Halla, Martin; Zweimüller, Martina

Working Paper

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Working Paper, No. 1402

Provided in Cooperation with:
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Suggested Citation: Halla, Martin; Zweimüller, Martina (2014) : Parental response to early human capital shocks: Evidence from the Chernobyl accident, Working Paper, No. 1402, Johannes Kepler University of Linz, Department of Economics, Linz

This Version is available at:
https://hdl.handle.net/10419/97440

DEPARTMENT OF ECONOMICS
JOHANNES KEPLER UNIVERSITY OF LINZ

Parental Response to Early Human Capital Shocks: Evidence from the Chernobyl Accident

by

Martin HALLA
Martina ZWEIMÜLLER[*)]

Working Paper No. 1402
January 2014

Johannes Kepler University of Linz
Department of Economics
Altenberger Strasse 69
A-4040 Linz - Auhof, Austria
www.econ.jku.at

martina.zweimueller@jku.at
phone +43 (0)70 2468 -8308, -8209 (fax)

Parental Response to Early Human Capital Shocks: Evidence from the Chernobyl Accident*

Martin Halla
University of Linz & IZA

Martina Zweimüller
University of Linz

January 29, 2014

Abstract

Little is known about the response behavior of parents whose children are exposed to an early-life shock. In this paper we interpret the prenatal exposure of the Austrian 1986 cohort to radioactive fallout from the Chernobyl accident as a negative human capital shock and examine their parents' response behavior. To identify causal effects we can rely on exogenous variation in the exposure to radioactive fallout (over time and) between communities due to geographic differences in precipitation at the time of the accident. We find robust empirical evidence of compensating investment behavior by parents in response to the shock. Families with low socioeconomic status reduced their family size, while families with higher socioeconomic status responded with reduced maternal labor supply. Compensating investment made by the latter group seems relatively more effective because we do not find any detrimental long-term effects for exposed children from higher socioeconomic backgrounds. In contrast, exposed children from low socioeconomic backgrounds have significantly worse labor market outcomes as young adults.

JEL Classification: I18, I20, Q48, Q53, J24.
Keywords: Fetal origins, parental response, Chernobyl, radiation, health, culling, human capital, fertility, labor supply.

*Corresponding author: Martina Zweimüller, Johannes Kepler University of Linz, Department of Economics, Altenbergerstr. 69, 4040 Linz, ph.: +43 70 2468 8308, fax: +43 70 2468 8209, email: martina.zweimueller@jku.at. For helpful discussion and comments, we would like to thank Anders Björklund, Daniel S. Hamermesh, Matthew Lindquist, Erik Plug, Torsten Santavirta, and Rudolf Winter-Ebmer, and participants at seminars (Vienna University of Economics and Business, Stockholm University SOFI, University of Hamburg, IAB Nürnberg, Tinbergen Institute Amsterdam) and conferences (NOeG in Vienna, SOLE in Boston, ESPE in Aarhus, EALE in Torino). The usual disclaimer applies. Moreover, we thank the following institutions for providing us with data: *Environment Agency Austria*, *Statistics Austria*, *Main Association of Austrian Social Security Institutions*, and the *Austrian Federal Ministry of Finance*. This research was funded by the Körner Fonds and the Austrian Science Fund (FWF), National Research Network S103, The Austrian Center for Labor Economics, and the Analysis of the Welfare State. A Web Appendix provides additional material and is available on the authors' websites.

1 Introduction

The importance of early-life conditions for outcomes in later life is now widely recognized among scholars in different disciplines (Barker, 1995). Economists are particularly interested in the effect of early-life events on the accumulation of human capital (Almond and Currie, 2011a). In this context several factors complicate the isolation and interpretation of causal effects. Even in an ideal setting, i. e. where one observes an exogenous early-life shock and later outcomes, estimated effects are hard to interpret. These effects may not only entail the (biological) effect of the initial shock, but also the parental response to it. As a consequence one should to interpret theses effects as reduced form estimates.

Until very recently, the design-based literature on early-life events completely ignored parental responses (often for data reasons) and applied a simplified interpretation of their estimates. However, given that it is *a priori* unclear whether parents will make compensating or reinforcing investments — which are potentially asymmetric along different dimensions of human capital — these reduced form estimates cannot be even unambiguously interpreted as lower or upper bounds of the biological effect (Conti *et al.*, 2011).[1] Thus, it is crucial to examine the behavior of parents whose children were exposed to an early-life shock in order to fully understand the effect of early-life conditions for outcomes in later life.

In this paper we examine a shock during the prenatal period, which is considered as the key developmental window (Almond and Currie, 2011b), and focus on subsequent parental response behavior. In particular, we interpret the prenatal exposure of the Austrian 1986 cohort to radioactive fallout from the Chernobyl accident as a negative human capital shock. Thereby we follow Almond, Edlund and Palme (2009) (henceforth AEP), who show that Swedish children born in 1986 that were prenatally exposed to radioactive fallout had significantly lower grades in compulsory school at the age of 16.

Our identification strategy is (equivalently to AEP) based on the difference in rainfall levels while the radioactive plume was over Austria — which led to stark geographic variation in the levels of radioactive fallout.[2] In contrast to AEP (and papers examining other shocks), our main outcome of interest is the parental response behavior to this early-life shock and its mediating impact on childrens' long-term outcomes. The main result of

[1] As discussed by Almond and Mazumder (2013) there are a number of papers providing evidence on parental response behavior based on family fixed effects or more indirectly (for instance, via a comparison of OLS and fixed effects estimates). However, only a handful of papers with highly credible research designs directly examine parental response behavior. Among the latter, those papers in a development context (Adhvaryu and Nyshadham, 2012; Akresh *et al.*, 2012; Venkataramani, 2012) find clear evidence for reinforcing responses. The only paper analyzing data from a developed country finds no effects (Bharadwaj *et al.*, forthcoming).

[2] Austria ranks among the countries that received the most radioactive fallout. Differences in rainfall immediately after the accident caused substantial geographic variation in ground deposition of Caesium-137 fallout (half-life of 30 years) with maximum values of nearly 200 kilobecquerels per square meter. Only Russia, Ukraine, Belarus and some parts of Scandinavia had fallout values higher than 200 kilobecquerels per square meter; see Figure 3.5. in IAEA (2006).

our paper is that parents responded to this early-life shock with compensating investment behavior. Families with low socioeconomic status reduced their family size (i. e. lower completed fertility), while families with higher socioeconomic status responded with reduced maternal labor supply. Both effects indicate that affected children needed more attention, and parents adapted their behavior, such that they can devote more time to their children and make necessary compensating investment. The timing of the labor supply effect is especially intriguing — mothers of exposed children reduce their labor supply shortly after their children enter school. This pattern is consistent with reduced cognitive abilities of exposed children as put forward by AEP.

We proceed in several analytical steps to establish our main result. We start by re-examining the effect of prenatal exposure to radioactive fallout on children's health; AEP did not detect any health damage. In contrast, we find robust evidence that the in utero exposure to radioactive fallout from the Chernobyl accident in Austria increased the incidence of early fetal death. As a consequence, the surviving children exhibit better health outcomes at birth. This result illustrates the more general phenomenon that, in the presence of an effect of the early-life shock on mortality, a naïve comparison of long-term outcomes of exposed and (unselected) non-exposed children gives downward biased estimates. This tension between so-called *culling* and *scarring* effects is widely acknowledged among epidemiologist, but has so far been largely ignored in the economic literature on early-life shocks. The ignorance of this *culling effect* is especially aggravating, if the sample selection entails a social gradient, as in the case of radioactive exposure, where fetal deaths are highly concentrated in families with low socioeconomic status. We suggest a straight-forward methodology — which exploits the random assignment of the early-life shock and the fact that we can quantify the degree of the culling effect — to disentangle the culling and the scarring effect. After accounting for the sample selection due to culling, we observe a negative scarring effect on the health of the surviving children. Culling effects seem to be more important in the case of early exposure (first trimester), for short-run health outcomes, and of little significance in the case of long-run human capital outcomes.

In a second step we present the main results of our paper. We document the parental response to the early-life shock along the dimensions of family size and maternal labor supply over a period of twenty years. We find clear evidence for compensating parental responses. In a third step, we present estimates of the long-term effect on educational and labor market outcomes. In contrast to AEP, we can already observe the exposed cohort on the labor market. This reveals the impact of the accident (sum of biological effect and parental response) on the productivity of exposed children. We find that exposed children from low socioeconomic backgrounds have — despite their parents' compensating response — significantly worse labor market outcomes as young adults. In the case of exposed children from higher socioeconomic backgrounds we do not find any robust effects.

Our findings have important ramifications for the economic literature beyond the studies on the effects of early-life events on the accumulation of human capital. For instance, our analysis of parental response behavior is also helpful to understand intra-household resource allocation more generally. Our evidence on the fertility response shows that parents are aware of the trade-off between the quantity and the quality of children (Becker, 1960; Becker and Lewis, 1973; Willis, 1973; Becker and Tomes, 1976). Or, consider the literature on environmental justice — studying the disproportionately high exposure of the low-income population to environmental hazards and the resulting impact on their health and economic well-being. This literature typically faces the econometric challenge that exposure to environmental hazards is correlated with a host of confounding factors (Banzhaf and Walsh, 2008) that if unaccounted for may lead to biased estimates. Our research design provides the unique opportunity to observe a randomly assigned environmental hazard free of any Tiebout sorting on endogenous socioeconomic characteristics. Our findings reveal different dimensions of treatment effect heterogeneity. Children from families with low socioeconomic status are more vulnerable to early-life shocks, both in terms of short-run health outcomes, as well as in terms of long-run labor market outcomes. This result is consistent with two complementary explanations. First, these children may suffer more since they have on average a lower birth endowment. Second, the effectiveness of later compensatory behavior may increase along the socioeconomic distribution. Each of these explanations suggests that in case of conventional environmental hazards (such as air pollution as a byproduct of the production of a marketable good) the average treatment effect on the treated should be higher than the average treatment effect. Finally, our estimates of the effect of prenatal radioactive exposure on health outcomes contributes to a long-standing discussion in the medical literature. Whether radioactive fallout from the Chernobyl accident in 1986 had detrimental effects on individuals living in Western European countries or not is still a controversial question.[3]

Our results hold important implications for public policymakers. In terms of social policy, our results reinforce the call for public support to disadvantaged families that assures a sufficient level of early childhood investment. More specifically, our results also speak to the ongoing debates on nuclear power. An informed discussion about the efficiency of nuclear power requires knowledge about the full cost of nuclear and radiation accidents. At least, after the accident in the Fukushima Daiichi Nuclear Power Plant in March 2011 and the ongoing contamination of the environment, there are serious doubts that even an advanced economy can master nuclear safety. The benefits of nuclear power due to comparable low emissions have to be contrasted not only with the private and

[3]The clean identification strategy employed by AEP and in our paper distinguishes both from earlier Chernobyl studies (summarized in Web Appendix A). The effects of in utero exposure to radioactive fallout caused by nuclear weapon testing have been analyzed by Black *et al.* (2013). Based on a design-based approach the authors identify a negative effect of this comparable lower dose of radioactivity on long-term human capital outcomes (see below) and a quantitatively small effect on height at age 18.

social cost involved in the normal operation, but also with the expected total cost of a nuclear accident. Our estimation results provide evidence that accidents in nuclear power plants have large and long-lasting negative externalities (due to radioactive fallout) even for individuals living about 1,000 miles away; which even translate into reduced economic productivity and income many years after an accident.

The remainder of the paper is organized as follows. Section 2 describes the Chernobyl accident and the resulting radioactive contamination of the western part of the former Soviet Union and Europe. Section 3 presents our identification strategy, the econometric specification, and the data used. Section 4 discusses our estimation results. Finally, Section 5 concludes the paper.

2 Contamination of the environment due to the Chernobyl accident

On April 26, 1986 at 1:23 A.M. an accident occurred during a systems test at the Chernobyl nuclear power plant in Ukraine (officially the Ukrainian Soviet Socialist Republic) that caused the worst nuclear power plant accident in history (until then). An explosion and fire released large quantities of radioactive contamination into the atmosphere that was not stopped until May 6, 1986.[4] As a result, a plume of highly radioactive fallout spread over an extensive geographical area and drifted in the following days over large parts of the western part of the former Soviet Union and Europe.[5] The radioactive particles were subsequently removed from the atmosphere solely due to gravitation (*dry deposition*) or by any form of precipitation (*wet deposition*). Given that wet disposition is by far a more efficient deposition mechanism (compared to dry deposition), the level of radioactive material deposited on soil and other surfaces (so-called *ground deposition*) was predominantly determined by the presence or absence of precipitation during the passage of the plume (Clark and Smith, 1988).

Radionuclides can enter the human body through inhalation, ingestion, and absorption through the skin. The IAEA (2006, Chapter 5) describes four main pathways by which humans were exposed to the radioactive material released by the accident: (i) external dose from cloud passage, (ii) internal dose from inhalation of the cloud and resuspended material, (iii) external dose from ground deposition, and (iv) internal dose from the consumption of contaminated food and water. The latter two exposure pathways are

[4] This incidence was not immediately announced by the authorities of the Soviet Union, but has been detected after radiation levels set off alarms at a nuclear power plant in Sweden located over one thousand kilometers away from Chernobyl. The world learned officially about the accident (two days later) on April 28, 1986 through a 20 second announcement by the state television broadcaster in the Soviet Union.

[5] The following link provides a computerized graphic reconstruction of the path of the first 14 days of the radioactive plume, tracking the release of Caesium-137, created by the *Institut de Radioprotection et Sûreté Nucléaire*: http://www.irsn.fr/FR/popup/Pages/tchernobyl_animation_nuage2.aspx.

considered as the most important. Thus, humans were exposed to high levels of radiation if they were located in areas with high levels of ground deposition and/or if they consumed large quantities of contaminated food and water. While it is not observable who consumed large quantities of contaminated edibles, the external dose from ground deposition should be highly correlated with the observable local level of ground deposition at individuals' place of residence. From a researcher's point of view the Chernobyl disaster provides a good natural experiment to study the consequences of exposure to radioactive ground deposition, since it seems save to assume that the spatial distribution of precipitation during the passage of the plume was random.[6]

The implementation of this research design is facilitated by the wide availability of data on local levels of radioactive ground deposition. In the aftermath of the accident the level of ground deposition of Caesium-137 (henceforth ^{137}Cs) and other radionuclides was measured comprehensively on the soil surface in most European countries (European Commission, 1998). In the mapping of the deposition the focus was on ^{137}Cs, because it is easy to measure (*ex post*), and because of its radiological significance. It turned out that the three countries (based on current borders) most heavily affected are Belarus, the Russian Federation, and Ukraine. However, Austria, Sweden and Finland also contain some heavily contaminated areas (see, for instance, Figure 3.5. in IAEA (2006)).

