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

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

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Abstract

Little is known about the response behavior of parents whose children are exposed to an early-life shock. We interpret the prenatal exposure of the Austrian 1986 cohort to radioactive fallout from the Chernobyl accident as a negative human capital shock. We can rely on exogenous variation in the exposure to radioactive fallout (over time and) between communities due to differences in precipitation at the time of the accident. Our approach provides robust empirical evidence for compensating investment behavior. Families with low socioeconomic status reduce their family size, while families with higher status respond with reduced maternal labor force participation.

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Keywords: Fetal origins, parental response, Chernobyl, radiation, health, culling, human capital, fertility, labor supply.

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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 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, that is, 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 interpret these estimates as reduced-form effects.

Until very recently, the design-based literature on early-life conditions 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, to reach a deeper understanding of the relationship between early-life shocks and the formation of human capital, it is crucial to examine the behavior of parents.

We examine a shock during the prenatal period, which is considered as the key developmental window (Almond and Currie, 2011b). 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. Thus, 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 (like in 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 to this early-life shock and its mediating impact on children's long-term outcomes. Given the Austrian institutional setting, where the major out-of-pocket expenses for children, such as health care and education (including college), are completely covered by the state, we consider parental time investment as the most important response behavior. To operationalize this idea we analyze fertility and

[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, the papers in a development context (Adhvaryu and Nyshadham, forthcoming; 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.*, 2013).

[2] Austria ranks among those 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 higher levels; see Figure 3.5. in IAEA (2006).

maternal labor force participation. These outcome variables should proxy well for parental time with children. The main result of our paper is that parents respond with compensating investment behavior. Families with low socioeconomic status reduce their family size (i. e., lower completed fertility), while families with higher socioeconomic status reduce 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, since mothers reduced their labor supply shortly after their children entered 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 at birth; AEP did not detect any health damage. In contrast, we find robust evidence that the in utero exposure to radioactive fallout increased the incidence of early fetal death. As a consequence, surviving children exhibit better health outcomes at birth. This result illustrates the more general phenomenon that in the presence of an effect 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 received less attention in the economic literature on early-life shocks. The neglect 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 trimming 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 the second step, we present the main results of our paper. We document the parental response behavior along the dimensions of family size and maternal labor supply over a period of twenty years. We interpret the negative effect along both dimensions as evidence for compensating parental investment. This result has two important implications for the literature on early-life events. First and foremost, it implies that estimated effects in this literature have to be interpreted as reduced-form effects. Second, it highlights that the commonly used estimation strategy of family fixed effects is problematic, if older otherwise unaffected siblings are affected by parental response behavior.

In the third step, we analyze the long-term effects on children. The estimates give us the overall impact of the accident, the sum of the biological effect and the parental response. Given that we know that parents try to compensate for the early-life shock, we can interpret our estimates as a lower bound of the biological effect. 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. In line with this, AEP do not find statistically significant effects for children from high socioeconomic backgrounds. A zero effect could result from effective compensating investment. This highlights that a complementary analysis of parental response behavior is indispensable to understand the relationship between early-life conditions and long-term outcomes.

Finally, in a fourth step, we quantify the intra-household spillover effects and compare older siblings of exposed and non-exposed children. We find that siblings of exposed children from low socioeconomic backgrounds have better human capital outcomes. Positive externalities of the compensating parental response (i. e., reduced family size) seem to outweigh any negative effects of the redistribution of private goods.

Our findings have important ramifications for the economic literature beyond the studies on the effects of early-life events. 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 and Tomes, 1976). Or, consider the literature on environmental justice, which 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 investment may increase along the socioeconomic distribution. Each of these explanations suggests that in case of conventional environmental hazards, 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]

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. Our results also speak

[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.

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. 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 and translate into reduced fertility and lower economic productivity of at least two generations.

