. The relative ease of monitoring co-workers' absence suggests that studying this type of behavior should be rewarding when trying to investigate whether and through which underlying mechanisms co-workers affect each other's effort.

To address many of the severe identification problems associated with the estimation of social interaction effects we make use of the exogenous variation in work absence incentive induced by a large-scale randomized experiment.[3] During the experiment, formal monitoring during absence was relaxed for half of all employees in the city of Göteborg, Sweden. Instead of having to provide a physicians certificate on the eighth day of an absence spell, the treated individuals did not need to provide a certificate until the 15th day of the spell. Treatment assignment was based on date of birth (even/uneven) and applied to all employees living in Göteborg municipality (pop.~500,000). As treatment was determined at the individual level, the number of treated workers within each workplace varied substantially. Using a rich administrative data set we are able to recover the treatment status of all workers, and hence the proportion treated, within each one of the around 3,000 workplaces in operation in Göteborg during the experiment.

We start off by showing that the experiment strongly affected the short term absence level among the treated workers in our sample.[4] We then focus on whether social preferences affect short-term absence. Our idea for testing for the prevalence of social preferences among the employees is straightforward. We first show that conditional on treatment status the proportion of treated within each workplace is strongly correlated with the change in individual worker's absence. This result suggests that co-workers indeed have an important influence on employees' behavior.

We then examine the heterogeneity of the treatment effect to explore what types of social preferences are most likely underlying the social interaction effect. Interestingly, we find stark differences in the effect of having many

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[3] See e.g Manski (1993, 1995) for a description of the difficulties in estimation of social interaction effects.

[4] Hesselius, Johansson and Larsson (2005) find a statistically significant and large effect on average duration of absence due to the experiment.

treated co-workers depending on the worker's own treatment status. While the proportion treated at the workplace is a good predictor for the change in absence level among the *non-treated*, no significant effects can be found among the *treated*. The heterogeneous spill-over effects suggest that the observed co-worker effect is *not* driven by preferences for joint leisure. Neither does it seem in line with a hypothesis suggesting that information sharing among co-workers is causing the observed peer effect. Given the randomized treatment assignment, if either of these two mechanisms would be responsible for the estimated peer effect we would expect that also the treated employees absence level should be correlated with the share of treated at the workplace. The heterogeneous response by treated and non-treated workers instead suggest that a non-negligible proportion of the workers have reciprocal type of preferences and/or display fairness concerns.[5]

The prevalence of social preference has previously been documented in several laboratory experiments. The evidence of such preferences outside of the laboratory is however more scarce. Our study complement and extend the evidence on the prevalence of social preferences in the workplace found by Bandiera, Barankay and Rasul (2006), Mas and Moretti (forthcoming), and Ichino and Maggi (2000). These studies all use observational data from single firms. Our main contribution to the literature on social preferences and worker effort is to provide evidence on the prevalence of similar social mechanisms at work using a randomized social experiment and linked employer-employee data from around 3,000 workplaces. These two features significantly improve both the internal as well as the external validity of the results found.

The paper unfolds as follows. In Section 2 we briefly describe the general context of our study, providing details on the Swedish sickness insurance system and the experiment. In Section 3 the data and empirical strategy is presented. Section 4 contains the results and Section 5 concludes.

2 Institutional background and the experiment

2.1 Temporary work absence in Sweden

In Sweden sickness insurance is compulsory and universal to all employees, students and unemployed. It is financed by a proportional pay-roll tax and

[5] See e.g. Rabin (1993), Fehr and Gächter (2000) for references and discussions on the importance of such preferences in the labor market.

replaces individuals earnings lost due to temporary health problems. The benefit level received is related to the loss of earnings during the absence spell.

In an international context the sickness benefit levels are, and have been, generous. For most workers the benefit level was set to 90% of previous earnings. Some workers at the very top of the wage distribution were however excluded from receiving the full 90% due to a benefit cap. Besides the public insurance, most Swedish workers are also covered by extra sickness insurance regulated in agreements between the unions and the employers' confederations. These top-up insurances generally cover about 10% of the lost earnings but there is considerable variation. Hence the total compensation in case of work absence due to illness could be fully 100%.[6]

The public insurance has no limit for how long and how often sickness benefits are paid. Many spells stretch over a full year and there are examples of even longer durations. The individual have a high degree of discretion when to report sick. The benefit payments are generous, and the monitoring before the eighth absence day is lax. A sickness absence spell starts when the worker calls the public insurance office (and her employer), then within a week (on the eighth day) he/she should confirm her eligibility with the insurance office by presenting a medical certificate proving reduced work capacity due to illness. The public insurance office reviews the certificate and then declines or approves *further* sick-leave. In all but *very* few cases the certificate is approved. In case the insurance office suspects abuse they can make unannounced visits to the claimant's home.

2.2 The experiment

In the fall of 1988 the regional social insurance board in the municipality of Göteborg, the second largest city in Sweden, and in Jämtland, a large and relatively sparsely populated region in the north of Sweden, agreed on performing a social experiment regarding the timing of the requirement for a physician's certificate. A randomly assigned treatment group was allowed to be absent from work due to illness for 14 days before they needed a physician's certificate in order to continue their absence spell with insurance compensation. The control group faced the ordinary restriction of seven days. Individuals were assigned to the treatment and control group based on their date of birth. Those born on even days ended up in the treatment group, and those born on uneven days in the control group. Hence, due to the universal insurance coverage, everyone who was of working age and lived in

[6] See e.g. Johansson and Palme (1996, 2002, and 2005), Henreksson and Persson (2004) for studies on effects of different compensation schemes on individual work absence.

the experiment regions was assigned to either the control or the treatment group.[7]

The insurance agencies had several arguments for running the experiment. All were based on a notion that extending the time-period without monitoring would *decrease* costs and *reduce* sickness absence. The main argument was that with the 15-day restriction unnecessary visits to a physician could be avoided, which would cut costs for both the individual and the public health care system. The insurance agency also believed that physicians by routine prescribed longer absence from work than necessary. With an extended certificate-free period of two weeks many individuals would have time to return to work before a medical certificate was needed, and thus individual and public costs would be reduced.

The experiment started on 1 July 1988 and besides the personnel at the social insurance office, all employers and medical centers were informed before or during the experiment. A massive information campaign also preceded the experiment at the two locations, including mass-media coverage, distribution of pamphlets and posters at workplaces, etc. Short information about the experiment was also written on the form that every insured reporting sick needed to fill in and send to the insurance office to receive sickness benefits. The experiment ended on 1 January 1989, at which point the previous system was resurrected.

Hesselius, Johansson and Larsson (2005) show that absence spell durations increased substantially among the treated group compared to the control group. The characteristic spike in exit rates from absence, which before the experiment typically occurred on day 7, was during the experiment postponed and instead occurred on day 14 of the spell (i.e. just before the need for a physicians certificate). They also report heterogeneous treatment effects between women and men. Men prolonged their work absence spells more than women.

The estimates provided by Hesselius et al. did however not control for potential spill-over effects between the treated and the controls. Whether doing so yields an upward or downward biased estimate of the true treatment effect is *a prior* uncertain. The direction of the bias will depend on if individuals care about e.g. co-workers' behaviour and if so *why* they care. In the following section we provide evidence on the effects of co-workers behaviour on individual absence decision and the reason why.

[7] Government employees were exempted, as they, by law, receive their sick leave compensation from the employer instead of from the social security office. The employer, in turn, receives the benefit from the social security office. We hence exclude all Governmental workplaces.

3 Data and empirical strategy

We use data from a set of administrative registers compiled by Statistics Sweden. The data contains, besides a set of individual background characteristics, data on start and end date of all absence spells during 1988. We also observe the workplaces where the individual is gainfully employed in November 1988. A few individuals have multiple workplaces, but for simplicity we assume that the workplace from which the highest yearly earning is received is also the main arena for co-worker interaction. The treatment status of each worker was decided by date of birth (even/uneven) and whether the individual is residing in Göteborg municipality or not. Figure 1 show the distribution of treated workers at the workplaces in Göteborg municipality.

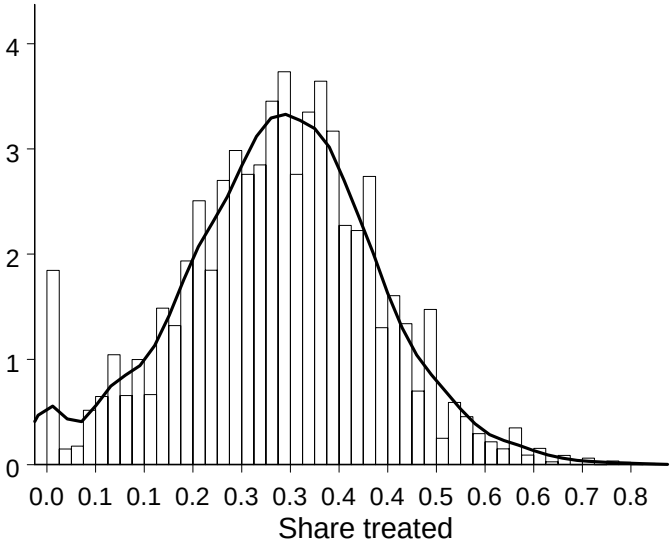

Figure 1 Distribution of treated workers at workplaces in Göteborg

As seen in Figure 1 the between workplace variance in proportion of treated is considerable. The average workplace has around 35% of treated workers. The variation in proportion treated workers stem from the random assignment of treatment, but also from the number of commuting co-workers. In the main analysis we focus on workplaces with between 10 and 100 employees as social interactions is probably most prevalent in small to medium sized workplaces. The workplaces with 10 employees and less are excluded from the sample as alternative rules may apply to these workplaces.

The large variation in proportion treated workers across workplaces provides us with a close to ideal setting to identify the effects of co-worker interactions. Our idea to identify the prevalence of co-worker interactions is straightforward. Given the random assignment of treatment status, if the proportion treated within each workplace affects individual work absence decisions we can be certain that co-workers indeed affect each other's behavior. The baseline model we estimate by OLS is specified in equation (1)

$$\Delta s_{ij} = \alpha_0 + \beta_0 treated_i + \beta_1 \pi_j + \varepsilon_{ij} \quad (1)$$

where Δs_{ij} is the change in number of days of short term absence (shorter than 15 days) between first and second half of 1988 of employee *i* at workplace *j*.[8] Treated takes the value 1 if the employee resides in Göteborg and is born on an even date, and 0 otherwise. π_j is the proportion of treated co-workers at employee i's workplace (excluding employee *i*). Given the first differencing and the random assignment of treatment, a significant estimate of β_1 identifies spill-over effects among the employees.[9] Inference is based on robust standard errors allowing for clustering at the workplace level.

4 Results

4.1 Main results

In column (1) of Table 1 we start by presenting the "naïve" estimate of the direct treatment effect, β_0, from a specification using only the treatment status as explanatory variable. The estimated effect suggests that being assigned to treatment increases the short-term work absence with 0.88 days on

[8] The empirical analysis focuses on the change in work absence to control for the possibility that workplaces with different shares of commuters also are systematically different in some unobserved way. The first differencing approach controls for time-invariant unobserved individual and workplace heterogeneity.

[9] Note that only if we only would use the directly non-treated individuals when estimating equation (1) this specification could be seen as a reduced form in the estimation of "endogenous effects" (see Manski, 1993). This second stage structural equation would, among others, require rational expectations of the individuals, that is; the correct prediction of the co-workers response from the more lenient monitoring. Equation (1) does not require that the observed reaction to the fraction of treated is due to an actual increase in absence among treated co-workers. Hence, (non-rational) expectation of an increase in shirking among treated co-workers during the experiment may have an effect on behavior.

average. In column (2) of Table 1 both estimates of β_0 and β_1 from equation (1) is reported. The share of treated co-workers indeed increases the short-term absence level substantially.