3 Research design

We present now the Austrian radiation data that we use to determine the individual level of exposure to radioactive fallout. Then we discuss the periods of prenatal exposure which we distinguish in our analysis and explain how we translate our research design into a regression framework. There we distinguish between radiation and non-radiation effects of the Chernobyl accident. When we introduce our outcome variables we also provide information on our data sources.

3.1 Spatial distribution of radioactive fallout

In Austria radioactive fallout (due to Chernobyl) was measured at 1,881 sites, which provides on average one measurement per 45 square kilometers (Bossew *et al.*, 1996, 2001). Radioactive fallout is measured as ground deposition of ^{137}Cs (with a half-life of 30 years) and ^{134}Cs (with a half-life of 2 years) in kilobecquerels per square meter (kBq/m^2).[7] We aggregate these measurements to the community level and focus on the

[6] To be precise, our estimates may not only capture the effect of the exposure to radioactive ground deposition, but partly also the effect of the internal dose from the consumption of contaminated food and water, to the extent which this is correlated with the external dose from ground deposition. It is hard to asses how large this correlation is, since it depends on the structure of the food supply chain.

[7] Immediately after the arrival of the radioactive cloud 336 dose rate meters distributed over the territory of Austria quantified the gamma radiation (in millisievert). These measurements show a high

average level of ground deposition of ^{137}Cs.[8] This provides us with data for 924 (out of 2,331) communities, where each data point refers to May 1, 1986.[9] The accident happened on April 26, however, the radioactive plume arrived on April 29 in Austria. Figure 1 depicts the spatial distribution of contamination, where we distinguish between communities with a ground deposition of ^{137}Cs below 17 kBq/m^2, between 17 and 36 kBq/m^2, at least 37 kBq/m^2, and communities without data. UNSCEAR (2000) considers regions with a ^{137}Cs ground deposition of 37 kBq/m^2 or more as contaminated. In Austria, the average level of contamination was around 20 kBq/m^2. Communities with the lowest level of contamination recorded only 0.7 kBq/m^2, while the most contaminated areas had values of about 150 kBq/m^2. It is this wide range of (within country) variation in radioactive fallout (resulting from the very local presence or absence of precipitation during the passage of the plume) that makes the Austrian case so particularly well suited for studying the impact of the Chernobyl accident.

In order to define our treatment and control group, we distinguish between communities (and their residing population) who were exposed to different levels of radioactive fallout. We follow the criteria suggested by UNSCEAR (2000) and define the 175 communities in our sample with a ^{137}Cs ground deposition of 37 kBq/m^2 or more as treatment group 1 ($T1$). We specify two further treatment groups with higher levels of contamination. To the 130 communities with a ^{137}Cs ground deposition of 42 kBq/m^2 or more we refer as treatment group 2 ($T2$), and to the 93 communities with a ^{137}Cs ground deposition of 47 kBq/m^2 or more we refer as treatment group 3 ($T3$). As a control group (C) we use (in each case) the 427 communities with a ^{137}Cs ground deposition of 16 kBq/m^2 or less. Communities with medium levels of ^{137}Cs ground deposition (i. e. between 17 and 36/41/46 kBq/m^2) are excluded from the analysis.[10] Table 1 summarizes this grouping

correlation with the deposition measurements of ^{137}Cs and ^{134}Cs (Bossew *et al.*, 2001).

[8] These measurements include the global fallout from the atmospheric atomic bomb tests in the 1950s and 1960s. For a reduced number of sites we have equivalent data on the ground deposition of ^{134}Cs. This fallout stems exclusively from the Chernobyl accident and allows to isolate (with some error) the ^{137}Cs ground deposition originating from the Chernobyl accident only. Estimations based on these alternative measurements gives very comparable results. In particular, the point estimates are very similar, however, the standard errors increase. The latter fact can be explained by the reduced sample size (about 65 percent of the original estimation sample) and the increased measurement error in the alternative treatment variable.

[9] Table B.1 in Web Appendix B compares birth outcomes of children born and conceived before Chernobyl in the communities with and without data on ^{137}Cs ground deposition. No quantitatively important differences can be found. Aggregating the Caesium data to a higher administrative level — the county level — increases the geographic coverage at the expense of introducing measurement error. Based on 109 counties (including 99 percent of communities) we have checked the robustness of our main results for birth outcomes. The county-level results (available upon request) are very similar to the results presented in Table 5.

[10] Our main results for birth outcomes (for the three treatment groups defined above) are quantitatively and qualitatively similar (to those presented in Table 5) when including communities with medium levels of contamination. The point estimates for communities exposed to medium levels are lower and not significant throughout. We have also used a continuous measure of exposure to ^{137}Cs ground deposition. Again, these results are robust. Note that these results are available upon request.

of communities and provides the population-weighted average of ^{137}Cs ground deposition for each group.

Given that the level of radioactive fallout was predominantly determined by the level of precipitation during the passage of the plume, we observe a correlation between ground deposition and the *general* level of precipitation. Since precipitation intensity is mainly determined by high-altitude, we find higher levels of ground deposition in high-altitude areas. The average altitude of control communities is about 433 meters above sea-level, while those of $T1$ communities amounts to approximately 602 meters above sea-level. Since the population composition differs between low and high-lands, we find some pre-treatment differences in average socioeconomic characteristics of non-exposed and exposed communities. Notably, in terms of birth outcomes — such as sex of child, premature birth or low birth weight — we do not see any statistical significant differences. (See, for instance, Panel (a) of Figure 3). In any case, our difference-in-differences estimation framework (to be explained below) will clear any time-constant differences.[11]

3.2 Periods of prenatal exposure

It is conjectured that radiation exposure is especially critical at a prenatal stage.[12] While a human embryo or fetus is protected in the uterus, and the radiation exposure to a fetus should be lower than the dose to its mother, an embryo or fetus is particularly sensitive to ionizing radiation. The most important determinant of potential health effects is radiation dose and gestational age (ICPR, 2003). Exposure to radiation in the pre-implantation period has very likely lethal effects. During the period of major organogenesis (weeks 2–7 postconception), most human organs are formed and the embryo is sensitive to malformations and growth retardation. Negative effects on the brain development are most likely in weeks 8–15 (and to a lesser degree in weeks 16–25) postconception. Beyond about 26 weeks, the fetus is believed to be 'relatively radio-resistant' (i. e. equally sensitive to radiation as a newborn).

AEP (who are mainly concerned with cognitive outcome) focus on children of gestational age 8 to 25 weeks at the time of the accident. We employ a broader specification and include all children who where prenatally exposed to Chernobyl. Our estimation sample comprises all conceptions between August 1, 1984 and July 31, 1987. Including conceptions from this time span also guarantees that we can control for seasonal effects at the monthly level. As depicted by Figure 2 we distinguish between four different birth cohorts.

[11] Our results for birth outcomes are robust to the inclusion of indicators for maternal age, labor market status, foreign nationality, religious denomination, marital status (and detailed maternal education).

[12] The empirical evidence on the effects of prenatal exposure on child health is either based on case studies of children born to women who had been treated with high doses of medical radiation while pregnant (De Santis *et al.*, 2005) or on children who have been prenatally close to the hypocenter of the atomic bomb explosions in Hiroshima and Nagasaki (Otake and Schull, 1998; Yamazaki and Schull, 1990). The only two exceptions we are aware of are the aforementioned papers: AEP and Black *et al.* (2013).

Birth cohort 0 (BC_0) includes all children who were conceived before August 1, 1985 and born before the Chernobyl accident.[13] Further we distinguish between two birth cohorts who were exposed to Chernobyl in utero. Birth cohort I (BC_I) comprises children who were conceived between August 1, 1985 and January 31, 1986. These children have been in utero for more than 3 months at the time of the accident (second & third trimester) and should be comparable resistant to radioactive exposure due to their gestational age. Children belonging to birth cohort II (BC_{II}) were conceived between February 1, 1986 and April 30, 1986. That means, they have been in utero for less than 3 months at the time of the accident (first trimester) and should still be very vulnerable.[14] For simplicity we refer to children from BC_I and BC_{II} as the 1986 birth cohort. Finally, children from birth cohort III (BC_{III}) were conceived and born after the accident (between May 1, 1986 and July 31, 1987). While this prenatally non-exposed cohort is *per se* not interesting, its inclusion allows us to fully account for seasonal and year effects.

Our broader specification does not only allow us to check the effect of in utero exposure at different gestational age, but also to identify potential non-radiation effects triggered by an early parental response behavior that is causally related to the Chernobyl accident, but not caused by radiation. For instance, since BC_{II} has been in utero for less than 3 months at the time of the accident, an induced abortion would have still been possible. In contrast, children from BC_I have not been at risk to be aborted.

Naturally, we do not observe the exact day of conception in our data. Based on the stated gestation length measured in commenced weeks (gl) and the birth day (bd) we compute the conception day (cd) as follows $cd = bd - 7 * (gl - 0.5)$. That means, we assume that a pregnancy with a stated gestation length of 38 weeks has lasted 38.5 weeks or 269.5 days. In order to minimize errors in group assignment, we exclude conceptions 7 days before and after each cutoff date. Moreover, we exclude births by very young and very old mothers and focus on children born to mothers between the age of 20 and 40. In case of families where one child belongs to BC_I and she has a younger sibling in BC_{III}, we exclude the later child from our analysis, since s/he might be affected by her sibling's treatment status.

After applying those sample selection criteria, our most extensive sample includes 94,920 children (see Table 1). The number of observations depends on the treatment group definition that we use. In case of $T1$, we observe 22,455 exposed children and 72,465 non-exposed children and exclude 36,685 children from communities with medium levels of ^{137} Cs ground deposition. We do not exclude multiple births (about 2 percent of children), however, when analyzing parental response behavior we include only one

[13] Post-term births (i. e. with gestational length of 40 weeks or more) may be born after the accident.

[14] In line with AEP we find that an exposure at an earlier gestational age has the most detrimental effects. In particular, we find that an exposure before week 8 is particularly critical for fetal mortality. AEP define this group as non-treated. As we will show below, this provides a plausible explanation why AEP do not find any health effects of Chernobyl.

observation per birth.

3.3 Econometric specification

Our research design translates into the following regression framework, which is performed for each definition of the treatment group $T\#$ ($T1$, $T2$ and $T3$):

$$\begin{aligned} Outcome_{i,c} = \alpha + \rho_1 BC_I + \rho_2 BC_{II} + \tau_1 BC_I \times T\#_{i,c} + \tau_2 BC_{II} \times T\#_{i,c} \\ + \eta T\#_{i,c} + \beta_1 BC_{III} + \beta_2 BC_{III} \times T\#_{i,c} + \mathbf{X}_{i,c} + \gamma_y + \delta_m + \theta_c + \epsilon_{i,c}. \end{aligned} \tag{1}$$

In this equation i denotes individual and c denotes community. This *difference-in-differences* (DiD) estimation framework includes binary variables BC_I, BC_{II} and BC_{III} to distinguish between children from three birth cohorts as defined above, a binary variable indicating the treatment status of each child's community of residence at birth ($T\#_{i,c}$), and an interaction term between each birth cohort indicator and the treatment status variable. Further, we control for conception year fixed-effects (γ_y), conception month fixed-effects (δ_m) and community fixed-effects (θ_c). Depending on the specific outcome we control for further covariates $\mathbf{X}_{i,c}$. (The treatment indicator $T\#_{i,c}$ is dropped because of perfect collinearity with the community fixed-effects θ_c.) With one exception (live births) all outcomes are measured on an individual-level. Depending on whether we analyze a child outcome or parental behavior, the index i refers to either the child or its parent(s).

The parameters τ_1 and τ_2 provide the estimated prenatal radiation effects (i. e. the true causal effect of radioactive fallout) for BC_I and BC_{II}. We refer to these effects as *radiation effects*, which are the parameters of primary interest. The identification of these parameters relies on variation in the exposure to radioactive fallout (over time and) between communities due to geographic differences in precipitation after the accident. To be precise, given that we estimate these effects with a DiD procedure, all we have to assume is that exposed and non-exposed children would have followed a parallel trend in the respective outcome, in the counterfactual situation without the Chernobyl accident. This assumption seems quite plausible.

Given that the local level of ground deposition was not know at the time of the accident, all parents (or even potential parents) may have been stressed and anxious in the aftermath of the accident and may have changed their behavior immediately in response to the accident irrespective of their treatment status.[15] Put differently, one might

[15] We have scanned three major national newspapers (*Die Presse*, *Neue Kronen Zeitung*, *Oberösterreichische Nachrichten*) in the period from April 29 through June 18, 1986 for all articles relating to the Chernobyl accident. In general the coverage was very confusing and inconsistent. For instance, while the population was informed about radioactivity in milk and dairy products and was requested to carefully wash vegetables and fruit, an expert from the Institute of Atomic and Subatomic Physics at the Vienna University of Technology considered the level of radioactive fallout erroneously as low as the level of

distinguish between two treatments: everyone was treated with the Chernobyl accident (first treatment), but only a sub-population was in addition exposed to significant levels of ground deposition (second treatment). The parental response to the first treatment may have affected different dimensions, such as maternal diet (of pregnant women), the decision to have a child, and even the likelihood of having an induced abortion. That means, this early parental response behavior may have generated scarring and/or culling effects. Given that these effects are causally related to the accident, but constitute a distinct channel which should not be mixed up with the biological effects of radiation, we refer to them as *non-radiation effects*.

Non-radiation effects for BC_I and BC_{II} are captured by the parameters ρ_1 and ρ_2, if early parental response did not vary with the exposure to radioactive fallout. While the validity of this assumption is not as clear as the identifying assumption of the radiation effects, it can be justified by the fact that the actual level of *local* radioactive fallout was verifiably not known at the time of the accident. Therefore, we think it is reasonable to assume that early parental response behavior is not systematically correlated with the actual level of radiation exposure (as measured in retrospect).

The method of estimation is least squares and robust standard errors — allowing for clustering by community and heteroskedasticity of unknown form — are calculated throughout. Given that we find a strong social gradient in the effects of prenatal exposure to radiation, we present a discussion based on a separate estimation analysis for children from low and high socioeconomic backgrounds. In particular, we use the available information on mother's educational attainment at the time of birth to distinguish between low socioeconomic backgrounds (i. e. mother has compulsory schooling or less) and higher socioeconomic backgrounds (i. e. mother has any degree higher than compulsory schooling). According to this definition about 26 percent of children have a low socioeconomic background.

3.4 Outcome variables

We examine health and human capital outcomes available in administrative data sources that allow us to infer on the effects of the early-life shock on children at a prenatal stage, at the time of birth, during adolescence, and early adulthood. Parental response behavior is evaluated in terms of fertility and maternal labor supply in the post-treatment period. Table 2 provides an overview of all (potential) outcomes with information on measurement and data source.