The remainder of the paper is organized as follows. Section 2 describes the Chernobyl accident and the resulting radioactive contamination in the western part of the former Soviet Union and in 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 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 that caused the worst nuclear power plant accident in history. 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 of Europe. The radioactive particles were subsequently removed from the atmosphere solely due to gravitation (*dry deposition*) or by any form of precipitation (*wet deposition*). Since 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. 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 are considered the most important exposure pathways. 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 we do not observe 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. The Chernobyl accident

[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.

provides an ideal natural experiment to study the consequences of exposure to radioactive ground deposition, since it seems safe to assume that the spatial distribution of precipitation during the passage of the plume was exogenous.[5]

The implementation of this research design is facilitated by the wide availability of data on local levels of ground deposition. In the aftermath of the accident, the level 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.

3 Research design

In this section, we first present the radiation data that we use to determine the individual level of exposure to radioactive fallout. We then discuss the periods of prenatal exposure between which we distinguish and explain how we translate our research design into a regression framework. 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).[6] We aggregate these measurements to the community level and focus on the average level of ground deposition of ^{137}Cs.[7] This provides us with data for 924 (out of 2,331) communities, where each data point refers to May 1, 1986.[8]

[5] 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 assess how large this correlation is, since it depends on the structure of the food supply chain.

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

[7] These measurements include the global fallout from the atmospheric nuclear 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 give very comparable results. In particular, the point estimates are very similar, though 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.

[8] 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 levels. 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 verified the robustness of our results for birth outcomes. These results are

The accident happened on April 26 and the radioactive plume arrived in Austria on April 29. 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.

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.[9] Table 1 summarizes this grouping of communities and provides the population-weighted average of ^{137}Cs ground deposition for each group.

Since 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. Precipitation intensity is mainly determined by altitude. This explains why 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. 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, preterm birth, or low birth weight we do not see any statistical significant differences. (See, for instance, Panel (a) of Figure 2). Our estimation framework (to be explained below) will clear any time-constant differences.[10]

available upon request.

[9] Our results are quantitatively and qualitatively similar when including communities with medium levels of contamination. The point estimates for these communities are lower and not significant throughout. We have also used a continuous measure of exposure to ^{137}Cs ground deposition. Again, our results are robust. All results are available upon request.

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

3.2 Periods of prenatal exposure

It is conjectured that radiation exposure is especially critical at a prenatal stage.[11] 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 post conception), 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) post conception. 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 8–25 weeks at the time of the accident. We use a larger window and include all conceptions between August 1, 1984 and July 31, 1987 in our estimation sample. Including conceptions from this time span allows us to control for seasonal effects at the monthly level. We distinguish between four different birth cohorts (see also Figure B.1 in Web Appendix B): Birth cohort 0 (BC_0) includes all children who were conceived before August 1, 1985 and born before the Chernobyl accident. 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 and third trimesters) and should be relatively 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. They have been in utero for less than 3 months at the time of the accident (first trimester) and should still be very vulnerable. 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 (between May 1, 1986 and July 31, 1987) and born after the accident. While this prenatally non-exposed cohort is *per se* not interesting, its inclusion allows us to fully account for seasonal and year effects.

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

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

[11] 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).

day (cd) as follows: $cd = bd - 7 * (gl - 0.5)$. We assume that a pregnancy with a stated gestation length of 38 weeks has lasted 38.5 weeks or 269.5 days. 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 ages of 20 and 40. We do not exclude multiple births (about 2 percent of children), but, when analyzing parental response behavior, we include only one observation per birth. We also exclude some children because of potential intra-household spillover effects (see below). After applying those sample selection criteria, our most extensive sample includes 95,103 children (see Table 1). The number of observations depends on the treatment group definition that we use. In case of $T1$, we observe 22,496 exposed children and 72,607 non-exposed children and exclude 37,335 children from communities with medium levels of ^{137}Cs ground deposition.