The finding that the share of treated co-workers within a workplace affects individual worker's absence decisions provides clear evidence on the importance of peer effects in the workplace. The indirect effect is furthermore sizable in relationship to the direct effect of being treated. An increase in the proportion of treated colleagues from 0 to 1 increases the change in absence by 0.55 days in average. Both the direct and the indirect treatment effects are furthermore substantial given that during the first half of 1988 the average number of short-term absence days was 2.32 in our sample.

Table 1 Direct and indirect effects of experiment

Specification	(1)	(2)	(3)
Sample:	All	All	All: pre-experiment
Treated	.886***	.889***	.061
	(.057)	(.058)	(.050)
Proportion treated		.554**	-.134
		(.210)	(.193)
R-squared	.0035	.0036	.000
# observations	79,643	79,643	77,647

Notes: Dependent variable is the change in number of days in short term absence (spells shorter than 15 days). */**/*** denoted statistical significance at 10/5/1 percent level respectively. The number of co-workers, gender, age and annual earnings is included as control variables. Standard errors are reported in parenthesis and are cluster adjusted at the workplace level. Number of workplaces is 3,008 for the experiment period and 2,910 for the pre-experiment period (1987).

We have also performed a number of specification checks in order to assess the robustness of the results. First we re-ran the same analysis using data from the first and second half of 1987, i.e. the year prior to the experiment. The result from this exercise is presented in column (3) of Table 1. The effects of the share of treated and individual treatment are both statistically insignificant and in the case of share of treated co-workers the sign is changed. This is in sharp contrast with the effect on absence during the experiment. That is, in the year prior to the experiment the share of employees being born on an even date had no significant effect on absence, but a large positive effect during the experiment. This result strengthens our confidence in the validity of the estimation strategy.

We have also re-estimated the same models for different workplace sizes and also used the change in total number of absence days as well as the change in number of absence days in spells shorter than eighth days as the

dependent variable.[10] These changes in specification yielded qualitatively similar results. In the smallest workplaces (10-20 employees) considered the estimated spill-over effect is largest and then decreases monotonically as we stepwise enter larger workplaces. We have also checked for nonlinearities by adding a quadratic term in the share of treated to the specification. These estimates suggested that there is a tendency towards a concave relationship (significant at 10% level) between work absence and the share of treated co-workers (maximum when 42% of the co-workers are treated). Using the natural log of the share of treated instead of the share of treated yielded similar results.[11] Finally, we also estimated separate models for males and females. The separately estimated models produce slightly higher and more precise estimates for males than for females.

4.2 What mechanisms are underlying the spill-over effect?

Many studies have found that social interactions matter for individual behavior in various settings. Very few studies have however with any certainty been able to tell what type of mechanisms is underlying the effect of peers.[12] This is naturally a much more difficult question to answer empirically. We believe that the spill-over effects found in this setting can be explained in three ways: 1.) *Joint leisure:* Co-workers may form tight bonds and hence enjoy leisure time together. A higher absence level as a result of a higher proportion treated co-workers could therefore be explained by preferences for joint leisure time. 2.) *Information sharing:* Prior to the experiment, information about the absence leave system may have been incomplete. A higher proportion of treated workers may therefore be correlated with average absence level at the workplace simply because more workers now become aware of how the system works. However, given the large information campaign about the setup of the experiment in action prior to and during the experiment, the importance of co-workers as an information channel is probably reduced in this context. We therefore do not expect information sharing between co-workers to have a large effect on work absence. 3.) *Reciprocity/Fairness:* Observing a sudden increased absence level among treated co-workers may induce resentment and lead to ill feelings towards the shirking co-workers. For example, if the workload of the absent worker is shifted to the remaining workers, the absence of a co-worker is costly as

[10] These results are retained due to space limitations but are available upon request from the authors.

[11] In this specification the fraction of treated was multiplied by 100 and those without any treated were then assigned as having 1% treated before taking the logs.

[12] Two notable exceptions are Mas and Moretti (forthcoming) and Bandiera, Barankay and Rasul (2005).

the remaining workers may need to increase their effort. If shirking co-workers induces such costs, formal or informal social sanctions of this behavior might be warranted. While there is a number of imaginable ways through which punishment may take place, a natural retaliation could be to increase one's own absence level for fairness reasons.

The two first hypotheses are more in line with altruistic type of social preferences while the third is more in line with non-altruistic social preferences. Moreover, while the first two hypotheses arguably predict similar (or smaller) effects on non-treated and treated, the third hypothesis suggests that non-treated should respond more strongly to an increasing proportion of treated co-workers.[13]

Table 2 Heterogeneous spill-over effects

Specification	(1)	(2)	(3)	(4)
Dependent variable	#(<8 days)	#(<8 days)	#(<15 days)	#(<15 days)
Sample:	Treated	Non-treated	Treated	Non-treated
Proportion treated	-.023	.365***	.270	.600***
	(.249)	(.141)	(.457)	(.223)
# Observations	23,803	55,840	23,803	55,840

Notes: Dependent variable is the change in number of days in short-term absence less than 8 and 15 days in columns (1),(2) and (3),(4) respectively. The number of co-workers, gender, age and annual earnings is also included as a control variables */**/*** denotes statistical significance at 10/5/1 percent level respectively. Standard errors presented in parenthesis are clustered at the workplace level. Number of workplaces is 3008.

The results presented in Table 2 help us distinguish which one of the suggested explanations is most likely driving the observed spill-over effect. Column (1) and (2) reports the estimate of β_1 from equation (1) for the treated and non-treated employees separately. Interestingly, the share of treated has a negligible and insignificant effect on the treated workers' absence decisions. On the contrary, for the non-treated workers we find a large and significant effect of having many treated co-workers. Columns (3) and (4) display that the same pattern holds when instead the change in number of days in spells shorter than 15 days is used as the dependent variable.

The results in Table 2 clearly point towards the *reciprocity* hypothesis and away from the other two hypotheses suggested; *joint leisure* and *information*

[13] An additional channel through which an increase in work absence among co-workers might affect individual work absence is through direct negative effects on health; from e.g. the stress of facing an increased workload. Although we cannot completely rule it out, we believe that the short duration of the experiment and our focus on short-term absence most likely diminishes the risk of such effects to have any major influence on the estimates. Also, note that such a mechanism would probably yield a similar effect on both treated and non-treated.

sharing. If joint leisure was the main motivator behind the response to the share of treated within the workplace, due to the random assignment of treatment, we would expect that not *only* the non-treated reacted but *also* the treated co-workers. As seen above this is not the case. The same argument holds for the information sharing hypothesis. Information sharing should increase absence not *only* for the non-treated but arguably also the treated. The large information campaign implemented prior to and during the experiment as discussed above probably reduced the importance of treated peers as an information channel. The relatively weak effects among treated and strong effect among non-treated therefore more likely reflect reciprocal type of social preferences or fairness concerns among the workers in our setting.

However, as pointed out by e.g. Sobel (2005), it should be noted that due to the possibility of repeated interactions it is notoriously difficult to distinguish between reciprocal social preferences and pure self-interest outside of the laboratory. At the very least, as for Mas and Moretti (forthcoming) and Bandiera, Barankay and Rasul (2005), our results suggest that *pure altruistic* social preferences can be ruled out as an explanation for the interaction effect in behaviour among co-workers.

5 Concluding remarks

We provide preliminary evidence suggesting that social interactions are an important determinant of worker effort as measured by work absence by using data from a social experiment affecting work absence incentives of 70,000 employees in 3,000 workplaces. As previous evidence on the prevalence of social preferences comes from laboratory experiments or from studies using observational data from *single* firms, our study constitutes a significant contribution to the current literature on spill-over effects at work.

It should be noted that the reduced form analysis we employ here prohibits us from drawing definite conclusions about the exact nature of the underlying causes of the observed social interaction effects. In future work we plan to apply a more structural approach, and also use data on the exact timing of the absence spells for treated and non-treated in order to get a better understanding of the nature of the underlying social preferences.

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Essay 4: Businesses, Buddies and Babies: Social Interactions and Fertility at Work♠

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1 Introduction

Remarkable fluctuations in fertility rates across time and space in both developing and developed countries are well documented (see e.g. Bongaarts and Watkins, 1996; Kohler, 2000). Sweden for example experienced several baby-booms and baby-busts during the 20th century (see Figure A1). A longstanding debate exists among social scientists over the causes of similar sudden changes in fertility both in developing and developed countries. Economists have traditionally focused on the effect of price and income in determining individual's fertility decisions (Becker 1960; Mincer 1963; Mincer and Polachek, 1974; Easterlin 1975; Heckman and Walker, 1990; see Schultz, 2001 for a review). Sociologist such as Cleland and Wilson (1987) have however contested the view that e.g. the classical fertility transition was due to changes in parental demand for children based on shifts in economic costs and benefits of childbearing. Much of the changes in aggregate fertility seem to occur so rapidly that they are unlikely to be explained by slow moving changes in economic conditions. Social interaction effects have instead been put forth as a complementary explanation, since it is believed to be able amplify the effects of small changes in e.g. underlying economic determinants (Montgomery and Casterline, 1996; Kohler, 2000).

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While previous studies on the role of social interaction effects in fertility typically consider the individual or the family as the primary decision-maker, surprisingly few studies have used micro data to test for the prevalence and importance of fertility spill-over effect in practice. Those studies that have used micro data either looks at interactions within developing countries, among very young women or within families. Moreover, the main interest has been focused either on intermediate outcomes such as contraceptive use, or on completed fertility. Further research is therefore clearly warranted in order to better understand if and how social interaction effects in fertility contribute to the high variation in childbearing across space and time.

Therefore, in order to shed further light on how social influences affect childbearing this paper focus on a tightly defined peer group whose role has previously not been considered: co-workers. Co-workers may constitute a particularly important peer group as most of us interact with this group on a day-to-day basis. Moreover, the frequency of exposure to childbearing co-workers is typically fairly high. Additionally, since childbearing represents one of the most prevalent and also one of the more costly types of career interruptions for women (see Edin et al., 1999; Bertrand, Goldin and Katz, 2009) the behavior of co-workers may be particularly influential. For example, employees may draw inference about the job-specific consequences of childbearing from the previous experiences of her co-workers.

Our main analysis is based on fertility decisions among half of the Swedish women in childbearing ages and *all* of their co-workers during an eight year period. The matched employer-employee data provides besides a rich set of background characteristics also the *complete* fertility history on a monthly basis for the individual and her co-workers, prior to and during the observation period. The unprecedented richness of the data allows us to thoroughly examine many of the pathways through which social interaction effects may affect fertility decisions. Importantly, the high frequency of the data allows us to focus on the *timing* of childbearing, which potentially is the key margin through which social influences affects fertility.

Mainly two econometric issues arise when attempting to identify the effect of co-workers' childbearing on individual childbearing (c.f. Manski, 1993; Moffitt, 2001). The first is the simultaneity bias (or the "reflection problem") generated by the simultaneous feedback from the focal subject to the group. The second problem is that of omitted variable bias. In our setting, it is for example essential to consider that family friendliness of jobs is a potentially important determinant of many women's employment decisions (Herr and Wolfram, 2009), and that that friends and relatives are a well utilized way to find jobs (c.f. Montgomery, 1991; Ioannides and Loury, 2004). It is therefore of crucial importance to address the concern that the estimated peer effect is not simply caused by endogenous sorting of workers with simi-

lar preferences or other unobserved determinants of childbearing in common across firms. Similarly, unobserved shocks that independently affect both the timing of individual and co-workers' fertility decisions could also lead to a spurious correlation between peers behavior. For example, changes in co-workers' childbearing could simply proxy for changes in firm policy, or an increased risk of mass lay-offs, etc.

The richness of the data and the possibility to focus on the timing of childbearing help us to address these identification problems. The simultaneity problem is in this context mitigated by focusing on the influence of co-workers past childbearing. Using lagged behavior of a reference group to identify the effect social influences is in general not a fail-proof plan, since it essentially requires the fairly strong assumption that the agents are not forward looking, or the maintained assumption that the transmission of the social effect really follows the assumed temporal pattern. However, in this context, the inherent random nature of *exact* timing of conception together with the monthly data on childbirth allows us to relax the assumption of non-forward looking agents. It is very difficult both for the individual and the co-workers to exactly predict when conception takes place. This key notion together with a possibility to consider a detailed lag-structure provides us with a possibility to form testable *a priori* predictions about the dynamics of the temporal pattern that the social interaction effects will need to follow in order for us to seriously worry about the reflections problem.