For the estimation of prenatal culling effects we use the *Austrian Birth Register*. This

radioactive fallout caused by nuclear weapon testing in the 1960s and did not expect any health effects on the Austrian population (Die Presse, May 17, 1986). Most importantly, we did not find any systematic information on local levels of radioactive contamination. There are some scattered statements refering to federal states.

includes the universe of all live births and stillbirths in Austria with individual-level information on socioeconomic characteristics and birth outcomes. This data allows to quantify the incidence of live births on a community-level (by socioeconomic groups). For the estimation of postnatal culling effects we link the *Austrian Birth Register* with the *Austrian Death Register*, which enables us to estimate the likelihood of infant mortality on an individual-level. The analysis of short-run scarring effects examines health at birth and focuses on different health indicators such as the gestation length, birth weight and Apgar scores which are documented in the *Austrian Birth Register*. To evaluate long-term scarring effects we focus on human capital formation and labor market outcomes in early adulthood (up to an age of 23). In particular, we obtain individual-level information on employment, broad occupation, apprenticeship training and wages in the *Austrian Social Security Database*. To evaluate parental postnatal investment behavior we use family size (based on completed fertility) and maternal labor supply.

4 Estimation results

In this section we first highlight the crucial tension between culling and scarring effects that has to be considered in the interpretation of empirical estimates of the effect of the Chernobyl accident, in order *not* to underestimate the true impact. We quantify the importance of culling effects and suggest a simple sample correction method. Following this we analyze the effect of Chernobyl on children's health at birth and reconcile our results with those of AEP. Then we present our main results on the parental response behavior and discuss non-radiation effects. Finally, we analyze the long-run effects on children's human capital outcomes.

4.1 Culling effects

The possible effects of prenatal radiation exposure include increased risk for medical conditions later in life (such as cancer), but also immediate effects, such as malformations or even fetal death (ICPR, 2003). Thus, radioactive exposure experienced in utero may do more than 'scar' exposed children. It may increase mortality at different stages of development. This so-called culling effect may lead to a potentially selected sample of survivors at any point in time after the initial shock — where selection is endogenous to the same shock as the scarring effect. This imposes two empirical challenges for our analysis of the parental response to this early-life shock and its impact on children's long-term outcomes. First, we need to estimate the extent of culling. Second, we need an empirical strategy to disentangle scarring and culling effects.

4.1.1 Quantification of culling effects

The *Centers for Disease Control and Prevention (CDC)* concludes that the risk of fetal death increases in the radiation dose and decreases with gestational age.[16] Mortality risk is especially high in the first weeks after conception, since an embryo is made up of only a few cells. A damage to one cell (the progenitor of many other cells) may cause the death of the embryo, and the blastocyst will fail to implant in the uterus. Beyond about 26 weeks, the fetus is believed to be 'relatively radio-resistent' (i. e. equally sensitive to radiation as a newborn).

Prenatal culling In order to calculate the extent of prenatal culling one would have to compare the number of conceptions with the number of live births. Clearly, we do not observe the number of conceptions. We also do not have information on the incidence of miscarriages (medically termed *spontaneous abortions*). Very early miscarriages (so-called early pregnancy losses) happen in many cases before a woman may know she is pregnant and, therefore, without clinical recognition. Later miscarriages, which occur after the sixth week since the woman's last menstrual period (so-called *clinical spontaneous abortion*) are not universally documented in Austria. As in most countries, Austria begins its comprehensive documentation of fetal mortality with stillbirths. A stillbirth is defined as birth of a child of at least 35 centimeter of length, without vital signs (body temperature, heart rate, blood pressure, respiratory rate).[17] Smaller fetus are categorized as miscarriages, and therefore, not documented. Finally, live births are very well documented in the *Austrian Birth Register* which comprises individual-level data on the parents and the new-born. Information on induced abortions is not available. Although, abortion is legal in Austria already since 1975, no official statistics exist.

In order to infer on the effects of Chernobyl on prenatal culling we offer three complementary strategies. First, we examine the incidence of live births on a community level. A lower estimated number of live births in exposed communities would provide evidence for prenatal culling. Second, we follow Sanders and Stoecker (2011) and use the sex-ratio of live births as a metric of fetal death. This methodology is based on an evolutionary theory advocated by Trivers and Willard (1973). The so-called *Trivers-Willard Hypothesis* states that the population sex-ratio responds to parental conditions through prenatal selection. It predicts that mothers in good conditions are expected to have more sons, while mothers in poor conditions should have more daughters.[18] The precise prenatal mechanism how

[16] In contrast, carcinogenic risks are assumed to be constant throughout the pregnancy.

[17] The definition of stillbirths (in particular, the differentiation to miscarriages) varies somewhat across countries (Heisler, 2012). In Austria, the stated definition was valid throughout our sample period (until 1994). Since 1995, a stillbirth is defined as a birth of a child of at least 500 grams weight without vital signs.

[18] This prediction can be rationalized by a biological mechanism that tries to maximize the reproductive success of the offspring. Given that offspring health is correlated with parental health and that differences in offspring health will persist into adulthood, the main assumption is that the relationship between

mothers (or their reproductive system) 'favor' either female or male offspring, depending on their condition, is still debated (Navara, 2010). The adjustment of the sex ratio may either take place at the primary or the secondary level. While a lower primary sex ratio is the result of a lower proportion of male offspring present at fertilization, a lower secondary sex ratio results from a lower likelihood of implantation of the blastocyst or a higher likelihood of male fetal loss. There are two empirical observations which are in line with the Trivers-Willard Hypothesis. First, male fetus are more fragile than female fetus (Kraemer, 2000). Second, there exist robust empirical evidence that women in poor health (or under less favorable conditions) are less likely to have male offspring (see, for instance Almond and Edlund, 2007; Catalano *et al.*, 2005; Catalano and Bruckner, 2006; Hansen *et al.*, 1999). In our empirical analysis we associate a decreased probability of male births with an increase in miscarriages (including early pregnancy losses).

Our third strategy is to examine the probability of a stillbirth based on the sample of all births (i. e. sum of stillbirths and live births). Table 3 summarizes the estimated effects on prenatal culling for BC_{II}, which was in the first trimester post conception at the time of the accident.

Families with low socioeconomic status The first panel shows the estimation results for the incidence of live births based on monthly community-level data. The dependent variable is equal to the number of live births per 1,000 female inhabitants aged between 15 and 39 in the respective educational attainment group divided by the respective sample mean. We find a statistically significant negative effect, which provides first evidence on prenatal culling. The effect amounts to 8.6 to 11.2 percent fewer live births in exposed communities and is significant for all three definitions of exposure to radiation. This reduction in live births should be accompanied by an increase in the incidence of miscarriages or stillbirths (or both). Put differently, either some children are stillbirths (which are documented) or they die at an earlier stage of the pregnancy.

The second panel summarizes the estimated effects on the likelihood of a stillbirth based on individual-level data. We do not find any statistically significant effects. This suggests that radioactive exposure should lead to a higher incidence of miscarriages. While we can not directly observe miscarriages, we can use the sex of the child as a proxy variable. As the results summarized in the third panel show, we indeed find a statistically significant negative effect on the likelihood of a male birth in exposed communities. Exposure to radiation in the first trimester post conception reduces the likelihood of a male birth by 4.3 to 8.8 percentage points. The size and significance of this effect increases with the level of radioactive exposure. Following the literature and interpreting this as evidence for miscarriages, this finding is consistent with the negative effect on live births (and the

offspring health and mating success is less pronounced for women (compared to men). Put differently, since males can in principle have children with multiple women, healthy males could secure several mates, while males in poor health secure none. In contrast, in the case of females, mating with healthy men is also possible for females in poor health.

zero effect on stillbirths).

In order to assess the quantitative importance of the effect of radiation exposure on prenatal culling it is useful to consider the baseline rate of conceptions which are lost (aborted) spontaneously. The incidence of spontaneous abortion is widely believed to be about 40 percent of all pregnancies (Macklon *et al.*, 2002).[19] That means, the effect of prenatal radiation exposure of roughly plus ten percentage points equates to an odds ratio of $[(0.4 \times 1.1) \times 0.6] \setminus [(1 - 0.4 \times 1.1) \times 0.4] = 1.18$. In comparison, women who smoked more than 10 cigarettes per day during the first trimester have an estimated increased risk of spontaneous abortion with an odds ratio of 1.40 (Chatenoud *et al.*, 1998).

Families with higher socioeconomic status For exposed mothers with higher socioeconomic status we find — with the exception of one coefficient — also point estimates in line with less live births and more miscarriages. However, the effects are not statistically significant at conventional levels. This suggest that there is a significant social gradient in the effect of prenatal exposure to radiation on culling. This finding is consistent with two explanations. First, it is well-documented that mothers with low socioeconomic status tend to have less favorable pregnancy outcomes (Kramer, 1987; Currie and Moretti, 2003). That means, unborn children of these mothers are also weaker at any prenatal stage, and any negative shock should have more detrimental effects. Second, our finding is in principle consistent with research on differences in the reaction of low and highly educated individuals to emerging health risk information. For instance, Aizer and Stroud (2010) show that highly educated women immediately reduced smoking in response to the *1964 Surgeon General Report on Smoking and Health*, while the low educated did not. Or, Anderberg *et al.* (2011) find evidence for a social gradient in the response to the measles, mumps and rubella (MMR) controversy in the UK. In the case of the Chernobyl accident this would imply that mothers with higher socioeconomic status — residing in exposed, as well in non-exposed communities — tried to reduce their exposure to radiation (without knowing their actual treatment status). This could only be achieved by staying inside over a longer period of time and/or by avoiding contaminated food and water. It is unclear to which degree such an avoidance behavior was feasible.

Exposure at higher gestational age For children from low socioeconomic backgrounds belonging to BC_I, which were already in their second or third trimester, we find less significant effects. Estimation output is provided in Table C.1.1 in Web Appendix C.1. The point estimates on live births are somewhat lower but the standard errors are unchanged. The effects on stillbirths are even smaller (and essentially zero). We do not find any evidence on the incidence of fetal death. These results are consistent with the existing evidence on the heterogenous impact of radioactive exposure over the gestation

[19] Macklon *et al.* (2002) suggest a rate of 30 percent early pregnancy losses and a rate of 10 percent clinical miscarriages ($30 + 10 = 40$ percent). Furthermore, 30 percent of conceptus fail to implant, resulting in 30 live births per 100 conceptions.

period. In the case of children from higher socioeconomic backgrounds the effects for BC_I are smaller compared to those for BC_{II}; i. e. they provide even less evidence for prenatal culling).

In sum this set of results provides robust evidence that prenatal radiation exposure (of about ^{137}Cs ground deposition of 37 kBq/m^2 or higher) during the first trimester post conception significantly increased prenatal culling among mothers with low socioeconomic status. Our analysis also reveals that male embryos and fetus are more vulnerable to radiation compared to female ones, and prenatal radiation exposure has the potential to distort the sex-ratio at birth.

Postnatal culling To test for any effects of radioactive exposure on postnatal culling, we examine infant survival at different points in time after birth: after twenty-four hours, after seven days, after one month, and after one year. The main estimation results for BC_{II} are summarized in Table 4. Overall, we find little evidence for culling after birth. In the case of exposed mothers with low socioeconomic status we do not observe any statistically significant effects. This suggest that exposed children (who survived the prenatal culling stage) were in no different physical condition as compared to non-exposed children, or at least sufficiently healthy to survive the first year. Among children from exposed families with higher socioeconomic status, we observe some significantly positive effects on survival in communities with the highest radioactive exposure. The effects are significant one week and one month after birth. One year after birth no significant difference are existent. This suggest that prenatal culling led to a slightly positively selected sample of live births in this particular group.

Exposure at higher gestational age In the case of children from low socioeconomic backgrounds the effects for BC_I are equal to those for BC_{II} (i. e. no evidence for postnatal culling). In the case of children from higher socioeconomic backgrounds we find some differences. In fact, post-natal culling is the only outcome, where we find more pronounced effects for children from BC_I (as compared to BC_{II}). We find a reduced infant survival of BC_I (where effects are driven by mortality within the first week after birth). Estimation output is provided in Table C.1.2 in Web Appendix C.1. Prenatal exposure to radioactive fallout at a higher gestational age might have not been harmful enough to cause fetal death, but may have caused harm, which lead to death shortly after birth.

4.1.2 Cancelling out culling effects

To motivate our empirical strategy which allows us to disentangle culling and scarring effects we formalize the tension between these two effects. While it is framed with respect to fetal mortality — which turned out to be empirically most relevant stage — the same arguments apply to mortality at any point in time after the initial shock.

We start by assuming that each fetus i has some potential endowment b_i, which is

distributed in the population with a cumulative distribution function $F(b)$ and density function $f(b)$. Fetus with an endowment below or equal to the cutoff z will not be born alive. Radioactive exposure in utero may have two effects: First, it may reduce the odds of survival conditional on the birth endowment (*culling*), and second, it may shift the distribution of birth endowments to the left (*scarring*). Let us first consider the culling effect only; radioactive exposure shifts the survival cutoff from z to $z + r$, such that a fetus is born alive only if

$$b_i > z + r \tag{2}$$

The fetal mortality rate m^T in exposed communities T is then given by

$$m^T = F(z + r). \tag{3}$$

In addition, radioactive exposure may also reduce the fetus' birth endowment by some fraction τ of r, which shifts the distribution of birth endowments to the left. (We may want to call τ the scarring-parameter.) Given that this fraction is permanently lost, radioactive exposure may also have long lasting effects on the survivors.[20] Thus, for survivors, we have

$$\tilde{b}_i = b_i - \tau r. \tag{4}$$

The average birth endowment of the survivors in exposed communities is thengiven by

$$\bar{b}^T = \underbrace{\frac{\int_{z+r}^{\infty} b \cdot f(b) db}{1 - F(z + r)}}_{\text{Culling}} - \underbrace{\tau r}_{\text{Scarring}}. \tag{5}$$

The first term is increasing in the level of radioactive exposure (i. e. the average birth endowment increases), while the second term is negative and increases in absolute terms with higher levels of radioactive exposure. This tension between culling (positive effect on birth endowment) and scarring effects (negative effect on birth endowment) has been long recognized in epidemiology.