3.3 Intra-household spillover effects

In the presence of intra-household spillover effects, otherwise unaffected siblings of exposed children are potentially affected by the accident. The first case is where a child was prenatally exposed and his younger sibling was not. The spillover effects apply to the younger sibling's outcomes measured at any point in time; these observations are generally problematic. The second case is where a child was prenatally exposed and his older sibling was not. In this case, the spillover effects apply to older sibling's outcomes measured after the exposed child's birth. Thus, these observations cannot be used in the analysis of long-term outcomes; but are unproblematic in the analysis of prenatal culling and health at birth. In the analysis of parental response outcomes, the inclusion of problematic observations should lead to a downward bias in radiation effects (this applies to compensatory and reinforcing investment), since affected and unaffected siblings share the same parental response in terms of family size and maternal labor supply. In the case of children's long term outcomes, the sign of the bias is unclear (see Section 4.5). To preserve a clean research design, we exclude all problematic observations from our analysis.[12] Moreover, we abstain from using specifications with family fixed effects, since these are problematic in the case of spillover effects.

3.4 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 $T3$):

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

[12] Since our sample includes only few siblings, the results do not change substantially.

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 four birth cohorts, 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 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 accident. While this assumption seems quite plausible, we relax it further in a sensitivity analysis by including community specific time-trends and other community-level covariates (such as local unemployment rates). Moreover, we also do some placebo testing by assuming that Chernobyl took place at an earlier point in time. All these additional analyses confirm our results and support the validity of the identifying assumption.

Since the local level of ground deposition was not known 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.[13] Put differently, one might 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. This means that this early parental response behavior may have generated scarring and/or culling effects. Given that these effects are

[13]We have scanned three major national newspapers (*Die Presse*, *Neue Kronen Zeitung*, and *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 fruits, 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 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 referring to federal states.

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 the early parental response did not vary with 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. 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 higher socioeconomic backgrounds. We use 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. In Web Appendix C.1 we summarize the main estimation output also for the pooled sample.

3.5 Outcome variables

We examine health and human capital outcomes 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 force participation in the post-treatment period. Table B.2 in Web Appendix B provides an overview of all outcomes with information on measurement and data source.

For the estimation of prenatal culling effects, we use the *Austrian Birth Register*. This includes the universe of all live births and stillbirths in Austria with individual-level information on socioeconomic characteristics and birth outcomes. These data allows us to quantify the incidence of live births on a community-level (by socioeconomic groups) and to conduct an individual-level analysis of the likelihood of a stillbirth. 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 effects examines health at birth and focuses on different health indicators such as gestation length, birth weight, and Apgar scores. To evaluate long-term effects, we focus on human capital formation and labor market outcomes in early adulthood (up to the age of 23). In particular, we obtain individual-level information on employment, broad occupation, apprenticeship training, and wages from the *Austrian Social Security Database*. To evaluate parental investment behavior, we use family size and maternal labor force participation.

4 Estimation results

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 to *not* 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. In the next step, we analyze the long-run effects on children's human capital outcomes. Finally, we test for intra-household spillover effects by examining long-term outcomes of otherwise unaffected siblings.

In the paper, we focus on the results for BC_{II}, which was in the first trimester post conception at the time of the accident. An equivalent analysis for BC_I, which was in the second and third trimester post conception is provided in Web Appendix C.2. A comparison of these two sets of results corroborates the conjecture that prenatal exposure to radiation is less critical at a higher gestational age.

4.1 Culling effects

The possible effects of prenatal radiation exposure include increased risk for medical conditions later in life (such as cancer), and 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 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. First, we need to estimate the extent of culling. Second, we need an empirical strategy to disentangle the 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 with radiation dose and decreases with gestational age.[14] Mortality risk is especially high in the first weeks after conception, since an embryo is made up of only a few cells. Damage to one cell (the progenitor of many other cells) may cause embryo death. Beyond about 26 weeks, the fetus is believed to be "relatively radio-resistant".

Prenatal culling To calculate the extent of prenatal culling, we would have to compare the number of conceptions with the number of live births. Clearly, we cannot observe conceptions. We also do not have information on the incidence of miscarriages. Very early miscarriages (so-called early pregnancy losses) happen in many cases before a woman may even know

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

she is pregnant, and therefore, without clinical recognition. Later miscarriages, which occur after the sixth week since the woman's last menstrual period 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 the birth of a child of at least 35 centimeter of length, without vital signs.[15] Smaller fetus are categorized as miscarriages, and therefore, not documented. Finally, live births are very well documented in the *Austrian Birth Register*. Information on induced abortions is not available. Although, abortion has been legal in Austria since 1975, no official statistics exist.