Similarly, we are also able to form *a priori* predictions on in which way endogenous sorting of co-workers across workplaces are likely to affect the estimated dynamic pattern of the social effect. In additions to these predictions we also consider several falsification exercises where we test if the employee is affected by (i) the *contemporaneous* childbearing of *future* co-workers, (ii) the childbearing of the true co-workers' *siblings*, and finally (iii) the childbearing of the co-workers employed in the same *firm* but not in the same *workplace*. The individuals in these three "placebo peer groups" are likely to share many of the unmeasured attributes of the true co-workers and the focal worker, and are also likely to experience similar types of unobserved chocks as the focal worker. However, since they are not employed in the same workplace we do not expect them to influence the childbearing decisions of the focal worker unless the main association between fertility among the true co-workers and the focal worker is spurious.

Our main results indicate that co-workers' fertility indeed increases the propensity of childbearing among their fellow co-workers. The estimated effect of co-worker's childbearing on the probability of first birth follows an inverted u-shaped pattern with respect to time elapsed since the co-worker's child was born. The maximum effect (10% increase) is reached about 13-24 months after the birth of a co-worker's child, and then declines. This observed social influence controls for non-parametric monthly duration de-

pendence, time-effects, work place size, regional unemployment rate and several important individual and co-worker characteristics. The robust dynamic pattern of the main estimates across specifications and subgroups, and the results from the falsification tests jointly suggest that our results is most likely not generated by spurious correlations between co-workers' childbearing.

Further explorations allow us to draw some conclusions about how and why individuals are influenced by social effects in fertility. In line with a large sociological literature on friendship formation we find that individuals with similar characteristics are more strongly influence by their co-workers decisions.[1] Much more weight is put on the fertility decisions made by female co-workers and co-workers who are close in age. However, there are also interesting deviations from this same-type pattern. For example, while the parity of the childbearing co-worker does not seem to matter for first-time mothers, in contrast *only* the childbearing among co-workers' of the *same parity* matter for mothers with previous childbearing experiences. These strong *within*-parity peer effects and complete absence of *between*-parity peer effects among higher order births allows us to distinguish between alternative mechanisms and also gives further support to the validity of the identifying assumption that omitted variables is not driving the results. Omitted determinants of the individual's childbearing must be completely uncorrelated across women with different number of previous children within the workplace to spuriously generate the within-parity pattern just described.

Additionally our results indicate that social status may matters for social influences in the workplace since individuals are affected by co-workers who have the same or higher, but not lower, educational level. This result is consistent with evidence from laboratory experiments showing that individuals are influenced by those with higher, but not lower status. We also provide suggestive evidence that fertility spills-over between different social networks; childbearing decision seems to be transmitted from the childbearing of the sibling of a co-worker *via* the co-worker *to* the focal worker with an additional lag of approximately 18 months. While all models of social effects assume that this is the case, as far as we know this is the first time that such across-network spill-over effects has been confirmed empirically.

Our study contributes mainly to two strands of the current literature. First, most studies on social influence on individual fertility investigate either peer effects in developing countries (Behrman et al, 2002; Madhavan et al, 2003; Munshi and Myaux, 2006; Bloom et al, 2008;), or among teenagers (c.f.

[1] This pattern is also found by Manski and Mayshar (2003) and Munshi and Myaux (2006). See Currarini, Jackson and Pin (2008) for a recent study in the economics of friendship formation.

Hogan et al., 1985; Case and Katz, 1991). Moreover, most previous studies have focused on spill-over effects within neighborhoods. One of the exceptions is Kuziemko (2006) who study social influence on fertility between siblings in the US using the NLSY. The types of social mechanism underlying the peer effects in the workplace, among siblings, or teenagers in the same neighborhoods are likely to differ. Apart from being relatively scarce, evidence from the previous studies may additionally be difficult to generalize to individuals living in the developed world, or to the population at large.[2] Second, our work also complements a growing body of studies documenting co-workers' influence on employee's behavior. Using matched employee-employer data these recent studies show that social interactions affect individual productivity (Bandiera, Barankay and Rasul, 2007; Mas and Moretti, 2006), pension planning (Duflo and Saez, 2003), and work absence (Ichino and Maggi, 2000; Hesselius, Johansson and Nilsson, 2008). Except for Hesselius et al. all of these studies focus on social effects within a single firm.[3] The population wide micro data used in this study are likely to improve the possibility to generalize the results found to other settings.

The remainder of this paper is organized as follows. Section 2 briefly describes the mechanisms through which peers may matter for fertility decisions. Section 3 describes the data. Section 4 introduces the empirical model Section 5 presents the results and Section 6 concludes.

2 Conceptual framework

Why should individuals be influenced by their peers' childbearing? Two main arguments can be traced in earlier work.

First, peer effects can arise from social learning. This means that individuals deal with uncertainty by using their network as a source of information. A frequently suggested example of this key mechanism is the role of social influences on the classical fertility transition through the dissemination of information about the use of modern contraceptives. In our case information about contraceptives is likely of limited relevance, but social learning may still be important since individuals can draw inference from the experiences of co-workers to learn about the pros and cons of childbearing (Montgomery and Casterline, 1996). Besides collecting information about

[2] For instance it is conceivable that friends and family constitute a greater source of information in developing countries where other information channels are limited whereas in developed countries where women are active in the labor market to a larger extent we might expect other types of peer influences that are closer related to career and family decisions. Similarly, teenagers may be more (or less) susceptible to peer influences than the general population.

[3] Additionally Åberg (2003) study the risk of divorce as a function of co-workers divorce rates using linked employer-employee panel data for Sweden.

the childbearing experience itself learning in our context may also include learning about parental skills and institutional arrangements and requirements regarding parental leave (Kuziemko, 2006).

A second explanation for why individuals could be influenced by the fertility of peers is that they may derive higher/lower utility from joint childbearing. Such network externalities arise when the utility from a specific activity depend on the number of participants (Katz and Shapiro, 1985). Utility from joint leisure (Hamermesh, 2000), or in our case joint parental leave, is an example of why network externalities may be influential in this setting. Joint parental leave may be especially relevant for Sweden since parents can (and women typically do) stay home for a relatively long time with their newborn child before returning to work. However, individuals may also benefit from sharing their childbearing experiences with co-workers who are in the same situation or by the direct economies of scale that arise from coordinated childcare and the sharing of material expenses.

So far, the mechanisms described are rather general and could apply to nearly any peer group. However, since childbearing for most women imply a non-negligible time of absence from work there may also be other forms of network externalities that are unique for the workplace setting.[4] A priori it is not evident in which direction childbearing of a co-worker affects individual fertility. For instance employees may draw inferences from their co-workers' labor market related consequences of childbearing or co-workers childbearing can give rise to peer pressure that alter the individual costs of childbearing. Additionally if employees' compete for e.g. promotion opportunities within the workplace they may take strategic considerations into account when deciding about whether and when to have children. This argument can be motivated by a human capital model where time out of work leads to loss of human capital, as well as by a signaling model where there is a penalty for being the "first-mover" in the workplace. Hence if individuals take the relative timing of childbearing into account it is easy to imagine how one worker's fertility can be very contagious within the workplace. The period of parental leave that typically occurs directly after birth may be costly also for the establishment, particularly in small workplaces where labor substitution is generally more difficult. If workers internalize such costs then the individual's probability of own childbearing may be reduced.

Just as described, there are several potential explanations for why social interactions can influence fertility. However, a priori it is not evident which of these mechanisms that dominates. Determining the net effect of co-worker influence on individual fertility decisions is therefore an empirical question. Moreover, an important task of this paper is also to try to characterize the

[4] In Sweden mothers take 329 days of parental leave on average (which are fully financed through the social insurance system) during the first 4 years of a child's life (RFV 2004:14)

explanations for our result. We return to this matter in Section 4.3 where we explore the way that the fertility peer effects operate with respect to individual, co-worker and workplace characteristics. This provides us with several interesting patterns that together help us to draw conclusions about the mechanisms described above.

3 Data

The data used in this study comes from the IFAU database and it contains register information for the entire Swedish population aged 16-65. In addition to detailed individual background characteristics (LOUISE) the database holds information on the firm and workplace identifiers for all workplaces in which the individual is employed (RAMS). The data are further linked to the "multi-generation" register that holds information on the number of children born as well as the month of birth of each child. These data allow us to construct our measure of peer fertility and our binary outcomes variable; whether the focal worker gave birth to a child in a given month or not.

We focus on female workers between age 20 and 44 and restrict the analysis to workplaces with less than 50 employees.[5] The size restriction is important since it helps us to focus the analysis on a well-defined peer group where individuals in the network are likely to interact on a day-to-day basis. The rational for choosing to focus on women are first of all that their fertility cycle is well-defined, but also that both from the individual and the firms' perspective childbearing among women generates much more variation in terms of working hours and thereby costs due to the overwhelming share of parental leave time utilized by women. It should however be noted that this restriction does not apply to the co-workers. Thus the analysis looks at the impact of both male and female co-workers' fertility on female workers fertility.

To make the dataset manageable we select a random sample of 50 percent of the working individuals in 2004 and follow these eight years back in time (1997-2004). This means that women are defined to be under risk from 1997 through the end of 2004 as long as they are observed in a workplace, until

[5] The medical literature defines the childbearing age as years falling between 15 and 44 years old. However for simplicity we restrict our sample to individuals who were above 20 years old. Our choice is motivated by the fact that due to compulsory schooling in Sweden it is very rare that individuals start working and having children before this age. In 2004 only 3.4 percent of Swedish women had their first child before their 20th birthday and the average age at first birth were 29 and 31 for women and men respectively in 2004 (National Board of Health and Welfare).

the month when they give birth or until the month they turn 45.[6] To avoid including individuals who are only loosely connected to the workplace we retain workers with yearly labor income above the 10th percentile[7]. For simplicity, for workers employed in multiple workplaces, we assume that the workplace giving the primary source of earnings also is the main arena for social interaction.[8] Because individual fertility as well as the social influence of peers may be different for women having their first, second and third child we consider up to three fertility spells. For the first-time mothers we define the duration as the number of months from age 20 and up to their first birth and for the second- and third-time mothers it is defined as the number of months from their previous child birth up to the second birth or until they are censored. Individuals who did not give birth during the observation period are followed from when they became fertile (had their previous child) and as long as they are of fertile age between 1997 and 2004.

We combine this data with time varying information on the co-workers in the particular year, month and workplace and create indicators for whether any co-worker had a child in a specific month. We also add information on the age structure, gender composition, the share of co-workers with college education, workplace size, number of children of the co-workers and the sector of employment (public/private).

Table A1 reports summary statistics for the first, second and third order fertility spells respectively. We see that the typical women under risk of having her first child is 27.6 years of age and works in a workplace with 18 employees. Furthermore, the average probability of having a child in a specific month is 0.005 and it varies considerably in our sample (sd. 0.07). Column (2) shows that the mean probability of having a second child is more than twice as high as the monthly probability of having the first child (0.011) suggesting that those who already have a child are much more likely to give birth to a(nother) child. The probability of third birth is only 0.002. These patterns reflect rather short timing between first and second order births and the common practice in Sweden to stop reproducing after the birth of the second child.

Figure A2 and A3 in Appendix A shows the baseline hazard functions for the first two births. The first graph illustrates that the likelihood of childbearing for first-time parents in our sample peaks around age 30. This is somewhat higher than the average age (29 years) which is likely due to the fact that we have restricted our sample to women with a relatively strong connec-

[6] Since we require that the individuals should be working we include them in our sample only those years that we observe them in a workplace. This restriction implies that we will over sample individuals with stable positions on the labor market.

[7] The threshold is based on all employees at the labor market, both males and females.