Since the distribution of b_i, the value of z and the scarring parameter τ are unknown, we generally cannot disentangle culling and scarring effects without further assumptions. However, due to random assignment into treatment we can assume that the untruncated distribution of birth endowments is equal in exposed and non-exposed communities. Panel

[20] For simplicity and following Bozzoli *et al.* (2009) we assume that only the birth endowment of a survivor is reduced by τr. In the case that all fetus' birth endowments are reduced the mortality rate is given by

$$\hat{m}^T = F(z + r + \tau r)$$

because then a fetus only survives if the reduced birth endowment $b_i - \tau r$ is above the survival cutoff $z + r$, implying

$$b_i > z + r + \tau r.$$

(a) of Figure 3 shows the empirical cumulative distribution of gestation length for mothers with low socioeconomic status in BC_0 providing evidence that substantiates this assumption. The average birth endowment of children born in non-exposed communities — i. e. the untruncated distribution — is then given by

$$\bar{b}^C = \frac{\int_z^\infty b \cdot f(b)db}{1 - F(z)}. \tag{6}$$

We suggest a sample correction method to disentangle culling and scarring effects. Given that assignment into treatment was random, we can implement a simple method, which adjusts the sample of non-exposed children such that it is comparable to the 'culled' sample of exposed children. The distribution of the birth endowment in the sample of non-exposed children is a mixture of two distributions: (i) the distribution for children who would survive irrespective of their treatment status (children with $b_i > z + r$), and (ii) the distribution for children who survive only in the absence of the treatment (children with $z < b_i \leq z + r$). We basically manually cull the control group by removing those children who would not have survived if they had been exposed to the Chernobyl accident.[21]

Therefore, we exploit our knowledge about the extent and nature of the culling effects caused by the Chernobyl accident. We have estimated the extent of prenatal culling to be about 10 percent among exposed mothers with low socioeconomic status. This is the difference in the fetal mortality rate between treated m^T and control regions m^C, which we denote by $\Delta m \equiv m^T - m^C$. Since the fetal mortality rate in control regions C is defined as $m^C = F(z)$, it follows that

$$\Delta m = F(z + r) - F(z) \tag{7}$$

Regarding the nature of the culling process, we have to decide *which* 10 percent of the control group have to be excluded. It seems plausible that culling tends to eliminate those in poor health (i. e., survivors of Chernobyl should generally be positively selected). To implement our sample correction method we use the gestation length as a proxy for the strength of the fetus (its birth endowment) and exclude the lowest decile of the control group. Given that the assignment into treatment was random, this simple sample correction method should give us two comparable samples of treated and control units; net of culling effects. Estimation based on the two culled samples — where one was culled by the treatment, and the other by our sample correction method — should allow us to isolate any scarring effects.

Panel (b) of Figure 3 shows the observed distribution of gestation length for mothers

[21] Our procedure is comparable to the one suggested by Lee (2008). In contrast, we use the culled sample to analyze further outcome variables and only compute the upper bound estimate (and not the lower bound estimate).

with low socioeconomic status from BC_{II} by treatment status. The graph illustrates that the percentage of premature births is lower among the treatment group as compared to the control group. This indicates that culling tends to eliminate children with low birth endowment. After manually culling the control group, the distribution of gestation length shifts to the right, as depicted by Panel (c) of Figure 3. Comparing the two culled samples, we find that the distribution of gestation length in the treatment group now dominates the distribution in the culled control group (see Panel (d) of Figure 3). This provides first evidence for a negative scarring effect on birth outcomes. In the case of mothers with higher socioeconomic status we do not apply a sample correction, since we did not observe any significant culling effects for this group.

4.2 Children's health at birth

To evaluate the impact of Chernobyl on health at birth we summarize in Table 5 estimation results based on individual level data for the commonly used outcomes — gestation length, birth weight, and Apgar scores. Gestational length is classified as premature if it is below 37 weeks. Weight at birth is typically considered as low if it is below 2500 grams.[22] The Apgar score assesses after one, five, and ten minutes quickly and summarily the health of newborn babies based on five criteria (appearance, pulse, grimace, activity, and respiration) and ranges from zero ("good") to ten ("bad"). For each outcome we present estimated coefficients based on the observed sample (first row) and the corrected sample (second row). In the former cases the estimates capture the sum of culling and scarring effects, while in the latter case only scarring effects should remain.

Families with low socioeconomic status The signs of all estimates based on the observed sample suggest a strong positive culling effect that overcompensates any negative scarring effects. In the case of the outcome premature birth, the overall effect is statistically significant across specifications, where the point estimates marginally increase with the level of radioactive exposure. Children born alive are estimated to be about 3 percentage points less likely to be a preterm birth. Given an average incidence of preterm births of about 5.5 percent, the estimated effect is substantial and supports the notion that the live births are a selected group of healthier newborns. This is in line with our estimation results on prenatal culling discussed above.

The estimation results after our sample correction reveal statistically significant scarring effects. In the case of premature births this is *de facto* a mechanical result, since we have manually 'culled' the lower tail of the gestational age distribution. The estimated scarring effects amount to about plus 3 percentage points. However, also in the case of the other outcomes, we observe negative scarring effects. In the case of birth weight, the

[22] Both a premature gestation length and a low birth weight are related to higher likelihood of infant mortality, but may also have long lasting effects on health, education, and labor market outcomes (see, for instance, Black *et al.*, 2007).

estimated effects are significant and suggest an increased likelihood of low birth weight between 2.3 and 3.1 percentage points, depending on the level of radioactive exposure. The estimated scarring effects for the Apgar score are, as expected, negative but remain statistically insignificant.

As further indicator for health at birth we use the duration of maternity leave. The statutory maternity leave is eight weeks before the delivery and usually eight weeks after the delivery. Under certain conditions this duration may be extended; the sample average of the before and after spell are approximately nine weeks. If a premature birth reduces the pre-birth spell, the post-birth spell is extended such that the total maternity leave duration adds up to sixteen weeks. Moreover, the post-birth spell can be extended if health complications arise (i. e. the health of the mother or child would be endangered). Thus, an extended post-birth spell may reflect a mechanical effect due to low gestational age and/or post-birth health conditions. In line with the result above we observe that the surviving treated population has shorter post-birth spells; these children are less likely to be premature births and/or of better health. Again, after we apply our sample correction method these effects vanish.[23]

Families with higher socioeconomic status In the case of children from mothers with higher socioeconomic status, we did not apply the sample correction method, since we found very little evidence for prenatal culling above. The analysis of health at birth (consequently based on the observed samples), however, suggest partly evidence for positive culling effects. While estimated effects on the likelihood of premature birth and the Apgar scores are insignificant and essentially zero, we find a reduced likelihood of low birth weight and a small negative effect on the post-birth maternity leave spell.

Exposure at higher gestational age In the case of children from BC_I we find basically no evidence of radioactive exposure on health at birth (see Table C.1.3 in Web Appendix C.1). This applies to children irrespective of their socioeconomic backgrounds and is in line with our results on prenatal culling. The estimated coefficients for premature birth, low birth weight and Apgar scores are all statistically insignificant and essentially zero. The only exception are some negative effects on the Apgar scores of treated children from higher socioeconomic backgrounds.

4.2.1 Reconciliation with the no health effects result by AEP

There are at least three potential explanations why we find health effects of the Chernobyl accident (in terms of culling and scarring), while AEP do not identify any effects in their Swedish data. First, the level of ^{137}Cs ground deposition the average inhabitant was exposed to was considerably higher in Austria as compared to Sweden. AEP report a mean level of contamination of about 6 kBq/m^2 for Sweden and a mean level of about 44

[23] We do not the use the pre-birth spell duration, since the interpretation of the results is complicated — the mechanical effect and the health effect have opposite signs.

kBq/m^2 in areas with the highest exposure. In contrast, the average Austrian was exposed to a ^{137}Cs level of about 20 kBq/m^2, and the mean level of contamination in areas with the highest exposure ranges from 49 to 59 kBq/m^2 depending on our treatment group.[24]

Second, a comparison of the infant mortality rates suggests that the average Austrian child (unborn or newborn) had a significantly lower level of birth endowment at that time.[25] That means, the negative early shock should have more detrimental effects for the average Austrian child as compared to the average Swedish child.

Third, AEP exclude the birth cohort with a gestational age below eight weeks. It is possible that the prenatal culling is especially driven by this cohort who was exposed to radiation at a very early stage. For comparison, we present results based on an empirical model in the spirit of AEP in Table 6. We estimate two specifications: In the first one, the treatment group consists of children who have been in utero between 2 and 6 months (about 8 to 25 weeks) at the time of the accident. This specification uses the same definition of treatment status regarding timing as AEP. In the second one, we also include children who have been in utero between 0 and 2 months (about 0 to 7 weeks) at the time of the accident. Results based on the first specification are qualitatively similar to those found by AEP. There is no significant effect (neither positive nor negative) on health outcomes measured at birth. In line with that, there is no evidence for a distortion of the sex ratio, and in terms of live births only one out of three estimated coefficients is significant. In sum, we do not find much evidence for prenatal culling.

In contrast, based on the second specification (i. e. for children with a gestational age between 0 and 6 months), we do find evidence for prenatal culling in terms of a significantly negative effect on live births and the probability of a male birth. However, the estimated effects are weaker as compared to those for BC_{II} which includes only children at gestational age between 0 and 3 months (see Table 3 and Table 5). Moreover, we do not find any positive effects on health outcomes at birth — which would indicate prenatal culling — for children who have been in utero between 0 and 6 months at the time of the accident. Therefore, it seems that prenatal culling is actually driven by the birth cohort with a gestational age between 0 and 3 months.[26]

[24] According to UNSCEAR (2000) Austria and Sweden rank among those countries with the highest contamination levels in Western Europe. However, the relative size of the contaminated area was 10.3 percent in Austria as compared to 2.7 percent in Sweden (Source: Own calculations based on UNSCEAR (2000, Table 5, p. 520)). Moreover, the population density is substantially higher in Austria (92 inhabitants per km^2) than in Sweden (19 inhabitants per km^2). Therefore, the average Austrian was exposed to a higher ^{137}Cs level than the average Swede.

[25] The average infant mortality rate based on annual figures from the period from 1980 through 1985 was 12.2 in Austria and only 6.8 in Sweden (Source: Own calculations based on data from the *The World Bank*). A higher infant mortality rate reflects (among others) a low birth weight and a short gestational age among live births.

[26] In line with our analysis AEP report in an earlier version (Almond *et al.*, 2007) a negative effect of Chernobyl on cohort size and the probability of a male birth for children with a gestational age between 0 and 7 weeks.

4.3 Parental response

In this section we estimate the parental response to their child's early-life shock. We do not impose any assumption on whether the parents became aware of that their child was prenatally exposed to high levels of radioactive fallout. Our implicit assumption is that parents observe physiological or cognitive deficiencies of their child and might react to these.

To quantify parental response behavior we examine families' post-treatment fertility and maternal labor market outcomes.[27] We presume that both, a lower family size and a reduced maternal labor supply, enable more (time) investment in the child's human capital. Thus, we will associate either of these parental response behaviors with compensating investment in the treated child. In contrast, an increased family size and higher maternal engagement on the labor market will be interpreted as reinforcing investment. Table 8 summarizes the estimations results on post-treatment fertility. Each entry represents the results from a separate regression, where the dependent variable is equal to the number of children born to BC_{II} mothers in the respective year after treatment. Given that the mothers in our sample are between 40 and 63 years of age twenty years after the treatment, the estimated coefficient in the last row can be interpreted as the effect on completed fertility. Table 9 summarizes the estimations results on maternal labor market participation. The dependent variable is equal to one if the mother is in the labor force in the respective year after birth. Each entry represents the coefficient for exposed BC_{II} mothers interacted with years since the birth of the child. For both outcomes, we calculated in the case of families with low socioeconomic status estimates based on the observed samples and after applying our sample correction method. Since both estimations provide very comparable estimates, culling effects seem to be of second order importance in the case of long-term outcomes. Therefore, we focus on the results for the corrected sample (see Table 8 and Table 9) and provide the estimation output for the observed sample in Web Appendix C.2 (see Table C.2.1 and Table C.2.2).

Families with low socioeconomic status Exposed families with low socioeconomic status have significantly less children at any point in time after treatment (see Table 8). The size of the effect increases in absolute terms with the level of radioactive exposure; and over time. Twenty years after the birth of the pivotal child the effect amounts to minus 0.11 to minus 0.18 children. This is equivalent to a reduction in completed family size of about 15 to 24 percent. A closer inspection of how the estimated effects evolve over time (i. e. first differences in the estimates) reveals that the reduction is predominantly due to

[27] Due to a imperfect match between administrative data sources we lose 4 percent of the sample (compared to the analysis of children's health at birth) for our analysis of maternal labor market outcomes. We do not find a significant relationship between in utero exposure to radiation and a binary variable that indicates whether we observe maternal labor market outcomes. Moreover, the results on children's health at birth do not change when we exclude children with missing information on maternal labor market outcomes. For our analysis of post-treatment fertility we can use the full sample.

less births in the second, fifth, eight and eleventh year after treatment.

Starting from about four years after birth of the pivotal child exposed women are more likely to be active on the labor market (see Table 9). The estimated effects are not statistically significant for each single year. There is a pattern with a peak in the fifth and twelfth year after treatment with an estimated effect of about plus eight percentage points. We suggest *not* to interpret this labor market response as reinforcing investment, but as a direct consequence of the reduced family size. This interpretation is supported by the timing of these two responses. First, the initial and most pronounced fertility reaction (in the second year) predates the labor market adjustment. Second, the pattern of the labor market response (i. e. the peaks in fifth and twelfth year) coincides with the timing of the subsequent peaks in the fertility response.

Families with higher socioeconomic status In the case of exposed mothers with higher socioeconomic status we do not observe any effects on fertility (see Table 8). The estimates are all statistically not significantly different from zero and the coefficients are quite close to zero. That means, there is neither evidence for any impact in terms of timing of further births nor regarding completed family size.

In contrast, we find evidence that exposed mothers reduce their labor force participation temporarily (see Table 9). The timing of this effect is especially intriguing, since it coincides with the pivotal child's enrollment in primary school. The effect emerges in the seventh year, peaks in the eight year, and dissipates over time. After the tenth year the estimates are basically all zero. At the peak the effect is between minus 3.8 and minus 6.6 percentage points, depending on the level of radioactive exposure. This pattern is consistent with reduced cognitive abilities of exposed children as put forward by AEP. While it is not observable to us, when treated parents realize that their children have cognitive problems, a drastic intervention during primary school (enrollment) seems plausible. Due to the specific feature of *early tracking* in the Austrian education system, grades in primary school have already far-reaching consequences for later educational career.[28] Therefore, involved parents will take different measures to solve any learning differences at this stage. Given the basic content of the curriculum, professional tutoring is less uncommon for pupils of this age and typically parents study with their children after school to overcome any learning difficulties.

To summarize the main results of these analyses, we find for both socioeconomic groups statistically significant evidence for compensatory investment in the treated child. Families with low socioeconomic status reduce their fertility (trading child quality for quantity), while families with higher socioeconomic status respond with reduced maternal labor supply. Both effects indicate that affected children need more attention, and parents

[28] As we will discuss in more detail below, in Austria students are allocated already in grade five (i. e. at the age of 10) to two different educational tracks. Allocation to the *low track* or the *high track* are based on grades in third and fourth grade. Only graduates from the *high track* have access to university education.

adapt their behavior, such that they can devote more time to their children and make necessary compensating investment.[29]

The previous design-based literature on the parental response to child endowments is rather scarce (see footnote 1), at least with respect to evidence for developed countries. Bharadwaj *et al.* (forthcoming) exploit a discontinuity in a neonatal health care treatment for children with very low birth weight and find no evidence for parental response behavior in their Norwegian (and Chilean) data. In another context, Frijters *et al.* (2009) use left-handedness to instrument for poor early child development and find a negative effect on maternal labor supply, suggesting that parents make compensating investments.