We propose three complementary strategies to infer on the effects of Chernobyl on prenatal culling. 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. The precise prenatal mechanism how 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 offsprings 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 offsprings (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). The upper panel of Table 2 summarizes the estimated effects on prenatal culling.

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. We divide this variable by its sample mean to facilitate a percentage-change interpretation. We find a statistically significant negative effect, which provides first evidence on prenatal culling. The effect amounts to 8.6–11.2

[15] 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 the birth of a child of at least 500 grams weight without vital signs.

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 cannot directly observe miscarriages, we can use the sex of the child as a proxy. The results summarized in the third panel show 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 4.3–8.8 percentage points. The size and significance of this effect increases with the level of radioactive exposure, and is in line with evidence from other early-life shocks. For instance, Nilsson (2014) reports that the increased availability of alcohol reduced the probability of male birth by 7.3 percentage points. Our finding is consistent with the negative effect on live births (and the 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 that are lost spontaneously (aborted). The incidence of miscarriages is widely believed to be about 40 percent of all pregnancies (Macklon *et al.*, 2002).[16] This means that the effect of prenatal radiation exposure of roughly 10 additional 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 smoke more than 10 cigarettes per day during the first trimester have an estimated increased risk of miscarriage 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 — point estimates in line with fewer 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). This means that the unborn children of these mothers are also weaker at any prenatal stage, and any negative shock should have more detrimental effects. It would be revealing to explore this mechanism more directly, by splitting the sample by mothers' health status. While we do not have access to individual-level health information, we use mothers' age as a proxy for health. As expected, we find that the effect on the sex-ratio is driven by older mothers (above 30 years of age). Second, our finding is in principle consistent with research highlighting an educational gradient in the reaction to emerging health risk information. For instance, Aizer and

[16] 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.

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. 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 as in non-exposed communities, without knowing their actual treatment status — took measures that successfully reduced their exposure to radiation. Since it seems hardly feasible to reduce exposure to radiation (one would have to stay inside over a longer period of time and manage to avoid contaminated food and water), we consider the first explanation as the more plausible one.

In sum, this set of results provides evidence that prenatal radiation exposure (to ^{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 the lower panel of Table 2. 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 suggests that exposed children (who survived the prenatal culling stage) were in no different physical condition as compared to non-exposed children, or were 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 up to one month after birth. One year after birth, no significant differences exist. This suggests that prenatal culling led to a slightly positively selected sample of live births in this particular group.

4.1.2 Cancelling out culling effects

To motivate our empirical strategy, which allows us to disentangle the culling and scarring effects, we formalize the tension between these two effects. While we frame it with respect to fetal mortality — which empirically turned out to be the 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 $b_i > z+r$. The fetal mortality rate m^T in exposed communities T is then given by $m^T = F(z+r)$.

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.[17] Thus, for survivors, we have $\tilde{b}_i = b_i - \tau r$. The average birth endowment of the survivors in exposed communities is then given by

$$\bar{b}^T = \underbrace{\frac{\int_{z+r}^{\infty} b \cdot f(b) db}{1 - F(z+r)}}_{\text{Culling}} - \underbrace{\tau r}_{\text{Scarring}} . \tag{2}$$

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 the 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 cannot disentangle the 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 (a) of Figure 2 shows the empirical cumulative distribution of gestation length for mothers with low socioeconomic status in BC_0 providing evidence that substantiates this assumption. The untruncated distribution — i. e. the average birth endowment of children born in non-exposed communities — is then given by

$$\bar{b}^C = \frac{\int_{z}^{\infty} b \cdot f(b) db}{1 - F(z)}. \tag{3}$$

We suggest a sample correction method to disentangle the 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

[17] 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 $b_i > z+r+\tau r$.

survived if they had been exposed to the accident.[18]