[8] The vast majority of the individuals in our data is only employed at single workplace.

tion to the labor market. Figure A3 suggests that the probability of delivering the second child peaks after 28 months (2.3 years) and that most parents (70 percent) had their second child within 6 years from their first child.

4 Empirical specification

As discussed above we model the individual fertility decision as a function of co-workers past childbearing. An important feature of our setup is that we are able to provide evidence of what the lag structure of the transmission of the social effect looks like and we will see later in the estimation results that the estimated peer effect is consistent with that individuals indeed react to co-workers realized fertility and not with anticipation or joint planning of future childbearing.

To capture the dynamic pattern co-workers' fertility have on individual childbearing we estimate a conditional linear probability models which can be thought of as a linear approximation of a hazard model allowing for time-varying covariates, non-parametric duration dependence and time period effects (c.f. Allison, 1982).[9] Our baseline specification is

$$\begin{aligned} Y_{ijtc} = {} & \alpha_t + \beta_1 (\text{Any co-worker had a child within 12 months})_{\text{ijtc}} \\ & + \beta_2 (\text{Any co-worker had a child wihtin 13-24 months})_{\text{ijtc}} \\ & + \beta_3 (\text{Any co-worker had a child wihtin 25-36 months})_{\text{ijtc}} \\ & + X_{ijtc}\gamma + C_{ijtc}\delta + \eta_c + \varepsilon_{ijtc} \end{aligned} \tag{1}$$

where the dependent variable Y_{ijtc} indicates whether employee *i* in workplace *j* had a child in calendar month *c* and duration *t*. α_t is a measure of duration dependence and is non-parametrically specified using specific month of duration dummies. These dummies captures that the baseline hazard of childbearing varies over the fertility cycle. The variables "Any co-worker had a child within 12, 13-24 or 25-36 months" are indicators for whether a co-worker had a child within 12, 13-24 and finally 25-36 months prior to month c.[10] X_{ijtc} is a vector of individual background characteristics, C_{ijtc} is a

[9] We have also re-estimated the model using a Maximum Likelihood estimator. This did not alter any of the conclusions.

[10] The variable "Any colleague had a child within 12 months" counts from t-1 to t-12. Hence by construction the dummy takes on the value zero if the colleague delivered in the *same*

vector of co-worker and workplace background characteristics such as the previous number of children to all co-workers, age distribution, gender and educational attainment composition, dummies controlling for establishment size in 10 worker intervals, sector of employment etc. η_c are calendar period (year*month) effects which are included to control for general trends in fertility and finally ε_{ijtc} is the error term.

The main focus in our analysis is how co-worker childbearing affects the timing of first births since the variation in timing is largest for these births (see Figure A2), but we also report estimates for higher order births. Hence we estimate equation (1) for individual at risk of having their first, second and third child separately using OLS.[11] For first births the duration dependence is accounted for by "months since age 20" specific indicator variables up until the first birth (or until censoring) and for higher order births the number of months from the previous birth. Note that the combination of the duration dummies (months since age 20) and period (month*year) fixed effects also accounts for general cohort effects.[12]

The dynamic impact of fertility peer effect is captured by the parameters of interest β_1, β_2 and β_3 in equation (1). The estimates of these parameters measure the impact of co-workers' recent fertility on the likelihood of childbearing in a specific month. The maintained assumption for identification is that there are no unobservable determinants correlated with both the lagged timing of co-workers fertility and the focal worker's probability of giving birth to a child in month *c*. However, unobserved common shocks that change the probability of childbearing for all co-workers may generate precisely this type of spurious correlation between the fertility of the co-workers and the focal worker. While the period fixed effects accounts for general fluctuations in fertility (due to e.g. general policy shift in e.g. childcare allowances, or business cycle effects), there can still be workplace specific shocks changing the probability of childbearing for all workers in a particular workplace e.g. increased job flexibility, management changes, or an increased risk of mass lay-offs.

Moreover, the identifying assumption could also be violated if co-workers share other unobserved characteristics that affect the timing of childbearing. One example is similar taste for childbearing. Sorting of this kind is a valid concern since family friendliness of jobs is an important determinant of many woman's employment decisions (Herr and Wolfram, 2009). It is furthermore well established that friends and relatives are a well-utilized way to find jobs (c.f. Montgomery, 1991; Ioannides and Loury,

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month as the individual. This implies that we avoid the possibility that two colleagues having a child together show up as one of them responding to the other.

[11] During our observation period higher order birth is uncommon.

[12] For the second and third births regressions we also include year of birth fixed effects.

2004). Sorting for these or other reasons associated with childbearing could lead to very homogenous workplaces and result in correlations between co-workers' childbearing even if they are not directly influenced by each other's behavior.[13]

However the difficulty in foreseeing exactly when conception takes place helps us to form expectations about how the estimates of the parameters of interest in equation (1) should behave for us to worry that omitted variables biases our estimator. First, suppose that two individuals (co-workers) start trying to conceive at the same time (e.g. due to unobserved common shocks at the workplace level). Due to the partly random nature of timing of conception some will conceive sooner than others. However, calculations in Kuziemko (2006) suggest the probability that the two individuals will end up having children more than 6 months apart is only around 14%. This implies that if unobserved common shocks are causing a spurious correlation between co-workers' fertility decisions then we expect the strongest effect to show up during the first 12 months period after the birth of a co-worker's child and then decline (i.e. $\beta_1 > \beta_2 > \beta_3$). Furthermore if the estimates simply reflect endogenous sorting of workers then we expect the timing of co-workers childbearing to be irrelevant. To make this clear, suppose that workers conceive independently of each other (i.e. no social interactions) with some given probability each month. Then since there is an equal chance to have a co-worker who gave birth within 12, 13-24, and 25-36 months we would expect that $\beta_1 = \beta_2 = \beta_3$. We will see that our estimates do not match either of these predictions. Moreover the validity of our main estimates is also strengthened by a number of robustness checks and falsification test. These are described in detail in Section 5.2. But first we turn to our main results.

[13] A simple but unfeasible path to follow in order to try to control for workers sorting would be to add workplace fixed effects to equation (1). However, considering that we have a panel stretching only over 8 years and that we include lagged dependent variables for up to 36 months (which would be what the "co-worker had a child" dummies would be characterized as in a within-workplace analysis) the within-workplace estimates would, as is well known, be severely downward biased using an OLS estimator (Nickell, 1981). An alternative way to solve this problem would be to aggregate the data to the workplace level and then run regressions using a GMM estimator. But since an important focus of our analysis is to study in which way peer effects operate in relation to individual characteristics we feel reluctant to take this measure, and instead focus on other ways to make sure that the peer effects are not driven by endogenous sorting across workplaces (see discussion below).

5 Results

5.1 Main results

Column 1 of Table 1 shows the baseline estimates of the three β's from equation (1) capturing the impact of co-workers' childbearing on own fertility for first-birth women after controlling for duration dependence $\alpha(d)$ and period (year×month) fixed effects. The first, second and third row report the estimates of β_1, β_2 and β_3, i.e. the estimated impact of being exposed to a co-worker who had a child 1-12, 13-24 and 25-36 months ago respectively. The estimates of β_1 are small and not significantly different from zero, but still precisely estimated. In contrast the estimates of β_2 show a positive association between the focal workers childbearing and the past childbearing of her co-workers. Evaluated at the mean probability of childbearing this estimate suggests that individuals are on average 10.9 percent (0.00057/0.00523) more likely to have their first child 13-24 months after the birth of a co-worker's child. The inclusion of additional individual level controls for marital status, and college education (Column 2), as well as co-worker and workplace controls (Column (3)) does not alter the picture (see Table A2 in Appendix for all controls). The robustness of the estimates to the inclusion of these important covariates is reassuring since it suggests that bias due to omitted variables probably also are less of a concern.

Together the three estimates suggest that the co-workers' fertility decisions primarily increase fertility decisions with a lag of about one year after the birth of a co-worker's child. Hence, for first order births the influence of peers' childbearing is not driven by the immediate news that a co-worker is expecting, since the peer effect shows up first after the co-worker's baby is born. Even more importantly, the inverted u-shaped pattern of the effect with respect to the time elapsed since a co-workers' child was born speaks against the alternative hypothesis of unobserved common shocks or that co-workers plan their births so to be able to enjoy joint maternity leave. As discussed above if unobserved common shocks would induce individuals to start trying to conceive simultaneously we would expect to find the largest effect within the first 6 months. In our case we do not even find a significant effect within the first 12-month period after a birth of a co-workers' child. This clear and consistent pattern across specifications and (as we show below) sub-samples suggests that common unobserved shocks is not driving the estimates of the social effect. Similarly, as motivated above the pattern does not seem to be consistent with a situation where endogenous sorting of workers is causing a spurious correlation in the timing of pregnancy.

Table 1 Baseline estimates of co-worker's fertility on the probability of **first** birth

Specification:	(1)	(2)	(3)
Sample:	*First births*	*First births*	*First births*
Any co-worker had a child within:			
1-12 months	0.00002	0.00003	0.00005
	(0.00007)	(0.00007)	(0.00007)
13-24 months	0.00057***	0.00056***	0.00047***
	(0.00007)	(0.00007)	(0.00007)
25-36 months	0.00029***	0.00028***	0.00013*
	(0.00007)	(0.00007)	(0.00007)
Duration dummies	Yes	Yes	Yes
Year*Month dummies	Yes	Yes	Yes
Own char.	No	Yes	Yes
Workplace char.	No	No	Yes
Mean Y	0.00523	0.00523	0.00523
Observations	5,575,497	5,575,497	5,573,397

Notes: *,** and *** denote statistical significance at 10/5/1 percent level respectively. Standard errors robust for serial correlation at the workplace level are shown in parenthesis. The level of analysis is the individual-month. In addition to the fixed effects indicated by the table regression (3) controls for establishment size dummies in intervals of ten employees as well as the regional (county/year) unemployment rate where the workplace is located. The workplace characteristics includes number of children of co-workers, the share of co-workers in fertile age, the share of co-workers, close-in-age, share of female co-workers, share married co-workers, share of co-workers with college education.

To put our estimates in perspective consider first that for example Del Bono et al. (2008) find that women are about 10% less likely to have a child in the first couple of years after losing their job. Similarly Mörk et al. (2008) find that increasing childcare subsidies with the equivalent of USD 10,000 increased fertility for eligible Swedish couples by about 5-10% within 18 months. The magnitudes of the social effect are furthermore very similar to those found in recent studies also focusing on co-worker peer effects in general. For example, Mas and Moretti (2009); Falk and Ichino, (2008); Ichino and Maggi (2000) and Hesselius, Johansson and Nilsson (2009) all find co-worker peer effects which are in the vicinity of our estimates.

5.2 Robustness checks and falsification tests

5.2.1 Robustness checks

Our results are based on the maintained assumption that unobserved determinants of the timing of fertility are uncorrelated within the workplace. While we can never test this identifying assumption directly, the richness of the data allows us to design several indirect tests to assess the plausibility of this identifying assumption. But we start of by showing that the baseline results are robust to changes in the specification of our baseline model.

In column (1) of Table 2 we have re-specified the baseline model by replacing the three 12-month indicators of interest with six 6-months interval dummies. The estimates confirm that the baseline specification indeed does a god job in modeling the dynamic impact of co-workers' childbearing on timing of fertility. As before in the first 1-6 and 7-12 month intervals the behavioral impact of being exposed to co-workers' childbearing is small and not statistically significantly different from zero. However, in month 13-18 the effect shoots up and then declines slowly until it turns insignificant after 31-36 months. Again, the absence of effects within the first 6 months strengthens the conclusion that common shocks are not driving the estimated social effect.

Next we assess whether increasing the dose of exposure that is the number of co-worker children born within each period, has additional effects. We do this by interacting the baseline variables of interest with dummy variables indicating whether more than one co-worker had a child 1-12, 13-24 and 25-36 months ago. The estimates in column (2) provide a clear dose-response pattern of being exposed to childbearing of several co-workers; the interaction terms are positive and of significant size. Controlling for additional births does however leave the baseline estimates essentially unchanged suggesting that the main effect is not driven by exposure to many births. We therefore stick to the more parsimonious specification for the remainder of the analysis.