Exposure at higher gestational age For families from BC_I we find a similar pattern for the fertility response behavior (see Table C.1.4). We see again a reduction by families with low socioeconomic status and no significant reaction by those with higher socioeconomic status. In the former group the estimated effects are now smaller (as compared to BC_I). They are about half in size and only sporadically individually significant from zero. Twenty years after the birth of the pivotal child the effect is between minus 0.07 and minus 0.11 children (significant at the 10 percent level). This is equivalent to a reduction in completed family size of about 9 to 15 percent. The effect on maternal labor force participation (see Table C.1.5) are less clear.

For families with low socioeconomic status we do not observe any significant effects on maternal labor force participation. This finding is consistent with the substantially weaker fertility response compared to BC_{II} families. In the case of families with higher socioeconomic status we observe a positive effect on maternal labor force participation in several years after child birth. The effect is between 1.6 and 5.1 percentage points and of varying significance, depending on the level of radioactive exposure. There are two possible explanations for this finding: First, it may reflect a reinforcing parental behavior in response to a negative scarring effect. We found some evidence that these children had a lower Apgar score at birth (see Table C.1.3). Alternatively, given that we find some evidence for postnatal culling in this group (see Table C.1.2), these children may be positively selected. A higher labor force participation may then simply reflect the fact that these children have higher cognitive endowments and less parental tutoring is needed. Given that we also do not find much evidence for scarring effects for these children in our analysis of labor market outcomes (see below), we consider the second explanation as more plausible.

4.3.1 Non-radiation effects

The local level of ground deposition was not known at the time of the accident. Therefore, parents (or even potential parents) in exposed and non-exposed communities may have

[29] We have also examined the length of parental leave spells; no difference between exposed and non-exposed families was found.

been stressed and anxious in the aftermath of the accident. This may have changed their behavior immediately after the accident and caused what we termed *non-radiation effects* with potential scarring and culling consequences for children. In case of live births and fetal death only culling effects are possible.

Most likely, women who were already aware about their pregnancy were extremely stressed and anxious. This stress *per se* may had detrimental effects on the embryo or fetus, or even lead to an miscarriage.[30] A very direct culling effect is given by an adaption in abortion behavior; pregnant women may have decided to have an induced abortion.[31] Less drastically, expecting mothers could have tried to reduce exposure to radiation. While it is general unlikely that such an avoidance behavior (i.e. change of diet) is effective in reducing exposure to radiation, it could have nevertheless affected the child in some way.

The scope of early parental response behavior differs across birth cohorts. Parents of a child belonging to BC_I had the smallest scope for action. An induced abortion was not possible anymore, since the pregnancy has advanced past the first trimester (i. e. the legal time limit). Their unborn child could have been exposed to maternal stress or a modified maternal diet during the last two trimesters of pregnancy.[32] In comparison, parents of a child belonging to BC_{II} could have reacted quite drastically and have an abortion.

The parameters ρ_1 and ρ_2 give the sum of the estimated non-radiation effects. Table 7 summarizes the effects for prenatal culling and health at birth. In case of the prenatal culling outcomes the parameter ρ_1 gives the estimated non-radiation effects (for BC_I) that work through miscarriages. Among the mothers with higher socioeconomic status we find some evidence for an increased number of miscarriages. While the effect is not statistically significant at conventional levels in the case of live births, we find a skewed sex ratio. In line with the Trivers-Willard-Hypothesis there is significant negative effect on the likelihood of male birth. The effect is almost identical across different levels of exposure.[33] This does not affect the outcomes gestational age and birth weight at the critical margins (i. e. premature birth or low birth weight). That means, The parameter

[30]Experimental evidence on the negative effects of in utero exposure to maternal stress on offspring outcomes is only available in animal studies (Kaiser and Sachser, 2005). For humans a number of observational studies report a negative effect of maternal stress (measured by cortisol levels). Nepomnaschy *et al.* (2006) find evidence for increased fetal death. Aizer *et al.* (2009) reports negative effects on educational attainment, the probability of a severe chronic health condition and verbal IQ at age of seven. Similar results are obtained for birth weight by design based papers using earthquakes (Torche, 2011) and terrorist attacks (Camacho, 2008; Mansour and Rees, 2012). Currie and Rossin-Slater (2012), exploiting hurricane exposure, find some evidence for complications of labor and delivery but no effect on birth weight and gestation.

[31]In fact, there is some evidence indicating a temporary increase in the number of induced abortions in Greece, Italy and Sweden (see Web Appendix A).

[32]There is some evidence for a high responsiveness of birth weight to nutritional changes in the third trimester of pregnancy (Painter *et al.*, 2005).

[33]While in the case of radiation effects one would expect variation in the estimated effects according to the degree of exposure to radioactive fallout, in the case of non-radiation effects a uniform response across regions can be expected given that individuals have not been aware of the local level of ground deposition.

ρ_2 gives the estimated non-radiation effects (for BC_{II}) due to miscarriages and induced abortions. We do not find any significant effects. By imposing the assumption that the non-radiation effects on miscarriages are equal for BC_I and BC_{II}, it is possible to interpret the difference between ρ_1 and ρ_2 as the effect that is due to induced abortions.[34] We find statistical significant evidence that induced abortions have decreased the fetal death rate between 3.8 and 4.3 percentage points (depending on the level of radiation).

Among the mothers with low socioeconomic status we do not find any significant non-radiation effects. This suggest that early parental response behavior was only prevalent (or at least discernible) among families with higher socioeconomic status.

4.4 Children's human capital outcomes

To assess the long-term effects on exposed children we examine human capital outcomes between the age of 15 and 24.[35] Due to a widespread dual education system and low rates of university graduates, the vast majority of the Austrian population is already in the work force at this age (i. e. either as an apprentice or as an regular employee). Table 10 shows that this applies in particular for children from families with low socioeconomic status.

To motivate our estimation strategy we outline the average Austrian student's transition from school to work. This is driven by two distinguishing features of the Austrian education system: *early tracking* and the widespread dual education system. Students are allocated already in grade five (i. e. at the age of 10) to two different educational tracks. The lower secondary schools (*low track*) comprise grades 5 to 8, provide basic general education and prepare students for vocational education either within an intermediate vocational school or within the dual education system. The higher general schools (*high track*) comprise a first stage (grades 5 to 8) and a second stage (grades 9 to 12), provide advanced general education and conclude with an university entrance exam.[36]

Low track career path The majority of the students (about 72 percent) completes the *low track*. This share is higher among children from parents with low socioeconomic

[34] Given that miscarriages are generally far more common in the first trimester than in the second or third trimester, we interpret the abortion effect with caution.

[35] This analysis is based on a sub-sample (as compared to the analysis of children's health at birth). We are able to link only 75 percent of the cases in the *Austrian Birth Register* with the ASSD. Fortunately, whether we observe a child's human capital is not related to in utero exposure. Moreover, the results on children's health at birth are robust to the exclusion of those 25 percent of children.

[36] A further institutional detail of the Austrian education system impedes an analysis of test scores. Tests in either track are decentralized. That means, they are prepared and graded by the respective teacher. This rules out a meaningful comparison of test scores across schools. Test scores from the *Programme for International Student Assessment* (PISA) and *Trends in International Mathematics and Science Study* (TIMSS) would be comparable across time and space; however, neither study covers the treated birth cohort. The only feasible data are cognitive test scores collected by the Austrian military. (All male Austrian citizens are subject to compulsory military service and have to enlist and muster for different examination within one year after attaining their 17th birthday.) This data is, however, until now not available to researchers.

status (87 percent) than among those from parents with higher socioeconomic status (66 percent).[37] Approximately 82 percent of students from the low track enter the work force around 15 years of age; ideally via the dual education system or as an unskilled worker. The dual education system combines an apprenticeship in a firm and (vocational) education at a vocational school. Not all students who want to enter the dual education system, manage to find an employer. They either register unemployed or find a job as an unskilled worker. The remaining 18 percent continue with an intermediate vocational school and enter the workforce at around 17 years of age.

Higher track career path Only about 30 percent of all students complete the *high track*. Among children from low socioeconomic backgrounds this share is only 14 percent, while among children from higher socioeconomic backgrounds it amounts to 34 percent. Students from the high track enter the workforce either after graduation from a higher general school (at around 18 years of age), a higher vocational school (at around 19 years of age) or a university.

While our data derived from the ASSD does not include any information on educational attainment, it comprises detailed information on all workers in Austria on their labor market status in employment (including basic employer information), unemployment, and various other qualifications on a daily basis. In particular, we can distinguish between an apprenticeship training, different types of regular employment and unemployment (each measured on the first day of the quarter that includes their birthday). Table 11 summarizes our main estimation result, where the dependent variable is equal to one if the child is in the labor force at a certain age.[38] Each entry represents the coefficient for exposed children BC_{II} interacted with their age. In Table 12 we further estimate the impact on the likelihood of being an apprentice at the age 16, 17 and 18, as well as, the effect on the overall labor income earned between the age of 15 through 23.

Families with low socioeconomic status For this group we have again compared the estimates based on the observed and the corrected samples. As in the case of parental response behavior, the sample correction has little impact. That means, there are no culling effects present in the long run. They seem to have vanished since birth. In fact, in the case of children's labor market outcomes the point estimates are almost identical. Therefore we report the estimates based on the corrected sample and provide the estimation output for the observed sample in Web Appendix C.2 (see Table C.1.6 and Table C.1.7).

To put it simply, due to a high intergenerational educational persistence in Austria (OECD, 2010), there are two realistic successful career paths for children from low so-

[37] This figures are own calculations based on retrospective data from Knittler (2011) and refer to the sum of graduates and drop-outs from the low track.

[38] Labor force participation includes any form of dependent employment (blue-collar, white-collar, civil servant), self-employment, employment as farmer, apprenticeship training, freelance service contract ('Freier Dienstvertrag/Werkvertrag'), subsidized employment, sick leave and unemployment except marginal employment.

cioeconomic backgrounds. They either start an apprenticeship training at the age of 15, graduate and are employed from there on. Alternatively, they graduate from an intermediate vocational school and start working at the age of 17. Those who do not graduate and become (employed or unemployed) unskilled workers are the low performers.

Our estimation results highlight two robust effects. First, treated children are less likely to be an apprentice. The effect is strongest at the age of 17 and amounts to approximately minus 8 percentage points (see Table 12). Second, exposed children are less likely to be employed throughout the whole time period under consideration (see Table 11). While not each coefficient is individually significant, they are consistently negative and jointly significant. The effect is on average minus 8 percentage points. In sum, these two results suggest that treated children are less likely to finish an apprenticeship, and (due to a lack of vocational career options) they are less likely to be employed thereafter.[39] That means, exposed children from low socioeconomic backgrounds have worse educational and labor market outcomes and have (at least until the age of 24 years) accumulated less human capital. We conclude that prenatal exposure to radiation has for this group — despite compensating behavior of their parents — substantial long-term scarring effects. Based on wage regressions the estimated loss in annual before-tax income amounts to 8,851 Euro or 53.7 percent. A quantification of the corresponding loss in the counterfactual situation where parents of exposed children would have not adjusted family size (as a response to the treatment) is hard, since this compensating investment is endogenous.

Families with higher socioeconomic status For children from higher socioeconomic backgrounds we find little evidence for effects on their labor market outcomes. At the age of sixteen they are somewhat more likely to be in the labor force (about four to five percentage points). A comparison between Table 11 and 12 shows that this effect is driven by an increased likelihood of being an apprentice. Since this is a below-average career path for this group, the effect should be interpreted as a negative scarring effect. At higher ages the labor market participation is not statistically significantly different between exposed and non-exposed children; the same holds for the likelihood of being an apprentice and the total earned labor income. The majority of the point estimates (especially those at higher ages) are also quite close to zero. This suggests, even if some negative scarring effects are present in adolescence, they seem to vanish over time.

[39] Strictly speaking, our estimation results are also consistent with an interpretation where exposed children are more likely to graduate from the high track and proceed to college, and are for this reason 8 percentage points less likely to be employed. Theoretically, this could be the result of very effective parental response behavior; which overcompensates the effect of the early-life shock. This interpretation, however, seems far fetched. Only 14 percent of children from low socioeconomic backgrounds complete the high track — this would imply an effect of almost 60 percent. Still, to provide supportive evidence for our interpretation, we have estimated the effect on so-called *marginal employment*. This type of employment contract is for jobs with a low number of working hours, low pay (up to just over USD 284 per month in 2002) and covers only accident insurance. This type of employment is very common among college students who work while enrolled. We do not find any significant effects on the likelihood of marginal employment.

These results conform with existing evidence on the impact of in utero exposure to radioactivity on long-term human capital outcomes. AEP find that Swedish children in low-educated families (who were prenatally exposed to radioactive fallout of the Chernobyl accident) had significantly lower grades in compulsory school at the age of 16. In contrast, for children in highly-educated families they identify no comparable effect. Black *et al.* (2013) show with their Norwegian data that in utero exposure to radioactive fallout caused by nuclear weapon testing reduced IQ scores, educational attainment and earnings. Some of these effects are more pronounced for individuals from low socioeconomic backgrounds.

The social gradient in the long-term effects on treated children can be explained by a comparable more effective compensating investment made by families with higher socioeconomic status. It remains unclear at which stage and how this investment took place. It seems plausible that families from low socioeconomic backgrounds are more restricted in their compensatory investment; for instance, binding financial constraints may not allow an adjustment of maternal labor supply or changes along other none-observable dimensions (such as private tutoring or the social job-finding networks).

Exposure at higher gestational age For children from BC_I we do not find robust evidence for any long-term effects on human capital outcomes (see Tables C.1.6 and C.1.7). The vast majority of the estimates have a negative sign (this applies to children from all socioeconomic backgrounds), however, the estimates are mostly statistically insignificant. In sum, these results corroborate the conjecture that prenatal exposure to radiation is less critical at a higher gestational age.

5 Conclusions

The literature on the long-term effects of early childhood conditions on human capital accumulation has devoted little attention to parental response behavior so far. In this paper we study the case of prenatal exposure of the Austrian 1986 cohort to radioactive fallout from the Chernobyl accident and examine how parents of exposed children respond to the early-life shock. Identification is guaranteed by random geographic variation in the exposure to radioactive fallout due to differences in precipitation at the time of the accident. Based on different administrative data sources we find robust evidence for compensating investment that differs in type and effectiveness across families' socioeconomic backgrounds. Families with low socioeconomic status reduce their family size. Exposed children from these families still have worse outcomes in young adulthood. Families with higher socioeconomic status reduce temporarily maternal labor supply. In their case we do not find any detrimental long-term effects for exposed children.