As common shocks do not seem to explain the observed peer effect pattern we now investigate whether sorting of workers based on e.g. child-friendliness of the workplace is a valid concern. It is important to remember that even in the baseline model we control for number of previous children in the workplace, which to a large degree should capture selective sorting. Still it is possible that workers who are planning to have a child systematically move to workplaces where childbearing is more frequent. As a first test of the validity of this concern we split the sample with respect to tenure and report the results separately in columns (3) and (4) of Table 2. Comparing the estimates we see that there are no major differences in the

Table 2 Robustness checks

Specification:	(1)	(2)	(3)	(4)
Sample:	Baseline	Baseline	< 5 years of tenure	> 5 years of tenure
1-6 months	0.00010			
	(0.00008)			
7-12 months	0.00012			
	(0.00008)			
13-18 months	0.00048***			
	(0.00008)			
19-24 months	0.00028***			
	(0.00008)			
25-30 months	0.00016**			
	(0.00008)			
31-36 months	0.00005			
	(0.00008)			
12 months		0.00002	-0.00001	0.00029
		(0.00008)	(0.00007)	(0.00021)
13-24 months		0.00043***	0.00044***	0.00059***
		(0.00008)	(0.00007)	(0.00021)
25-36 months		0.00013	0.00011	0.00040*
		(0.00008)	(0.00007)	(0.00021)
Multiple births:				
12 months × 1(>1 birth)		0.00024**		
		(0.00012)		
13-24 months × 1(>1 birth)		0.00030***		
		(0.0001)		
25-36 months × 1(>1 birth)		0.00001		
		(0.00011)		
Duration dummies	Yes	Yes	Yes	Yes
Year*Month dummies	Yes	Yes	Yes	Yes
Own characteristics	Yes	Yes	Yes	Yes
Workplace characteristics	Yes	Yes	Yes	Yes
Mean Y	0.00523	0.00523	0.0052	0.0057
Observations	5,573,397	5,573,397	4,559,220	1,014,177

Notes: *,** and *** denote statistical significance at 10/5/1 percent level respectively. Standard errors robust for serial correlation at the workplace level are shown in parenthesis. The level of analysis is the individual-month. In addition to the fixed effects indicated by the table regression (3) controls for establishment size dummies in intervals of ten employees as well as the regional (county/year) unemployment rate where the workplace is located. The workplace characteristics includes number of children of co-workers, the share of co-workers in fertile age, the share of co-workers, close-in-age, share of female co-workers, share married co-workers, share of co-workers with college education. The dummy 1(>1 birth) is equal to 1 if more than 1 co-worker gave birth with in the relevant time period.

impact of peers on women with more and less than five years of tenure. If anything the effect seems to be somewhat stronger for those with longer tenure, suggesting that sorting into establishments just before planning a pregnancy is not driving our results.

5.2.2 Falsification tests: Placebo co-workers

In Table 3 we continue to more rigorously assess the validity of the identifying assumptions. Here we re-estimate the specification in equation (1), but instead of focusing on the impact of the true co-workers, we now instead look at whether the childbearing behavior in three "placebo peer groups" also matter for individual childbearing. The placebo co-workers groups we consider are:

1) FIRM-LEVEL CO-WORKERS: These workers are employed in the same firm, region (21 regions), and 2-digit industry, but not in the same workplace as the focal worker. Since these are individuals that have sorted into the same firm-industry-region as the focal worker, they are first of all likely to experience the same type of shocks that could generate the type of spurious relationships between co-workers that we worry about. Secondly, for the same reasons they are likely to be highly similar to the focal worker even in terms of observed (this is shown in Table A3 and discussed below) and unobserved characteristics.

2) FUTURE CO-WORKERS: This placebo-peer group consists of the future co-worker's to the female workers in our sample that switch workplace during the observation period.[14] The idea is that the contemporaneous behavior of these future co-workers should not have any effect on the contemporaneous behavior of the focal worker unless i) they already are friends, or ii) they share unobserved characteristics that both induces them to sort into the same workplace at a later stage and that also affect the timing of childbearing.

3) SIBLINGS OF ACTUAL CO-WORKERS: This placebo-peer group is likely to share many of the co-workers observed and unobserved characteristics. They have experienced similar upbringing and might therefore have formed similar preferences for the timing of childbearing. If these unobserved preferences or characteristics are correlated between co-workers we expect that the childbearing of the co-workers siblings should not affect the focal worker unless i) they already know each other or ii) they share unobserved characteristics which affect the timing of childbearing. However, Kuziemko (2006) find evidence of fertility peer effect among siblings and hence if childbearing really is contagious then it is conceivable that the childbearing of siblings could also spill-over to the focal worker via the fertility decisions of the actual co-worker. In this case we would expect the effect to show up after the additional lag it takes for first the co-worker and

[14] To make sure that we capture actual job switchers we restrict the sample to women who switch jobs only once during the observation period and we require that the individual is observed for at least 2 years before and after the change in jobs.

then the focal worker to react. Alternatively, if the sibling, co-worker and the focal worker share unobserved characteristics, or if the sibling and the focal worker influence each other directly, we would expect to find a spurious placebo co-worker effect that follows the same pattern as the baseline effect of true co-workers.

Table A3 presents descriptive statistics for the main samples and the three placebo peer groups. We conclude from this table that observed characteristics of the true co-workers are all highly similar to the placebo peer groups. There are essentially two exceptions; the average number of co-workers in the average firm is naturally much higher than in the average workplace, and since the labor market is segregated with respect to gender the average share of females among the true co-workers is higher than that among the co-workers' sibling since this placebo group to a higher extent consist of brothers. In the empirical specification we address these differences by controlling for co-worker' sibling characteristics and we also include 9 dummies for firm size where relevant. Since the three placebo-peer groups are fairly balanced on observed characteristics it is reasonable to believe that they are similar in terms of unobserved characteristics as well. However, we do not expect to find the same fertility peer effects for these placebo workers as for the true co-workers unless the childbearing of the co-workers simply proxy for some unobserved determinant that the focal worker, the co-workers and the placebo co-workers have in common.

Table 3 shows the estimates from these falsification tests. I.e. column (2) report the estimates for the first placebo peer group, "the firm co-workers", column (4) presents the results for second placebo peer group "the future co-workers", and column (5) shows the estimates for the third placebo peer group "co-workers' siblings". In addition since the placebo-tests restrict the samples to women who work in private firms with more than one workplace in column (1) and to those who switch jobs in column (3) for comparison we also report the impact of the true co-workers childbearing in each of these samples. While the estimates for these true co-workers are highly similar to the baseline estimates in Table 1, neither one of the three placebo co-worker regressions provides results that are even close to these results.[15]

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[15] One concern is that since the number of co-workers in the same firm can be much larger than the number of co-workers within the same workplace we have also estimated the "same firm different workplace" regression using only firm that have less than 50 employees in total. These estimates were very similar to the full placebo group sample estimates.

Table 3 Falsification exercise

Specification:	(1)	(2)	(3)	(4)	(5)
Sample:	Private firms with multiple workplaces	Private firms with multiple workplaces	Job switchers	Job switchers	All
	True: *Same firm, same work-place*	***Placebo:*** *Same firm, different work-place*	***True:*** *Contempora-neous co-workers*	***Placebo:*** *Future co-workers*	***Placebo:*** *The Co-workers siblings*
Any co-worker had a child within:					
12 months	0.00012	0.00015	0.00026	-0.00003	0.00005
	(0.00016)	(0.00025)	0.00021	(0.00020)	(0.00007)
13-24 months	0.00067***	-0.00015	0.00072***	0.00015	0.00011
	(0.00015)	(0.00025)	0.00021	(0.00020)	(0.00007)
25-36 months	0.00019	0.00010	0.00032	0.00000	0.00031***
	(0.00016)	(0.00025)	(0.00022)	(0.00020)	(0.00007)
Duration dummies	Yes	Yes	Yes	Yes	Yes
Year*Month dummies	Yes	Yes	Yes	Yes	Yes
Own characteristics	Yes	Yes	Yes	Yes	Yes
True Co-worker characteristics	Yes	Yes	Yes	Yes	Yes
Placebo co-worker characteristics	No	Yes	No	Yes	Yes
Mean Y	0.00503	0.00503	0.0058	0.0058	0.00523
Observations	1,066,052	1,066,052	729,767	729,767	5,403,084

Notes: See table 2. The specification in column (2) additionally control for Firm size dummies in nine intervals (2-9, 10-19, 20-29, 30-39, 40-49, 50-99, 100-199, 200-499, >499 employees)

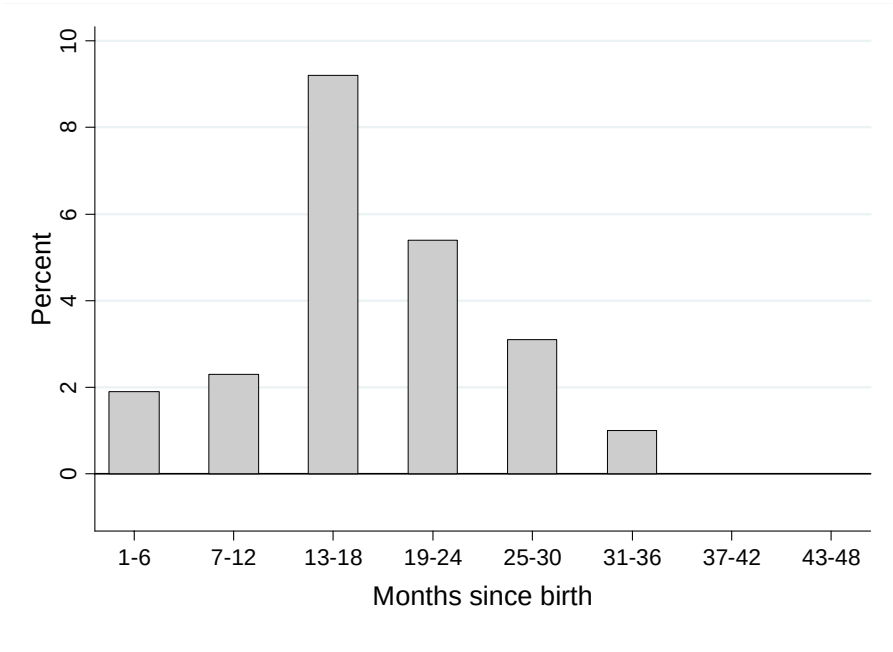

Figure 1 Peer effects of *true* co-worker childbearing

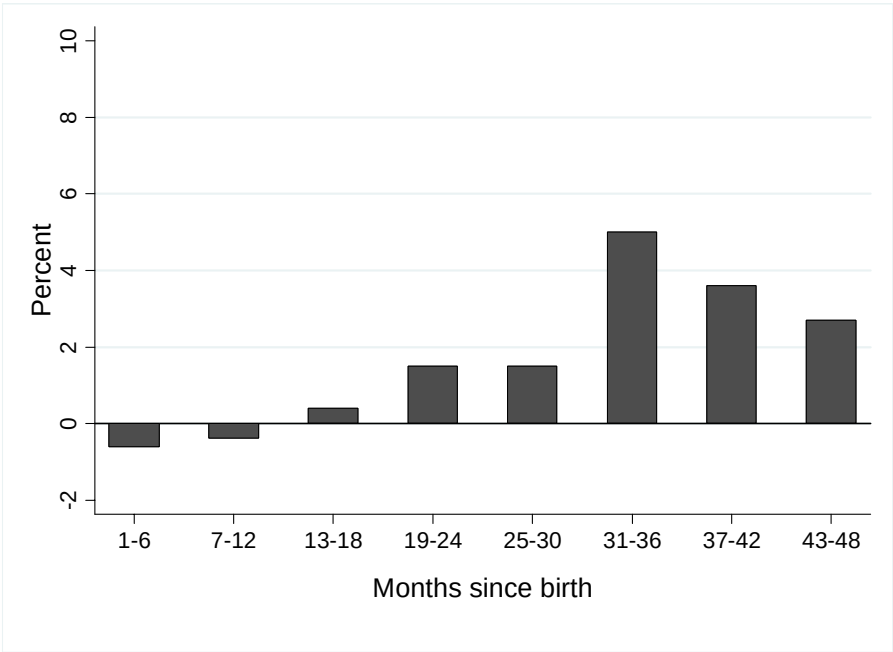

Figure 2 Peer effects of the co-workers' siblings' childbearing

Interestingly, the only estimate that is significantly different from zero in any of the placebo peer group regressions is the 25-36 month lagged effect in the co-workers' sibling sample. To further assess this intriguing pattern we estimated a model where we allowed co-workers' siblings to affect childbearing decisions of the focal worker in 6-months intervals for up to 52 months. The results are presented and compared to the baseline 6-month interval estimates (from Table 2) in figure 1 and 2. Interestingly the parameter estimates are small and insignificant for the first 30 months after a birth by a co-worker's siblings only to show up after a lag of 31-36 months and then fade out slowly. This suggests that the fertility decision spills over from the sibling of the co-worker via the co-worker to the focal worker supporting the notion that fertility decisions truly are contagious and that they may also spill over across different networks.

To summarize, the results of the robustness checks in Table 2 and falsifications tests in Table 3 strongly support a causal interpretation of the baseline estimates. We now proceed by further investigation of the underlying mechanisms that can explain the fertility peer effects.

5.3 Heterogeneity: individual and co-worker characteristics

Next we examine if the influence of the peers varies with respect the focal workers own, her co-workers and the workplace characteristics. In conjunction with the results we also discuss alternative explanations for the heterogeneous effects.

5.3.1 Individual characteristics: fertility cycle, education, and civil status

We begin in Table 4 by looking at whether the peer effect differs depending on where the individual is in the fertility cycle. We divide the fertility cycle into an early (age 20-27), primary (age 28-36) and late (age 37-44) stage.[16] Columns (1) - (3) in Table 4 show that women are influenced in all stages of the fertility cycle and in fact most strongly towards the later stages.[17] This pattern could be due to the formation of tighter bonds between older co-workers because of longer joint tenure at the workplace. Alternatively, due to the concaveness of the life cycle earnings profile the cost of career interruptions should be relatively lower towards the end of the fertility cycle.

[16] Since we focus on women without any previous children the number of months under risk corresponds to their age.

[17] Evaluated at the mean, the estimates correspond to an increase in own childbearing of 7.3 percent in the early stage, 9.4 percent in the primary stage and 14.5 percent in the late stage of the fertility cycle.

Thus the lower costs of reacting to peer influences is another explanation for why women respond more strongly to their peers in the later stages of the fertility cycle.

Since we do not have data on completed fertility for all workers in our sample, the distinction between pure timing effects and effects on completed family size is difficult. The fact that peers childbearing also influence women without previous children who are above their primary childbearing age does however indicate that social interactions may not only affect the timing of childbearing but also the decision of whether to have a child or not. We will return to this matter below when we investigate the influence of peers on higher order births.

Next we look at whether the response to peers childbearing choices differs between married and unmarried women. This effect is ex ante ambiguous since on the one hand unmarried women may on average have less stable relationships making them unable to react as fast as married women can. On the other hand, married women may be less prone to be affected by outside influences if they already have made plans about the timing of childbearing. However, it is important to remember that more than 2/3 of the first time mothers are unmarried at the birth of the first child in Sweden, suggesting that marriage status perhaps is not such an important factor with respect to peer influences on childbearing. In columns (4) and (5) we see that although the point estimates are larger for the married women, when evaluated with respect to the mean the effect is actually larger among unmarried (10.6 %) than among married (7.11%). However, when looking at the cumulated effect for the entire 13-36 month period, the effect is largest for the married co-workers. All in all there seems to be no remarkable difference in the reaction to peers based on own marriage.

Table 4 Differential peer effects and individual characteristics: fertility cycle, education and earnings

Dependent variable: Individual had it's first child in month t

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
First births	Early (age 20-27)	Primary (age 28-36)	Late (age 37-44)	Married	Not Mar-ried	College	No College
Any co-worker had a child within:							
12 months	-0.00004	-0.00009	-0.00013	-0.00030	0.00005	0.00010	0.00006
	(0.00008)	(0.00025)	(0.00020)	(0.00039)	(0.00007)	(0.00012)	(0.00009)
13-24 months	0.0003***	0.0009***	0.00043**	0.0012***	0.0004***	0.0004***	0.0005***
	(0.00008)	(0.00019)	(0.00020)	(0.00038)	(0.00007)	(0.00012)	(0.00009)
24-36 months	0.00007	0.00032*	0.00033	0.00063*	0.00010	0.00029**	0.00007
	(0.00008)	(0.00019)	(0.00020)	(0.00038)	(0.00007)	(0.00012)	(0.00009)
Duration dummies	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
*Year*Month dummies*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
Own characteristics	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
Workplace characteristics	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
Mean Y	0.00409	0.00921	0.00297	0.01645	0.00386	0.00562	0.00498
Observations	3,838,904	1,324,836	409,657	605,112	4,967,841	2,140,535	3,432,418

Notes: see Table 2

Finally columns (6) and (7) present the peer effect for women with college and no college respectively. The estimates suggest that the peer influence for women with college education (column 6) is stronger than for those without college education (column 7), a results that squares poorly with that the peer influence should be due to economies of scale associated with coordinated childbearing.[18]

5.3.2 Heterogeneous peer effects with respect to the type of co-worker

In the economic as well as in the sociological literature it is well established that people who are similar tend to associate more (Bandiera, Barankay and Rasul, 2007; Currarini, Jackson and Pin, 2008; McPherson, Smith-Lovin and Cook, 2001). However, so far in the baseline model (equation 1) we have assumed that the fertility peer effect is homogenous irrespective of whom of the co-workers that is having a child. In this section we allow the response to co-workers childbearing to vary depending on how similar the childbearing co-worker is to the focal worker. This is implemented by estimating

$$\begin{aligned} Y_{ijct} = \Omega &+ \lambda_1 (\text{Any co-worker had a child within 12 months} \times \text{TYPE})_{ijct} \\ &+ \lambda_2 (\text{Any co-worker had a child 13-24 months ago} \times \text{TYPE})_{ijct} \\ &+ \lambda_3 (\text{Any co-worker had a child 25-36 months ago} \times \text{TYPE})_{ijct} \end{aligned} \quad (2)$$

where Ω corresponds to the exact right hand side of equation (1) and TYPE is an indicator variable for if any of the co-worker who had a child in the previous periods were either of the same sex, close-in-age, or had the same educational attainment as the focal worker. Since we only focus on the impact on female workers, the same sex indicator measures the impact of female co-workers. The same education indicator is based on the college/no college education definition and is hence equal to 1 if the co-worker has the same level of educational attainment. Finally, two co-workers are defined as being close in age if they are born less than four years apart.

In the top panel of Table 5 the estimates of the three β's are presented (which as before corresponds to the impact of any co-workers' childbearing), and in the bottom panel the estimates of the three λ's (which reflects the

[18] For instance; parents can derive economic benefits due to the economies of scale that arise from coordinated childcare or from the sharing of necessary baby supplies (trolleys, clothes etc.). With the generous benefits attached to having children in Sweden we believe this effect to be of second order importance. First, Sweden has a generous parental leave benefit system which allows for benefits for 480 days (16 months). The benefit constitutes 80% up to a ceiling the first 390 days and another 90 days at flat rate. Furthermore childcare is heavily subsidized in Sweden and enrollment is very high. In 2004, 90 percent of all children 3-6 attended child care (National Board of Education).

additional effect the childbearing of similar co-workers have). The total effect of a same-type co-worker is obtained by adding the main effect and the interaction effect. Starting out by looking at the differential impact of male and female co-workers in column (2) we find that the entire baseline peer effect seems to be driven by the influence of female co-workers (i.e. same sex).[19] More frequent interaction among female co-workers and/or gender-specific learning are both possible explanations for this result. In our model we always control for the fraction of same type co-workers in the workplace so the stronger influence that female co-workers exhibit cannot be explained by tighter friendships with other women due to workplace gender segregation but rather that they associate more given the fraction of female co-workers in the establishment.[20] The close-in-age specification estimates reported in column (3) suggest that the influence of co-workers who are close-in-age are substantially stronger than from other co-workers; individual fertility increases with 10 percent within the first 12 months and 18 percent after 13-25 months.

Finally we look at the impact of co-workers with the same versus different educational level as the focal worker. Interestingly these estimates suggest that whereas highly educated women are affected only by other highly educated peers (column (4)), low educated women are influenced by all co-workers regardless of educational level (remember that the total effect of same type co-workers in column (5) is the sum of the main effect and the interaction effect). If individuals interact mainly with co-workers who have the same educational level then we expect both high and low educated women to be primarily influenced by their same type peers. However, the anomalies in the same-type pattern that we find; no peer influence of low educated co-workers on high educated workers but a significant impact of high educated co-workers on low educated workers rather speak to a literature suggesting that besides similarity, individual interactions may also be determined by social status (c.f. Akerlof and Kranton, 2000). Moreover this result is in line with laboratory experiments suggesting that people are influenced by the behavior of individuals with higher, but not lower, social ranking than themselves (Kumru and Vesterlund, 2008).[21]

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[19] Interestingly this is precisely the same pattern that Kuziemko (2006) finds when studying peer effects among siblings. In her case it was only sisters' and not brothers' childbearing that influenced the siblings' childbearing decisions.

[20] The feature of networks that similar individuals tend to associate more is often referred to as *"homophily"* and it can occur for various reasons. The literature often distinguish between *baseline homophily* which arises due to contact availability and *inbreeding bias* where same-type friendships form at rates that exceed these relative fractions in the population (see McPherson et al (2001) for an overview on homophily).

[21] Kumru and Vesterlund (2008) show that individuals are more likely to mimic the behavior of high-status individual than low- status individuals in charitable contributions.

Table 5 Heterogeneous peer effects: Similarity

First births	(1)	(2)	(3)	(4)	(5)
Co-worker type:	All co-workers: Baseline	Same sex Co-workers:	Close in age Co-worker:	Same education Co-workers: College	Same education Co-workers: No College
Any co-worker had a child within:					
12 months	0.00004	0.00007	-0.0003***	0.00011	-0.00035**
	(0.00007)	(0.00010)	(0.00008)	(0.00015)	(0.00016)
13-24 months	0.00048***	0.00016	0.00009	0.00011	0.00063***
	(0.00007)	(0.00011)	(0.00008)	(0.00014)	(0.00017)
24-36 months	0.00018**	0.00000	-0.00014*	0.00005	-0.00021
	(0.00007)	(0.00011)	(0.00008)	(0.00014)	(0.00017)
This *type* of co-worker had a child within:					
12 months		-0.00000	0.00088***	-0.00005	0.00052***
		(0.00012)	(0.00012)	(0.00019)	(0.00017)
13-24 months		0.00047***	0.00107***	0.00058***	-0.00011
		(0.00012)	(0.00012)	(0.00018)	(0.00018)
24-36 months		0.00026**	0.00096***	0.00042**	0.00034**
		(0.00012)	(0.00012)	(0.00018)	(0.00017)
Duration dummies	Yes	Yes	Yes	Yes	Yes
Year*Month dummies	Yes	Yes	Yes	Yes	Yes
Own char.	Yes	Yes	Yes	Yes	Yes
Workplace char.	Yes	Yes	Yes	Yes	Yes
Mean Y	0.00523	0.00523	0.00523	0.00562	0.00498
Observations	5,575,497	5,575,497	5,575,497	2,140,535	3,432,418

Notes: see previous Tables and text for the definition of variables.