These results should be of interest to scholars and policy-makers alike. They demonstrate that simple empirical estimates of the impact of exogenous early-life shocks on long-term outcomes can only be interpreted as reduced form estimates, since parental

compensatory investment behavior is an empirically relevant phenomenon. The most straightforward policy implication of our result is another strong argument for providing disadvantaged families with the necessary economic and social resources that allow early childhood investment. It is widely documented that (i) children from low socioeconomic backgrounds typically grow up in less favorable environments and (ii) there is also some evidence that early conditions matter more for children from this group. Our results shed light on the underlying mechanism. They suggest that all parents — irrespective of their socioeconomic backgrounds — adjust their behavior to invest in their children according to their specific needs. However, parental response behavior of families with higher socioeconomic status seems comparable more effective. It seems plausible that families from low socioeconomic backgrounds are more restricted in their compensatory investment along pecuniary and non-pecuniary dimensions.

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6 Tables and figures

Figure 1: Average Caesium-137 ground deposition in Austria on May 1, 1986

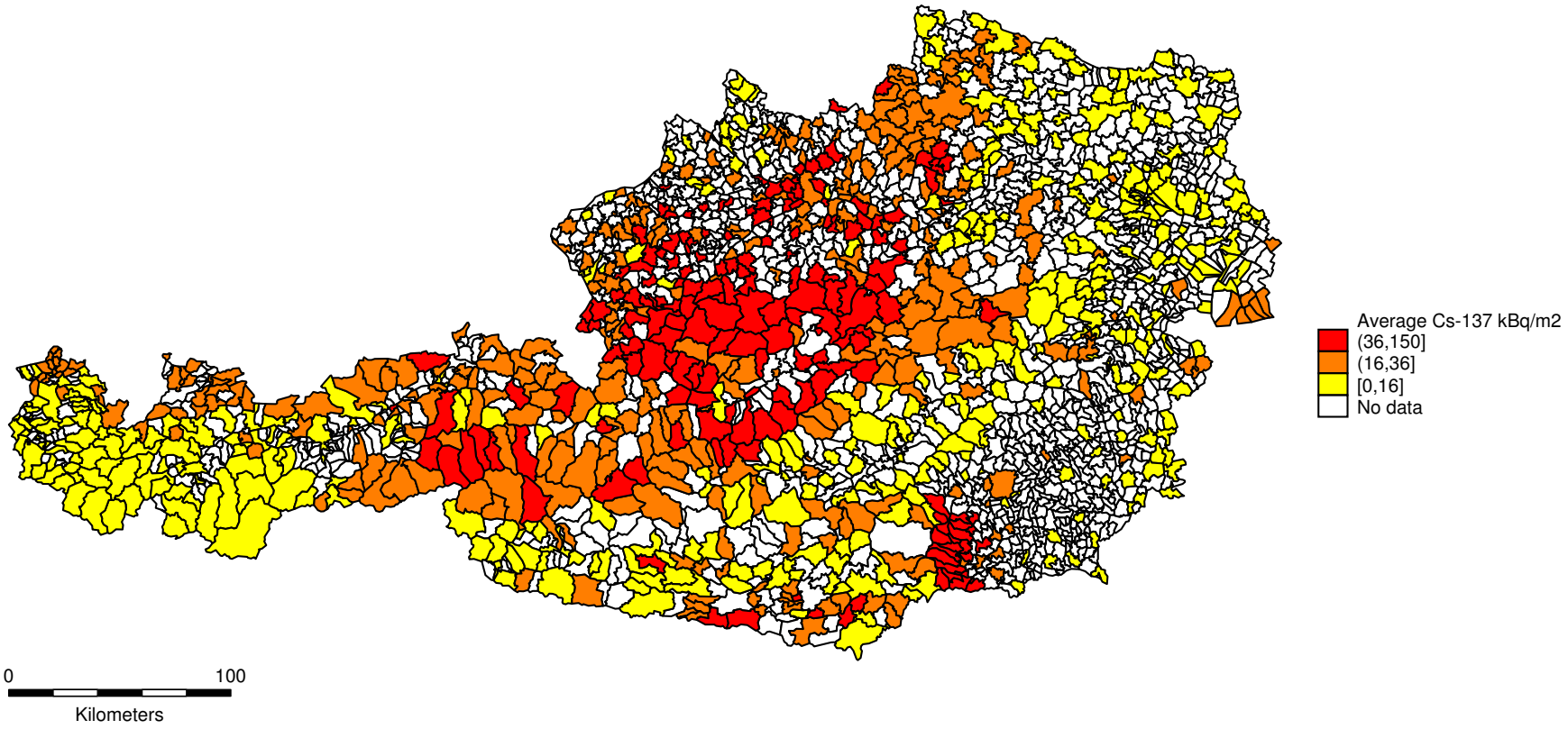


Table 1: Definition of treatment and control groups

Group	Acronym	Average level of ^{137}Cs ground deposition (in kBq/m^2)	No. of communities	Mean ^{137}Cs	Std.Dev. ^{137}Cs	No. of children
Control group	C	less than 17	427	8.1	(4.6)	72,465
Treatment group 1	$T1$	37 ore more	175	49.1	(12.5)	22,455
Treatment group 2	$T2$	42 ore more	130	54.4	(12.3)	14,783
Treatment group 3	$T3$	47 ore more	93	59.1	(12.5)	9,967
Excluded[a]		between 17 and 36	322	27.3	(5.5)	36,685
		between 17 and 41	367	29.3	(6.6)	44,357
		between 17 and 46	404	30.8	(7.8)	49,173

[a] The cutoff-value, the number of communities and the population-weighted mean of ^{137}Cs ground deposition depend on the respective treatment group.

Figure 2: Birth cohorts included in the estimation samples

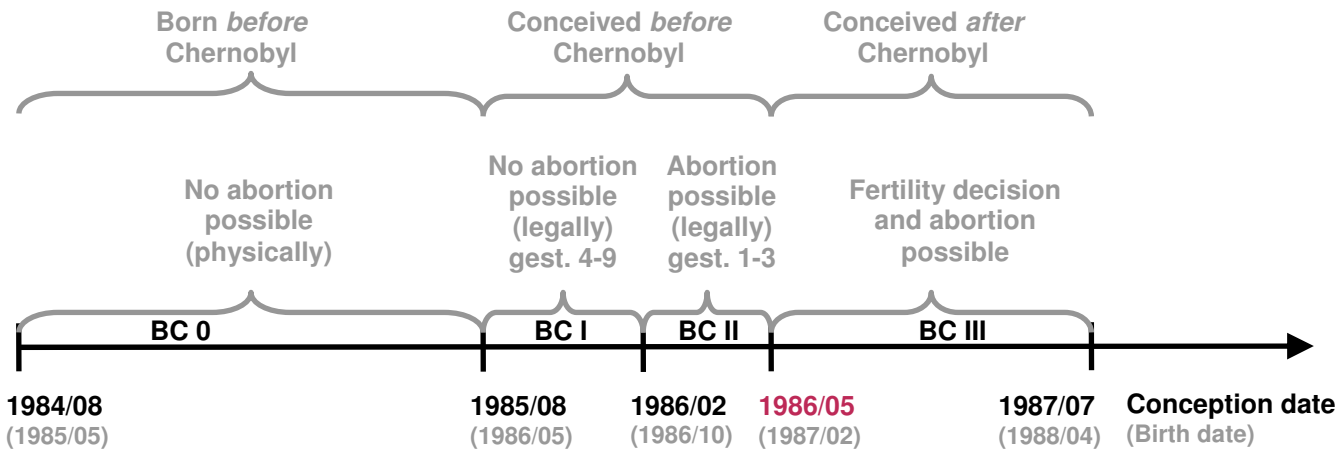


Table 2: Outcome variables

Outcome	**Level of available data**	**Measurements**	**Data source**
Live births	Community	Absolute number per 1,000 female inhabitants aged 15-39 in 1981 in the respective education group	*Austrian Birth Register*
Stillbirth	Individual	Binary variable	*Austrian Birth Register*
Spontaneous abortion	Not observable	Proxied by sex-ratio (binary variable that indicates whether child's sex is male)	*Austrian Birth Register*
Health at birth	Individual	Gestation length, weight, Apgar scores	*Austrian Birth Register*
Maternity leave	Individual	length in days	*Austrian Social Security Database*
Infant mortality	Individual	Binary variables that indicate whether child is still alive after 24 hours/7 days/1 month/1 year	*Austrian Birth & Death Register*
Parental leave	Individual	Length in days, take-up	*Austrian Social Security Database*
Labor market outcomes	Individual	For mother and child: apprenticeship training, employment, wages	*Austrian Social Security Database*
Post-treatment fertility	Individual	Number of children	*Austrian Birth Register/ Austrian Social Security Database*

Table 3: Radiation effects on prenatal culling

	Low SES				Higher SES			
	Mean	37 kBq	42 kBq	47 kBq	Mean	37 kBq	42 kBq	47 kBq
Live birth								
Live birth rate[a]	3.031	−0.086**	−0.112**	−0.106*	8.918	−0.061	0.054	−0.025
		(0.043)	(0.049)	(0.058)		(0.126)	(0.153)	(0.178)
Stillbirth								
Prob(Stillbirth)[b]	0.005	0.004	0.009	0.009	0.004	−0.001	0.000	−0.002
		(0.005)	(0.007)	(0.008)		(0.002)	(0.002)	(0.002)
Fetal death								
Proxy: *Prob*(male)[c]	0.510	−0.043	−0.075*	−0.088**	0.514	−0.014	−0.005	−0.016
		(0.036)	(0.039)	(0.040)		(0.019)	(0.022)	(0.027)

This table summarizes estimation results based on community-level data (first row) and individual-level data (second and third row) from the *Austrian Birth Register* and the *Austrian Death Register* covering births conceived between 08/1984 and 07/1987. Each entry represents a separate regression, where the dependent variable is indicated in the first column, and shows the estimated coefficient for treated units from the BC_{II}. This cohort was conceived between 02/1986 and 04/1986 and was between 0 and 3 months post conception at the time of the accident. Each specification controls for community, conception-year, and conception-month fixed-effects. Method of estimation is a least squares. Robust standard errors (clustered at the community level) are shown in parentheses. *, ** and *** indicate statistical significance at the 10-percent level, 5-percent level, and 1-percent. Families with a low socioeconomic status (SES) have mothers with compulsory schooling or less. Families with a higher SES have mothers with any degree higher than compulsory schooling. Means refer to the 37kBq-cutoff sample. [a] The dependent variable is equal to the number of live births per 1,000 female inhabitants aged between 15 and 39 years of age in 1981 (in the respective education group) divided by the respective sample mean. [b] The dependent variable is equal to one if the child is a stillbirth and zero if the child is a live birth. [c] The dependent variable is equal to one if the child is male, and zero otherwise.

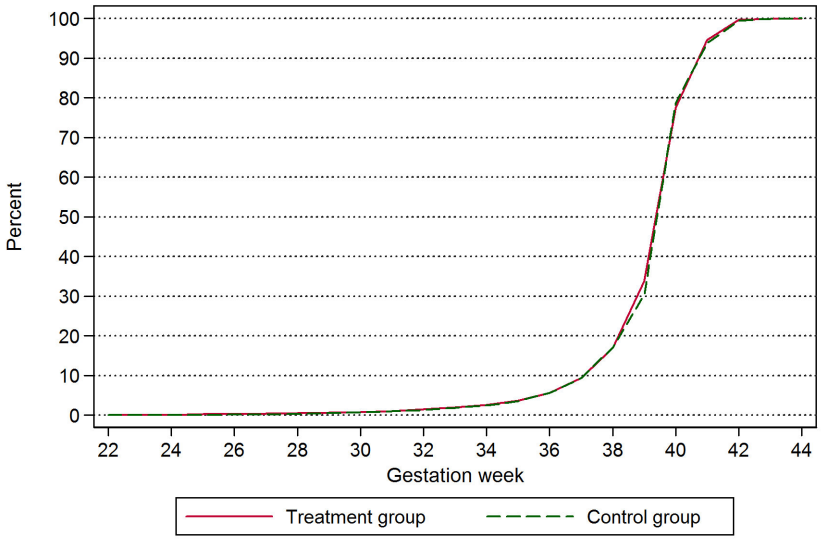
Table 4: Radiation effects for postnatal culling

	Low SES				Higher SES			
	Mean	37 kBq	42 kBq	47 kBq	Mean	37 kBq	42 kBq	47 kBq
Alive after 24 hours	0.993	−0.008	−0.013	−0.017	0.995	0.002	0.001	0.003
		(0.007)	(0.009)	(0.011)		(0.002)	(0.003)	(0.002)
Alive after 7 days	0.990	−0.007	−0.013	−0.011	0.992	0.002	0.003	0.005**
		(0.008)	(0.010)	(0.011)		(0.003)	(0.003)	(0.002)
Alive after 1 month	0.988	−0.006	−0.011	−0.010	0.991	0.002	0.004	0.006**
		(0.008)	(0.010)	(0.011)		(0.003)	(0.003)	(0.002)
Alive after 1 year	0.983	0.004	−0.004	−0.003	0.988	0.001	0.005	0.006
		(0.009)	(0.011)	(0.013)		(0.004)	(0.004)	(0.004)

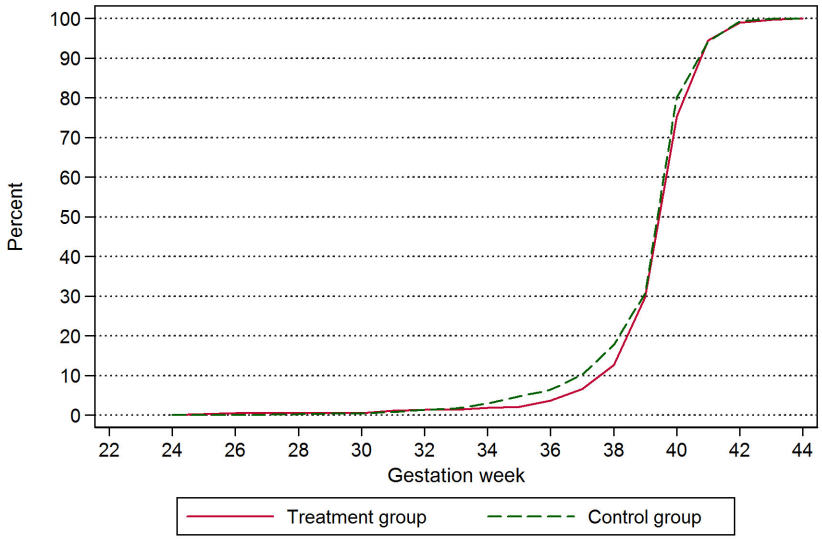
This table summarizes estimation results based on individual-level data from the *Austrian Birth Register* and the *Austrian Death Register* covering births conceived between 08/1984 and07/1987. The dependent variable is equal to one if the child is still alive after the respective time period after birth. Each entry represents a separate regression, where the dependent variable is indicated in the first column, and shows the estimated coefficient for treated units from the BC_{II}. This cohort was conceived between 02/1986 and 04/1986 and was between 0 and 3 months post conception at the time of the accident. Each specification controls for community, conception-year, and conception-month fixed-effects. Method of estimation is a least squares. Robust standard errors (clustered at the community level) are shown in parentheses. *, ** and *** indicate statistical significance at the 10-percent level, 5-percent level, and 1-percent. Families with a low socioeconomic status (SES) have mothers with compulsory schooling or less. Families with a higher SES have mothers with any degree higher than compulsory schooling. Means refer to the 37kBq-cutoff sample.

Figure 3: Cumulative distribution of gestation length for mothers with low socioeconomic backgrounds

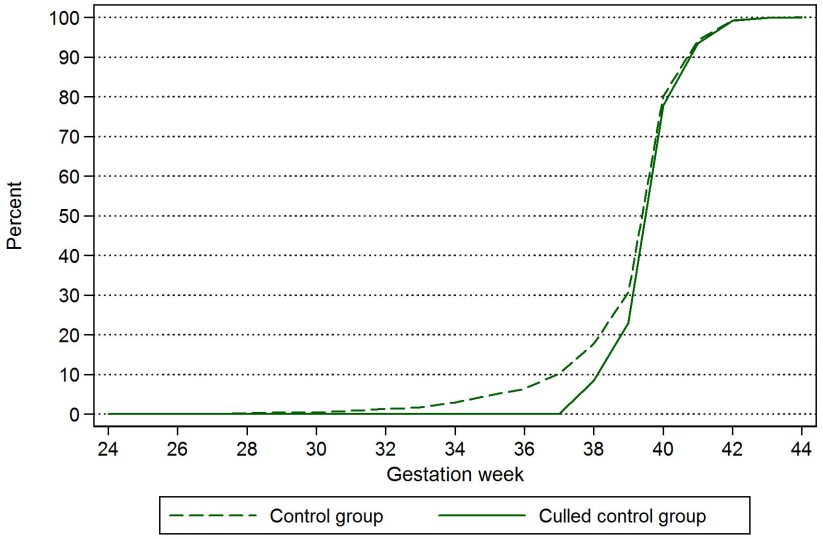
(a) BC 0 — treatment group vs. control group



(b) BC II — treatment group vs. control group



(c) BC II — control group vs. culled control group



(d) BC II — treatment group vs. culled control group

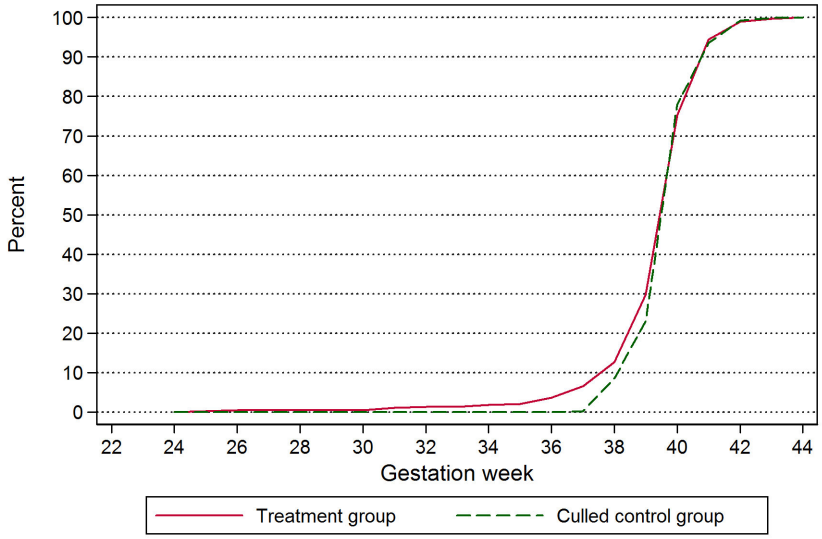


Table 5: Radiation effects (culling & scarring) on health at birth

	Low SES				Higher SES			
	Mean	37 kBq	42 kBq	47 kBq	Mean	37 kBq	42 kBq	47 kBq
Premature birth[a]								
Culling & scarring	0.056	−0.028**	−0.029**	−0.032**	0.047	−0.008	−0.009	−0.011
		(0.011)	(0.013)	(0.014)		(0.008)	(0.009)	(0.010)
Scarring	0.053	0.035***	0.035***	0.032***				
		(0.009)	(0.010)	(0.012)				
Low birth weight[b]								
Culling & scarring	0.066	−0.014	−0.012	−0.020	0.054	−0.025***	−0.032***	−0.028***
		(0.013)	(0.015)	(0.017)		(0.007)	(0.008)	(0.009)
Scarring	0.064	0.029**	0.031**	0.023				
		(0.012)	(0.014)	(0.016)				
Apgar score[c]								
Culling & scarring	9.870	0.025	0.005	−0.018	9.897	0.020	0.024	0.021
		(0.047)	(0.053)	(0.069)		(0.018)	(0.022)	(0.028)
Scarring	9.873	−0.023	−0.043	−0.066				
		(0.044)	(0.051)	(0.067)				
Maternity leave (post)[d]								
Culling & scarring	62.79	−1.792**	−1.525	−1.555	62.59	−0.804**	−0.805	−0.920*
		(0.859)	(1.058)	(1.197)		(0.401)	(0.520)	(0.537)
Scarring	62.70	−0.244	−0.024	−0.003				
		(0.834)	(1.039)	(1.181)				

This table summarizes estimation results based on individual-level data from the *Austrian Birth Register* and the *Austrian Social Security Database* covering births conceived between 08/1984 and 07/1987. Each entry represents a separate regression, where the dependent variable is indicated in the first column, and shows the estimated coefficient for treated units from the BC_{II}. This cohort was conceived between 02/1986 and 04/1986 and was between 0 and 3 months post conception at the time of the accident. Each specification controls for community, conception-year, and conception-month fixed-effects. Method of estimation is a least squares. Robust standard errors (clustered at the community level) are shown in parentheses. *, ** and *** indicate statistical significance at the 10-percent level, 5-percent level, and 1-percent. Families with a low socioeconomic status (SES) have mothers with compulsory schooling or less. Families with a higher SES have mothers with any degree higher than compulsory schooling. Means refer to the 37kBq-cutoff sample. [a] The dependent variable is equal to one if the gestation period is below 37 weeks, and zero otherwise. [b] The dependent variable is equal to one if the birth weight is lower than 2,500 grams, and zero otherwise. [c] The dependent variable is equal to the Apgar score after ten minutes. [d] The dependent variable is equal to the number of days on maternity leave after birth of the pivotal child. Further control variable: binary indicator for multiple birth.

Table 6: Reconciliation with the no health effects result by AEP

	Low SES				Higher SES			
	Mean	37 kBq	42 kBq	47 kBq	Mean	37 kBq	42 kBq	47 kBq
Live birth[a]								
2-6 months post conception	3.031	−0.062	−0.108**	−0.106	8.918	−0.067	−0.060	−0.095
		(0.047)	(0.054)	(0.070)		(0.090)	(0.101)	(0.117)
0-6 months post conception	3.031	−0.070*	−0.103**	−0.101*	8.918	−0.073	−0.021	−0.069
		(0.036)	(0.042)	(0.053)		(0.077)	(0.090)	(0.102)
Fetal death: Prob(male)[b]								
2-6 months post conception	0.511	−0.014	−0.022	−0.043	0.513	0.007	0.012	0.003
		(0.022)	(0.027)	(0.033)		(0.014)	(0.017)	(0.022)
0-6 months post conception	0.511	−0.031	−0.051**	−0.063***	0.514	−0.000	0.003	−0.005
		(0.019)	(0.021)	(0.024)		(0.011)	(0.015)	(0.020)
Premature birth[c]								
2-6 months post conception	0.057	0.012	−0.001	0.003	0.047	0.003	−0.001	−0.005
		(0.012)	(0.012)	(0.014)		(0.006)	(0.007)	(0.008)
0-6 months post conception	0.057	−0.005	−0.015	−0.015	0.047	−0.002	−0.003	−0.009
		(0.009)	(0.009)	(0.011)		(0.005)	(0.006)	(0.007)
Low birth weight[d]								
2-6 months post conception	0.067	0.007	−0.004	−0.001	0.054	−0.002	−0.001	0.002
		(0.013)	(0.014)	(0.016)		(0.006)	(0.008)	(0.009)
0-6 months post conception	0.067	−0.002	−0.009	−0.014	0.054	−0.010*	−0.012*	−0.011
		(0.010)	(0.011)	(0.014)		(0.005)	(0.006)	(0.008)
Apgar score[e]								
2-6 months post conception	9.869	0.037	0.034	0.026	9.896	−0.011	−0.017	−0.014
		(0.027)	(0.031)	(0.035)		(0.016)	(0.020)	(0.025)
0-6 months post conception	9.869	0.035	0.032	0.015	9.897	0.002	−0.001	0.001
		(0.027)	(0.032)	(0.037)		(0.013)	(0.016)	(0.021)

This table summarizes estimation results based on individual-level data from the *Austrian Birth Register* and the *Austrian Social Security Database* covering births conceived between 08/1984 and 07/1987. Each entry represents a separate regression, where the dependent variable is indicated in the first column, and shows the estimated coefficient for the cohort between 2 and 6 months post conception at the time of the accident (*AEP cohort*) and the cohort between 0 and 6 months post conception. These cohorts was conceived between 11/1985 and 02/1986 (04/1986). (We drop conceptions one week before and after each cutoff date.) Each specification controls for community, conception-year, and conception-month fixed-effects. Method of estimation is a least squares. Robust standard errors (clustered at the community level) are shown in parentheses. *, ** and *** indicate statistical significance at the 10-percent level, 5-percent level, and 1-percent. Families with a low socioeconomic status (SES) have mothers with compulsory schooling or less. Families with a higher SES have mothers with any degree higher than compulsory schooling. Means refer to the 37kBq-cutoff sample. [a] The dependent variable is equal to the number of live births per 1,000 female inhabitants aged between 15 and 39 years of age in 1981 (in the respective education group) divided by the respective sample mean. [b] The dependent variable is equal to one if the child is male, and zero otherwise. [c] The dependent variable is equal to one if the gestation period is below 37 weeks, and zero otherwise. [d] The dependent variable is equal to one if the birth weight is lower than 2,500 grams, and zero otherwise. [e] The dependent variable is equal to the Apgar score after ten minutes.

Table 7: Non-radiation effects (culling & scarring) on prenatal culling and health at birth

	Low SES				Higher SES			
	Mean	37 kBq	42 kBq	47 kBq	Mean	37 kBq	42 kBq	47 kBq
Live birth[a]								
BC I	3.031	0.034	0.035	0.036	8.918	−0.102	−0.085	−0.110
		(0.033)	(0.034)	(0.035)		(0.077)	(0.079)	(0.081)
BC II		0.035	0.035	0.034		0.058	0.080	0.031
		(0.058)	(0.060)	(0.062)		(0.130)	(0.135)	(0.138)
Fetal death: Prob(male)[b]								
BC I	0.510	0.006	0.006	0.017	0.514	−0.022*	−0.026**	−0.024*
		(0.018)	(0.018)	(0.018)		(0.012)	(0.012)	(0.012)
BC II		0.031	0.035	0.049		0.022	0.012	0.015
		(0.034)	(0.035)	(0.036)		(0.024)	(0.024)	(0.024)
Premature birth[c]								
BC I	0.056	0.002	0.004	0.004	0.047	−0.006	−0.006	−0.005
		(0.010)	(0.011)	(0.011)		(0.007)	(0.007)	(0.007)
BC II		0.001	−0.001	0.002		−0.009	−0.007	−0.004
		(0.016)	(0.017)	(0.017)		(0.009)	(0.009)	(0.009)
Low birth weight[d]								
BC I	0.066	−0.005	−0.004	−0.002	0.054	0.005	0.006	0.005
		(0.011)	(0.011)	(0.012)		(0.006)	(0.006)	(0.006)
BC II		−0.019	−0.027	−0.020		−0.002	0.001	0.001
		(0.018)	(0.019)	(0.020)		(0.009)	(0.009)	(0.009)
Apgar score[e]								
BC I	9.870	0.008	0.002	−0.002	9.897	−0.002	−0.004	0.000
		(0.023)	(0.024)	(0.024)		(0.013)	(0.013)	(0.014)
BC II		−0.009	−0.003	−0.015		0.015	0.008	0.011
		(0.039)	(0.040)	(0.040)		(0.021)	(0.021)	(0.022)

This table summarizes estimation results based on individual-level data from the *Austrian Birth Register* and the *Austrian Social Security Database* covering births conceived between 08/1984 and 07/1987. The dependent variable is indicated in boldface; the estimated non-radiation effects on treated units from birth cohorts I and II are displayed below. BC_I was conceived between 08/1985 and 01/1986 and was between 4 and 9 months post conception at the time of the accident. BC_{II} was conceived between 02/1986 and 04/1986 and was between 0 and 3 months post conception at the time of the accident. Each specification controls for community, conception-year, and conception-month fixed-effects. Method of estimation is a least squares. Robust standard errors (clustered at the community level) are shown in parentheses. *, ** and *** indicate statistical significance at the 10-percent level, 5-percent level, and 1-percent. Families with a low socioeconomic status (SES) have mothers with compulsory schooling or less. Families with a higher SES have mothers with any degree higher than compulsory schooling. Means refer to the 37kBq-cutoff sample. [a] The dependent variable is equal to the number of live births per 1,000 female inhabitants aged between 15 and 39 years of age in 1981 (in the respective education group) divided by the respective sample mean. [b] The dependent variable is equal to one if the child is male, and zero otherwise. [c] The dependent variable is equal to one if the gestation period is below 37 weeks, and zero otherwise. [d] The dependent variable is equal to one if the birth weight is lower than 2,500 grams, and zero otherwise. [e] The dependent variable is equal to the Apgar score after ten minutes.