5.3.3 Heterogeneous peer effects with respect to birth order of the child

The baseline results in Table 1 reported the peer effect for women at risk of having their first child. In this section we examine whether co-workers also influence the timing of the second and third child. Since these women already had previous children they should have little use of further information from peers about the nature of childbearing. However, looking at second time mothers in column (2) of Table 6 we see that the peer influence is almost as strong as for first time mothers. Moreover, for this group of women peers childbearing increases the propensity of giving birth even within 12 months after they had a child. This is not surprising since couples who already have had a previous child are likely to be able to react sooner than

couples who are about to have their first child.[22] This difference between first and second time mothers is furthermore consistent with that learning about the nature of childbearing itself is more important for first time mothers since it may take some time before the most relevant experiences of the co-worker is actually realized.[23]

Even for women with two previous children we find some weak evidence (a 5% increase within 13-24 months) of a peer effect as suggested by column (3). Besides the astounding homogeneity of the timing of the effect across the birth orders, the fact that also third-order births may be influenced again indicates that peers may potentially also shift the preferences for optimal family size. Women having their third child are reacting somewhat slower to peer influences than second order births which consistent with that Swedish couples generally decide to stop trying to have more children after the second child is born. Hence, the time it takes women to re-negotiate the views of the optimal family size with partners may perhaps delay and mute any response to the influences of peers. This notion is also supported by the fact that the estimate for the 25-36 month interval for the third order births is only slightly lower than the 13-24 months estimates, while the differences between the same two coefficients for the first and second order births are considerably larger.

Related to the above finding that similar co-workers do exhibit stronger peer influence on each others childbearing decision, in column (4)-(6) of Table 6 we now look at whether individuals are differentially affected by co-workers who have the same number of previous children. Intuitively, this could be the case if there is some type of information that is unrelated to the childbearing experience in general but specific to the birth order of the child. For instance, mothers with one child might look at the behavior of their two-children peers to draw inferences of about the labor market consequences of having a second child, the organization of work and family with two kids, or the optimal timing of the second child. Another plausible alter-

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[22] We have also estimated this model using 6-months intervals. The estimates from this more flexible specification show that the entire within 12 month effect is driven by women giving birth between 7 and 12 after the birth of a co-worker's child [est.: 0.00068 (std.err.: 0.00020)]. These estimates are retain for expositional purposes but are available upon request from the authors.

[23] Additional the quicker response among women about to have their second child could go through the information channel by diffusion of information about the peculiarities of the of the Swedish "speed premium" policy. This policy provides strong economic incentives for parents to space their children closely together. See Hoem, 1990 for more details on the Swedish speed premium. At the same time it is important to remember that the speed premium should have no impact on the estimated effect since we always compare mothers with the same distance from the previous child an hence any general speed premium effect should be controlled for by the duration dummies.

native is that co-workers who already have a child have formed tighter bonds with the co-workers who already have a child.

The estimates in columns (4)-(6) are estimated using the model in equation (2), where TYPE now is equal to 1 if the co-worker who just gave birth previously had the same number of children, hence we now allow for parity-specific peer effects. Starting with the first-time mothers in Column (4) we find that these women are influenced by their co-workers' childbearing irrespectively of the birth order of the co-worker's child. In contrast, for second and third time mothers (Columns (5) and (6)), we find clear evidence of within-parity peer effects while cross-parity effects are completely absent. That is, the childbearing co-workers are only influencing behavior of the focal worker if they are having a child of the same birth order.

This clear pattern is interesting for at least two reasons. First, it suggests that the mechanisms underlying the peer effect seems to differ depending on own previous childbearing experience. Because women without children are equally influenced by the childbearing of any co-worker perhaps the main peer mechanism in this case is social learning about the childbearing experience itself. Arguably this type of information could be inferred from any co-worker irrespectively of their number of previous children. However, for higher order births individuals only use their same parity-peers to attain information about the specific experience of having a second or a third child, or the optimal timing of child spacing. Lower order childbearing among co-workers is however disregarded and do not change the optimal timing of their next child, potentially because births among lower-parity women do not generate any information or experience which is of any use for the focal worker.

Second, the within-parity peer effects for the higher order births provide additional evidence that our identifying assumptions are valid. To see this clearly; if omitted variables were to generate the effect in column (5) and (6) they must be uncorrelated across parity groups in order to explain the pattern that we find. Standard omitted variables such as common unobserved shocks at the workplace level are unlikely to satisfy this condition: the shock would have to be due to something altering the childbearing incentives of the women of the same parity only and no one else. Thus we interpret these results as an additional and important piece of evidence that individuals responding to their co-workers' fertility rather than to a common unobserved shock.

Table 6 Heterogeneous peer effects: Birth order

	(1)	(2)	(3)	(4)	(5)	(6)
SAMPLE:	1st birth	2nd birth	3rd birth	1st birth	2nd birth	3rd birth
Any co-worker had a child within:						
12 months	0.00004	0.00044**	-0.00005	0.00001	0.00020	-0.00007
	(0.0001)	(0.00017)	(0.00005)	(0.00012)	(0.00019)	(0.00006)
13-24 months	0.0005***	0.0008***	0.00010*	0.0005***	0.00023	0.00009
	(0.0001)	(0.00017)	(0.00005)	(0.00011)	(0.00019)	(0.00005)
24-36 months	0.00018**	0.00033**	0.00008	0.00024**	-0.00009	0.00007
	(0.0001)	(0.00017)	(0.00005)	(0.00011)	(0.00019)	(0.00005)
*Any co-worker had a child of the **same birth order** within:*						
12 months				0.00003	0.00029	0.00022
				(0.00013)	(0.00028)	(0.00028)
13-24 months				0.00000	0.0015***	0.00040*
				(0.00012)	(0.00025)	(0.00022)
24-36 months				-0.00009	0.0010***	0.00040**
				(0.00011)	(0.00024)	(0.00019)
Dur. dummies	Yes	Yes	Yes	Yes	Yes	Yes
Year dummies	Yes	Yes	Yes	Yes	Yes	Yes
Own char.	Yes	Yes	Yes	Yes	Yes	Yes
Workpl. char.	Yes	Yes	Yes	Yes	Yes	Yes
Mean Y	0.00523	0.01105	0.00202	0.00523	0.01105	0.00202
# Obs	5,573,397	2,015,434	3,729,137	5,573,397	2,015,434	3,729,137

Notes: *,** and *** denote statistical significance at 10/5/1 percent level respectively. Standard errors robust for serial correlation at the establishment level are shown in parenthesis. The level of analysis is the individual-month. In addition to the fixed effects indicated by the table all regressions control for establishment size dummies in intervals of ten employees. Each regression is estimated on a sample of individuals at risk of having their first, second and third child. Hence in the second sample we condition on having a first child. The higher mean probability in column (2) reflects that a high share of one child parents choose to have a second child and for the same reason the number of person/month observations is lower for the second order birth than for the other births.

5.3.4 Heterogeneity: workplace characteristics

Next we look at whether the estimated peer effect varies with respect to workplace characteristics, starting with workplace size. The marginal peer effect may differ by workplace size either because the true fertility peer effect differs between workplaces with different size, or because co-workers interact differently within different sized workplaces. Note, however, that it is a priori not possible to determine the direction of the bias if for example the true peer group consists of a smaller subset of workers within each workplace (c.f. Manski, 1993). With these caveats in mind we divided the sample into 3 groups based on number of employees and estimated one separate peer regression for each sample. These estimates are reported in Table 7. As seen in column (1)-(3) the largest estimated peer effect 13-24 months since the birth to a co-workers child is found in the smallest workplaces (2-10 employees) (15%) and in the largest workplaces considered (30-49 employees) (9%). The smallest peer effect is found in medium sized workplaces with 10-29 employees (7%). This u-shaped marginal peer effect pattern with respect to workplace size is further reinforced when dividing the sample into smaller size brackets (2-9, 10-19, 20-29, 30-39, 40-49); the marginal peer effect remains strongest in the smallest and largest workplaces and lowest for the medium sized workplaces with 20-29 employees (not reported).

One potential explanation consistent with this intriguing workplace size pattern is that while the precision of our network measure decreases with workplace size, the frequency of exposure to co-worker childbearing increases with workplace size. Hence, when the network size becomes larger than a certain threshold the cumulative effect of multiple births among co-workers dominates the decreasing network precision effect. This is further consistent with the treatment-response pattern we found in Table 2; more exposure implies stronger peer effects. To explore whether more exposure can explain the peer effect in the largest workplaces we re-estimated the model including indicator for multiple births among co-workers 1-12, 13-24 and 25-36 months ago. As suggested by Table A4 in Appendix, including dummies for more than one birth does not change the u-shaped pattern of the peer effect with respect to the workplace size. Thus it seems as if exposure to multiple births cannot explain why the peer effect is stronger in larger workplaces than in middle-sized establishments.

Alternatively it could be that, as observed in other studies, when network size increases the possibility to form friendships with individuals of the same type (e.g. gender, age, parity) and hence the positive within-type specific fertility peer effects could dominate any general adverse trend in the quality of our network measure. In planned future work we intend to further empiri-

cally test this and other potential explanations that are most likely underlying the observed pattern.

Table 7 finally investigates if the marginal peer effect differs with respect to workplace sector. If employees take into account the costs of maternity leave imposed upon the establishment when deciding about own childbearing we would potentially see a weaker peer influence in the for-profit sector. However, as columns (4) and (5) shows there are significant spill-over effects of co-workers' childbearing both in public and private establishments. The effects are not significantly different from each other.[24]

Table 7 Heterogeneous peer effects: Workplace characteristics

First births	(1)	(2)	(3)	(4)	(5)
Any Co-worker had a child within:	2-9 Employees	10-29 Employees	30-49 Employees	Public sector	Private sector
-12 months	-0.0002	0.0001	0.0000	0.0001	0.0000
	(0.0002)	(0.0001)	(0.0001)	(0.0001)	(0.0001)
13-24 months	0.0008***	0.0004***	0.0005***	0.0005***	0.0005***
	(0.0002)	(0.0001)	(0.0002)	(0.0001)	(0.0001)
24-36 months	-0.0001	0.0001	0.0002	0.0002	0.0001
	(0.0002)	(0.0001)	(0.0002)	(0.0001)	(0.0001)
Duration dummies	Yes	Yes	Yes	Yes	Yes
Year* Month dummies	Yes	Yes	Yes	Yes	Yes
Own char.	Yes	Yes	Yes	Yes	Yes
Workplace char.	Yes	Yes	Yes	Yes	Yes
Mean Y	0.00512	0.00524	0.00535	0.00602	0.00494
# Obs.	1,760,442	2,664,386	1,148,125	1,523,316	3,733,621

Notes: see Table 1

[24] It should be noted that the direct costs for employers associated with maternity leave in Sweden is zero and thus the only costs upon the establishment is indirect costs related to e.g. temporary human capital loss and labor substitution. With that in mind, individuals' internalizing the establishment's costs seems also inconsistent with the findings of strongest peer effects in the smallest workplaces where potential costs can be expected to be highest due to lower flexibility and opportunity for labor substitution between employees.

6 Conclusions

This paper explores the influences that co-workers have on their female fellow workers' fertility decisions. Our results suggest that there are non-trivial peer effects on the choice of timing of childbearing. The main analysis shows that the effect of being exposed to co-workers' childbearing increases the probability of own childbearing during the following 13-24 months to the same extent as lowering childcare costs with USD 10,000 (Mörk et al., 2008), and as much as the decreases in childbearing after job displacement (del Bono et al., 2008).

The average effect however masks substantial differences in the size of the peer effect depending on the individual characteristics and in particular how these match the characteristics of the co-workers. Childbearing among co-workers who are of similar age, female co-workers and co-workers of the same parity are more influential. In contrast, childbearing of male co-workers and exposure to childbearing of co-workers with lower order births than the focal worker has no influence at all on the timing of childbearing. Apart from affecting the timing of childbearing, social influences may be able to affect completed fertility; both third-order births and women in the late stage of the fertility cycle (age 37-44) without previous children are affected. We also provide suggestive evidence that fertility peer effect spills-over across social multiple networks; from siblings of a co-worker via the co-worker to the focal worker with a lag of about 31-36 months.