Table 8: Radiation effects (scarring) on fertility

	Low SES				Higher SES			
	Mean	37 kBq	42 kBq	47 kBq	Mean	37 kBq	42 kBq	47 kBq
1 yr after	0.012	0.014*	0.008	0.005	0.007	−0.001	−0.001	0.005
		(0.007)	(0.008)	(0.010)		(0.003)	(0.005)	(0.005)
2 yrs after	0.120	−0.034	−0.066***	−0.076***	0.121	−0.006	−0.005	−0.001
		(0.032)	(0.023)	(0.027)		(0.011)	(0.014)	(0.018)
3 yrs after	0.219	−0.012	−0.026	−0.036	0.240	0.005	−0.004	−0.002
		(0.034)	(0.033)	(0.037)		(0.017)	(0.017)	(0.021)
4 yrs after	0.305	−0.039	−0.059	−0.077*	0.334	0.025	0.009	0.000
		(0.036)	(0.037)	(0.042)		(0.020)	(0.020)	(0.024)
5 yrs after	0.377	−0.058	−0.082*	−0.116**	0.407	0.019	−0.005	−0.018
		(0.040)	(0.045)	(0.051)		(0.022)	(0.021)	(0.026)
6 yrs after	0.438	−0.084*	−0.109**	−0.122**	0.465	0.027	−0.006	−0.015
		(0.044)	(0.048)	(0.055)		(0.026)	(0.023)	(0.029)
7 yrs after	0.494	−0.095**	−0.113**	−0.132**	0.513	0.039	0.015	0.010
		(0.047)	(0.053)	(0.058)		(0.025)	(0.027)	(0.034)
8 yrs after	0.537	−0.103**	−0.126**	−0.168***	0.549	0.033	−0.003	−0.006
		(0.050)	(0.054)	(0.061)		(0.028)	(0.028)	(0.035)
9 yrs after	0.574	−0.090*	−0.117**	−0.155**	0.578	0.029	−0.006	−0.010
		(0.052)	(0.057)	(0.062)		(0.027)	(0.027)	(0.035)
10 yrs after	0.604	−0.092	−0.118*	−0.157**	0.603	0.026	−0.016	−0.012
		(0.056)	(0.062)	(0.069)		(0.031)	(0.030)	(0.038)
11 yrs after	0.628	−0.108*	−0.138**	−0.173**	0.622	0.024	−0.017	−0.013
		(0.056)	(0.061)	(0.069)		(0.032)	(0.030)	(0.038)
12 yrs after	0.649	−0.111*	−0.143**	−0.179**	0.637	0.017	−0.023	−0.026
		(0.057)	(0.062)	(0.069)		(0.033)	(0.030)	(0.038)
13 yrs after	0.665	−0.105*	−0.132**	−0.164**	0.649	0.015	−0.029	−0.034
		(0.058)	(0.065)	(0.075)		(0.035)	(0.031)	(0.039)
14 yrs after	0.678	−0.112*	−0.148**	−0.179**	0.658	0.017	−0.028	−0.031
		(0.059)	(0.065)	(0.075)		(0.036)	(0.032)	(0.041)
15 yrs after	0.687	−0.124**	−0.158**	−0.189**	0.666	0.020	−0.026	−0.036
		(0.059)	(0.065)	(0.076)		(0.036)	(0.032)	(0.041)
16 yrs after	0.695	−0.126**	−0.166**	−0.193**	0.671	0.019	−0.027	−0.036
		(0.061)	(0.065)	(0.076)		(0.037)	(0.032)	(0.042)
17 yrs after	0.703	−0.126**	−0.165**	−0.188**	0.675	0.023	−0.022	−0.029
		(0.062)	(0.067)	(0.078)		(0.037)	(0.033)	(0.043)
18 yrs after	0.707	−0.129**	−0.167**	−0.189**	0.678	0.027	−0.018	−0.027
		(0.062)	(0.067)	(0.078)		(0.037)	(0.034)	(0.043)
19 yrs after	0.711	−0.128**	−0.170**	−0.192**	0.681	0.028	−0.019	−0.028
		(0.062)	(0.067)	(0.078)		(0.037)	(0.033)	(0.043)
20 yrs after	0.713	−0.130**	−0.172**	−0.191**	0.682	0.026	−0.021	−0.030
		(0.062)	(0.068)	(0.079)		(0.037)	(0.034)	(0.043)
Obs.		25,009	23,243	22,035		69,775	63,869	60,261

This table summarizes estimation results based on individual-level data from the *Austrian Birth Register* and the *Austrian Social Security Database* covering families with births conceived between 08/1984 and 07/1987. The dependent variable is equal to the number of children born to the mother the respective number of years after the birth of the pivotal child. Each entry represents a separate regression, where the dependent variable is indicated in the first column, and shows the estimated coefficient for treated units from the BC_{II}. This cohort was conceived between 02/1986 and 04/1986 and was between 0 and 3 months post conception at the time of the accident. Each specification controls for community, conception-year, and conception-month fixed-effects. Method of estimation is a least squares. Robust standard errors (clustered at the community level) are shown in parentheses. *, ** and *** indicate statistical significance at the 10-percent level, 5-percent level, and 1-percent. Families with a low socioeconomic status (SES) have mothers with compulsory schooling or less. Families with a higher SES have mothers with any degree higher than compulsory schooling. Means refer to the 37kBq-cutoff sample.

Table 9: Radiation effects (scarring) on maternal labor force participation

	Low SES				Higher SES			
	Mean	37 kBq	42 kBq	47 kBq	Mean	37 kBq	42 kBq	47 kBq
1 yr after	0.086	−0.006	−0.001	0.009	0.106	−0.002	−0.010	−0.013
		(0.019)	(0.022)	(0.025)		(0.012)	(0.014)	(0.016)
2 yrs after	0.353	0.004	0.016	0.003	0.383	−0.040**	−0.030	−0.027
		(0.032)	(0.033)	(0.042)		(0.017)	(0.022)	(0.024)
3 yrs after	0.371	0.013	0.011	−0.001	0.410	−0.020	−0.009	−0.002
		(0.028)	(0.033)	(0.039)		(0.017)	(0.021)	(0.025)
4 yrs after	0.399	0.028	0.038	0.044	0.448	−0.010	−0.024	−0.021
		(0.034)	(0.038)	(0.048)		(0.018)	(0.020)	(0.025)
5 yrs after	0.448	0.076**	0.086**	0.083*	0.487	−0.016	0.009	0.029
		(0.038)	(0.038)	(0.047)		(0.020)	(0.020)	(0.025)
6 yrs after	0.469	0.067*	0.084**	0.069*	0.515	−0.015	0.012	0.020
		(0.040)	(0.036)	(0.041)		(0.019)	(0.020)	(0.025)
7 yrs after	0.487	0.059*	0.064*	0.051	0.535	−0.038**	−0.039**	−0.041*
		(0.036)	(0.037)	(0.043)		(0.015)	(0.018)	(0.022)
8 yrs after	0.507	0.024	0.031	0.038	0.560	−0.038**	−0.052***	−0.066***
		(0.034)	(0.035)	(0.041)		(0.018)	(0.019)	(0.023)
9 yrs after	0.530	0.018	0.027	0.046	0.590	−0.018	−0.030	−0.034
		(0.035)	(0.034)	(0.039)		(0.017)	(0.021)	(0.025)
10 yrs after	0.557	−0.005	0.008	0.007	0.619	−0.013	−0.006	−0.013
		(0.038)	(0.039)	(0.047)		(0.018)	(0.025)	(0.029)
11 yrs after	0.580	0.042	0.056	0.046	0.644	−0.007	0.002	−0.008
		(0.035)	(0.040)	(0.049)		(0.018)	(0.023)	(0.027)
12 yrs after	0.601	0.073**	0.095**	0.080*	0.668	−0.001	−0.003	−0.004
		(0.037)	(0.038)	(0.044)		(0.016)	(0.021)	(0.025)
13 yrs after	0.620	0.046	0.065*	0.065	0.690	−0.004	−0.010	−0.020
		(0.031)	(0.037)	(0.043)		(0.017)	(0.022)	(0.026)
14 yrs after	0.634	0.022	0.044	0.042	0.710	−0.003	0.000	0.004
		(0.029)	(0.035)	(0.038)		(0.016)	(0.020)	(0.024)
15 yrs after	0.652	0.027	0.047	0.057	0.733	−0.001	−0.001	−0.001
		(0.029)	(0.035)	(0.041)		(0.016)	(0.020)	(0.023)
16 yrs after	0.665	0.013	0.021	0.029	0.750	−0.001	−0.003	−0.010
		(0.028)	(0.033)	(0.037)		(0.016)	(0.021)	(0.025)
17 yrs after	0.670	0.039	0.032	0.035	0.761	0.013	0.015	0.017
		(0.029)	(0.037)	(0.044)		(0.014)	(0.019)	(0.020)
18 yrs after	0.670	0.053*	0.062*	0.065	0.766	0.009	0.011	0.010
		(0.030)	(0.037)	(0.044)		(0.013)	(0.017)	(0.020)
19 yrs after	0.668	0.064**	0.071*	0.076*	0.768	−0.006	0.002	0.002
		(0.031)	(0.038)	(0.045)		(0.013)	(0.018)	(0.020)
20 yrs after	0.663	0.057**	0.051	0.062	0.769	0.004	0.007	0.003
		(0.027)	(0.033)	(0.039)		(0.014)	(0.018)	(0.021)
Obs.		726,330	674,653	638,600		2,069,920	1,894,956	1,787,448

This table summarizes estimation results based on individual-level data from the *Austrian Birth Register* and the *Austrian Social Security Database* covering births conceived between 08/1984 and 07/1987. The dependent variable is equal to one if the mother is in the labor force in the respective number of years after childbirth. Each entry represents the coefficient for treated units from the BC_{II} interacted with years since the birth of the child (ranging from −9 years before to 21 years after birth). This cohort was conceived between 02/1986 and 04/1986 and was between 0 and 3 months post conception at the time of the accident. Each specification controls for community, conception-year, and conception-month fixed-effects. Method of estimation is a least squares. Robust standard errors (clustered at the community level) are shown in parentheses. *, ** and *** indicate statistical significance at the 10-percent level, 5-percent level, and 1-percent. Families with a low socioeconomic status (SES) have mothers with compulsory schooling or less. Families with a higher SES have mothers with any degree higher than compulsory schooling. Means refer to the 37kBq-cutoff sample.

Table 10: Percent share in workforce by age and socioeconomic status of family

Age of child	All families	Low SES	Higher SES
15	59.51	76.28	52.47
17	71.88	86.45	65.77
18/19	89.12	95.55	86.43

Own calculations based on Knittler (2011); see footnote 37.

Table 11: Children's long-term outcomes: Radiation effects (scarring) on labor force participation

	Low SES				Higher SES			
	Mean	37 kBq	42 kBq	47 kBq	Mean	37 kBq	42 kBq	47 kBq
Age 15	0.057	−0.015	−0.026**	−0.026**	0.025	−0.003	−0.008	−0.008
		(0.011)	(0.011)	(0.012)		(0.005)	(0.006)	(0.007)
Age 16	0.475	−0.070**	−0.082**	−0.088**	0.241	0.039	0.040*	0.054*
		(0.033)	(0.040)	(0.045)		(0.027)	(0.024)	(0.030)
Age 17	0.591	−0.065*	−0.100**	−0.101**	0.310	0.028	0.039	0.042
		(0.036)	(0.039)	(0.043)		(0.028)	(0.024)	(0.031)
Age 18	0.613	−0.065	−0.088**	−0.080*	0.326	0.024	0.031	0.037
		(0.041)	(0.040)	(0.045)		(0.025)	(0.025)	(0.034)
Age 19	0.586	−0.054	−0.071*	−0.050	0.319	−0.002	0.001	0.001
		(0.036)	(0.039)	(0.042)		(0.021)	(0.022)	(0.028)
Age 20	0.581	−0.095**	−0.127***	−0.103**	0.359	0.001	−0.013	0.012
		(0.038)	(0.041)	(0.045)		(0.020)	(0.024)	(0.030)
Age 21	0.671	−0.018	−0.030	−0.015	0.459	0.010	0.024	0.030
		(0.035)	(0.041)	(0.047)		(0.026)	(0.026)	(0.027)
Age 22	0.711	−0.068**	−0.089**	−0.097**	0.513	0.007	0.008	0.014
		(0.035)	(0.041)	(0.048)		(0.024)	(0.026)	(0.025)
Age 23	0.727	−0.052	−0.060	−0.082*	0.553	0.007	−0.002	0.015
		(0.033)	(0.037)	(0.044)		(0.021)	(0.027)	(0.031)
Obs.		161,260	149,690	141,400		507,110	463,650	436,920

This table summarizes estimation results based on individual-level data from the *Austrian Birth Register* and the *Austrian Social Security Database* covering births conceived between 08/1984 and 07/1987. The dependent variable is equal to one if the child is in the labor force at the respective age. Each entry represents the coefficient for treated units from the BC_{II} interacted with age. This cohort was conceived between 02/1986 and 04/1986 and was between 0 and 3 months post conception at the time of the accident. Each specification controls for community, conception-year, and conception-month fixed-effects. Method of estimation is a least squares. Robust standard errors (clustered at the community level) are shown in parentheses. *, ** and *** indicate statistical significance at the 10-percent level, 5-percent level, and 1-percent. Families with a low socioeconomic status (SES) have mothers with compulsory schooling or less. Families with a higher SES have mothers with any degree higher than compulsory schooling. Means refer to the 37kBq-cutoff sample.

Table 12: Children's long-term outcomes: Radiation effects (scarring) on apprenticeship training & labor income

	Low SES				Higher SES			
	Mean	37 kBq	42 kBq	47 kBq	Mean	37 kBq	42 kBq	47 kBq
Apprenticeship training[a]								
Age 16	0.378	−0.043	−0.040	−0.058	0.191	0.042	0.046**	0.060**
		(0.033)	(0.039)	(0.043)		(0.028)	(0.024)	(0.029)
Age 17	0.488	−0.081**	−0.080**	−0.094**	0.249	−0.004	0.014	0.021
		(0.033)	(0.040)	(0.043)		(0.030)	(0.023)	(0.028)
Age 18	0.489	−0.033	−0.032	−0.023	0.257	0.012	0.027	0.031
		(0.037)	(0.042)	(0.046)		(0.028)	(0.024)	(0.033)
Obs.		161,260	149,690	141,400		507,110	463,650	436,920
Income								
Age 15-23 (wage sum)[b]	9.542	−0.527	−0.739**	−0.939**	7.596	−0.062	−0.123	−0.003
		(0.322)	(0.328)	(0.393)		(0.204)	(0.252)	(0.261)
Age 15-23 (total wage sum)[c]	9.710	−0.549*	−0.770**	−0.971**	7.737	−0.055	−0.113	0.025
		(0.325)	(0.329)	(0.394)		(0.208)	(0.257)	(0.263)
Obs.		16,126	14,969	14,140		50,711	46,365	43,692

This table summarizes estimation results based on individual-level data from the *Austrian Birth Register* and the *Austrian Social Security Database* covering births conceived between 08/1984 and 07/1987. Each entry represents the coefficient for treated units from the BC_{II} interacted with age. This cohort was conceived between 02/1986 and 04/1986 and was between 0 and 3 months post conception at the time of the accident. Each specification controls for community, conception-year, and conception-month fixed-effects. Method of estimation is a least squares. Robust standard errors (clustered at the community level) are shown in parentheses. *, ** and *** indicate statistical significance at the 10-percent level, 5-percent level, and 1-percent. Families with a low socioeconomic status (SES) have mothers with compulsory schooling or less. Families with a higher SES have mothers with any degree higher than compulsory schooling. Means refer to the 37kBq-cutoff sample. [a] The dependent variable is equal to one if the child is in apprenticeship training at the respective age. [b] The dependent variable is equal to the sum of the deflated annual labor income between ages 15 and 23 in the main job. [c] The dependent variable is equal to the sum of the deflated annual labor income between ages 15 and 23 in all jobs.