We also discuss which types of mechanisms that is most consistent with the observed peer effects. One explanation is that since the timing of childbearing has large effects on future earnings in particular for women (see e.g. Bertrand, Goldin and Katz, 2009), co-workers use the experience that other women in the same situation are confronted with after childbirth. Given our results, this explanation seem more likely than for example joint planning, economies of scale or learning about the nature of the pregnancy and childbearing experience itself. For example, consistent with a model of social learning we find that the peer effect for first time mothers is similar irrespective of the birth order of the co-worker's child, while for higher order births within-parity effects are strong but cross-parity effects are entirely absent. These effects together with the results that the childbearing of more similar co-workers in other aspects are more important suggest that observational learning (for example about optimal timing of childbearing) is an important mechanism. Additionally we show that individuals are only influenced by co-workers who have the same or higher educational level. The anomalies in this same-type pattern are interpreted in line with theories suggesting that social status may be important in explaining individual behavior.

The results presented in this paper have several implications for both researchers and policy makers. There still exists a considerable controversy among demographers on whether public policies have the potential to affect

fertility rates at all (see e.g. Hoem, 2008). Since our findings provide evidence of significant spill-over effects within networks and potentially also across different social networks, when evaluating the impact of policies aiming to affect fertility it is important to take into account that the control group could also be indirectly affected. If missing to account for such spill-over effects the impact of the policy may be underestimated.

One should on the other hand also bear in mind that the net effect of a given policy is a combination of social interaction effects and the direct impact of the policy. Therefore the strong heterogeneity of the social effects found in this study suggest that caution is warranted before assuming that the same policy when applied in another context will have the same impact on fertility. In a different context the net effect of the same policy may yield considerably different effect depending on network composition and the number/strength of the social ties within the targeted treatment group.

Additionally the peer effects we find in such an important decision as the timing of childbearing clearly point at the importance of social influences also for other types of career related decisions. Many observers have e.g. claimed that (the lack of) female role models in leading positions are important for women's own propensity to consider similar career paths. Our findings suggesting that female employees are influenced by the behavior of their female, but not by their male co-workers lend some indirect support for these claims. If career and family choices have the tendency to spread within networks (for instance through observational learning) then such peer effects may be very important for understanding observed differences between men's and women's individual career choices and the organization of work and family. To uncover to what extent gender specific peer effects at work lie behind other trends in labor supply related decisions; the choices to e.g. opt out of the labor force (see Bertrand et al., 2009), change to part-time work or to take up managerial positions, are all important and interesting questions for future research.

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Appendix A

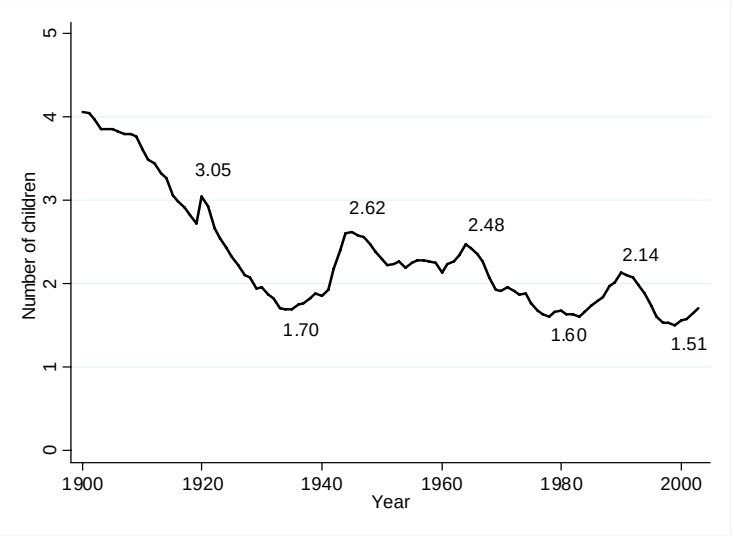

Figure A1 Total fertility rate, 1990-2003, *Source*: Socialstyrelsen (2005)

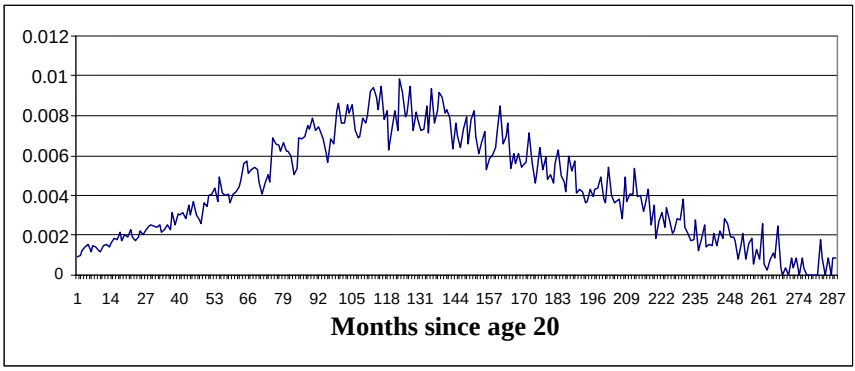

Figure A2 Baseline hazard of first order births

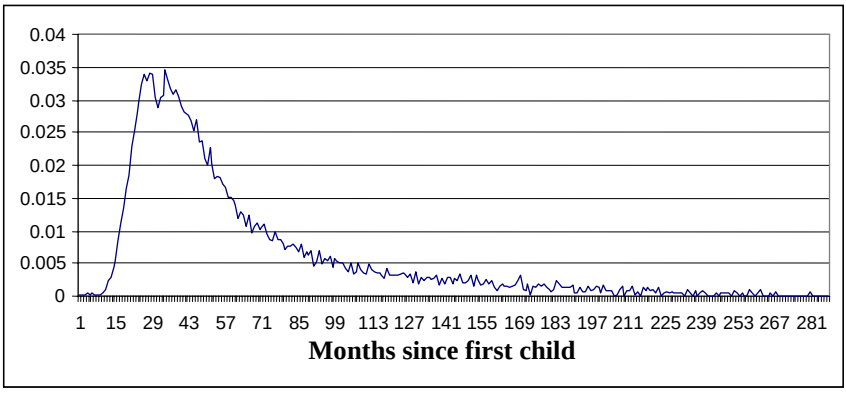

Figure A3 Baseline hazard of second order births

Table A1 Descriptive statistics

	First birth		Second birth		Third birth	
	Mean	SD	Mean	SD	Mean	SD
Had a child in current month	0.005	0.072	0.011	0.105	0.002	0.045
Age	27.6	5.4	32.5	5.1	35.3	4.3
College education	0.38	0.49	0.31	0.46	0.31	0.46
Number of children to co-workers	20.5	18.9	23.6	20.2	25.6	20.8
Share fertile co-workers	0.67	0.25	0.62	0.25	0.59	0.25
Share close in age co-workers	0.23	0.20	0.20	0.18	0.20	0.17
Share female co-workers	0.65	0.29	0.66	0.30	0.67	0.31
Establishment size	18.2	12.5	18.1	12.6	18.2	12.4
Public sector	0.27	0.45	0.34	0.47	0.40	0.49
Private sector	0.73	0.45	0.66	0.47	0.60	0.49
Observations	5,575,497		2,015,434		3,730,264	
Individuals	139,020		60,534		73,518	

Table A2 Baseline estimates of co-workers' fertility on the probability of first birth

	(1)	(2)	(3)
First births	All	All	All
Any co-worker had a child within:			
12 months	0.00001	0.00001	0.00004
	(0.00007)	(0.00007)	(0.00007)
13-24 months	0.00057***	0.00056***	0.00048***
	(0.00007)	(0.00007)	(0.00007)
24-36 months	0.00033***	0.00033***	0.00018**
	(0.00007)	(0.00007)	(0.00007)
Married		0.01184***	0.01177***
		(0.00016)	(0.00016)
College education		0.00034***	0.00030***
		(0.00008)	(0.00008)
No children to all co-workers			0.00005***
			(0.00000)
Share fertile co-workers			0.00017
			(0.00015)
Share close-in-age co-workers			0.00051***
			(0.00017)
Share female co-workers			0.00087***
			(0.00011)
Share married co-workers			0.00026
			(0.00016)
Share co-workers with college edu.			0.00034***
			(0.00012)
Duration dummies	Yes	Yes	Yes
Year*Month dummies	Yes	Yes	Yes
Own characteristics	-	Yes	Yes
Establishment characteristics	-	-	Yes
Mean Y	0.00523	0.00523	0.00523
Observations	5,575,497	5,575,497	5,573,397

Notes: *,** and *** denote statistical significance at 10/5/1 percent level respectively. Standard errors robust for serial correlation at the establishment level are shown in parenthesis. The level of analysis is the individual-month. In addition to the fixed effects indicated by the table regression (3) controls for establishment size dummies in intervals of ten employees.

Table A3 Descriptive statistics for true and placebo peer groups

Sample:	Private firms with multiple workplaces		Job switchers		All	
	True: *same firm same workplace*	***Placebo:*** *same firm different workplace*	***True:*** *Current co-work.*	***Placebo:*** *Future co-work.*	***True:*** *All co-work..*	***Placebo:*** *Co-work. siblings*
Age	35.3	36.2	37.6	36.1	36.7	38.2
	(7.3)	(6.4)	(7.1)	(7.0)	(7.6)	(8.0)
Total # of children	18.5	1,178	20.3	19.9	20.5	19.05
	(16.4)	(2196)	(18.6)	(18.5)	(18.9)	(17.93)
Female	0.64	0.64	0.66	0.65	0.65	0.49
	(0.27)	(0.26)	(0.29)	(0.29)	(0.29)	(0.211)
Fertile	0.69	0.66	0.64	0.63	0.65	0.57
	(0.22)	(0.18)	(0.24)	(0.23)	(0.24)	(0.242)
High Edu.†	0.58	0.57	0.30	0.32	0.31	0.27
	(0.25)	(0.20)	(0.28)	(0.28)	(0.28)	(0.215)
Married	0.35	0.36	0.41	0.39	0.38	0.36
	(0.22)	(0.18)	(0.24)	(0.24)	(0.24)	(0.224)
This peer had a child within:						
12 months	0.39	0.81	0.34	0.39	0.36	0.36
	(0.49)	(0.40)	(0.47)	(0.49)	(0.479)	(0.480)
13-24 months	0.42	0.82	0.38	0.40	0.39	0.36
	(0.49)	(0.39)	(0.49)	(0.49)	(0.488)	(0.479)
25-36 months	0.42	0.82	0.37	0.38	0.37	0.34
	(0.49)	(0.38)	(0.48)	(0.49)	(0.484)	(0.472)
# obs.	1,066,052	1,066,052	730,356	730,356	5,575,497	5,385,787

Notes:† High education is defined as having college education. The co-worker characteristics are calculated at the individual-year level.

Table A4 Frequency of exposure and workplace size

	(1)	(2)	(3)	(4)	(5)	(6)
Nr of Employees	2-9	10-29	30-49	2-9	10-29	30-49
12 months	-0.0002	0.0001	0.00002	-0.0003*	0.0001	-0.0001
	(0.0002)	(0.0001)	(0.0001)	(0.0002)	(0.0001)	(0.0002)
13-24 months	0.0008***	0.0004***	0.0005***	0.0009***	0.0002**	0.0004**
	(0.0002)	(0.0001)	(0.0002)	(0.0002)	(0.0001)	(0.0002)
24-36 months	-0.0001	0.0001	0.0002	-0.00005	0.0002	0.0001
	(0.0002)	(0.0001)	(0.0002)	(0.0002)	(0.0001)	(0.0002)
Duration dummies	Yes	Yes	Yes	Yes	Yes	Yes
Year*month dummies	Yes	Yes	Yes	Yes	Yes	Yes
Own char.	Yes	Yes	Yes	Yes	Yes	Yes
Est. char.	Yes	Yes	Yes	Yes	Yes	Yes
More than one child	-	-	-	Yes	Yes	Yes
# Obs.	1,760,442	2,664,386	1,148,125	1,760,442	2,664,386	1,148,125

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