We exploit our knowledge about the extent and nature of the culling effects caused by the 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 the 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

$$\Delta m = F(z + r) - F(z). \tag{4}$$

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 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. 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 2 shows the observed distribution of gestation length for mothers with low socioeconomic status from BC_{II} by treatment status. The graph illustrates that the percentage of preterm births is lower in 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 2. The comparison of the two culled samples in Panel (d) of Figure 2 shows that the distribution of gestation length in the treatment group now dominates the distribution in the culled control group. 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

Table 3 summarizes the estimation results based on individual level data for the commonly used outcomes, gestation length, birth weight, and Apgar scores. A birth is classified preterm if gestational length is below 37 weeks. Weight at birth is typically considered as low if it is below 2500 grams.[19] The Apgar score quickly and summarily assesses after one, five, and ten minutes the health of newborn babies based on five criteria (appearance, pulse, grimace,

[18] Our procedure is comparable to the one suggested by Lee (2008). However, we use the culled sample to analyze further outcome variables and are only interested in the upper bound estimate (and not the lower bound estimate).

[19] Both a preterm birth 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, e. g., Black *et al.*, 2007).

activity, and respiration) and ranges from zero ("bad") to ten ("good"). For each outcome, we present estimated coefficients based on the observed sample (first row) and the corrected sample (second row). In the former case, the estimates capture the sum of the culling and scarring effects, while in the latter case, only the 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 preterm birth, the overall effect is statistically significant across specifications, and the point estimates marginally increase with the level of radioactive exposure. Children born alive are estimated to be about 3 percentage points less likely preterm. Given an average incidence of preterm births of about 5.6 percent, the estimated effect is substantial and supports the notion that 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 based on our corrected sample reveal statistically significant scarring effects. In the case of preterm births, this is a mechanical result, since we have manually "culled" the lower tail of the gestational age distribution. The estimated scarring effects amount to about 3 percentage points. In the case of the other outcomes, we also observe negative scarring effects. The estimated effects on birth weight 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 another 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 preterm 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. Thus, an extended post-birth spell may be 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 preterm births and/or of better health. Again, after we apply our sample correction method, these effects vanish.[20]

Families with higher socioeconomic status In the case of children from mothers with higher socioeconomic status, we do not apply the sample correction method, since we find very little evidence for prenatal culling. The analysis of health at birth (consequently based on the observed samples), however, partly suggests evidence for positive culling effects. While estimated effects on the likelihood of preterm 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.

[20] 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.

Robustness of results As a sensitivity check of our identification strategy, we conduct a placebo-test by estimating an equivalent DiD estimation where we assume that Chernobyl happened one year earlier. Accordingly, we define a control cohort *BC-A*, which was conceived between 07/1983 and 06/1984, and a placebo cohort *BC-B*, which was conceived between 07/1984 and 06/1985. Since both cohorts were not affected by the accident in utero (i. e. they were born before the accident), we would be concerned if we find any effects based on this specification for pre- and postnatal culling, or health at birth. As the results (summarized in Table C.3.1 in Web Appendix C.3) show, we find no statistically significant effects. Also, there does not appear to be any systematic pattern in the results that would suggest that radiation effects presented above are in any way an artifact of differential trends between treated and control communities.

Any outcome measured later (of either cohort in exposed communities) may be influenced by Chernobyl via intra-household spillover effects (as detailed in Section 3.3), and is therefore not suited for a placebo analysis.

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 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.[21]

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.[22] This means that the negative early-life shock should have more detrimental effects for the average Austrian child as compared to the average Swedish child.

Third, AEP define the birth cohort with a gestational age below eight weeks as non-treated. It is possible that the prenatal culling is particularily 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 4. We estimate two specifications. In the first specification, the treatment group consists of children who have been in utero between 2 and

[21] Although both countries rank among those countries with the highest contamination levels in Western Europe, the relative size of the contaminated area is 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.

[22] 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.

6 months (about 8–25 weeks) at the time of the accident. This specification uses the same definition of treatment status regarding timing as AEP. In the second specification, we also include children who have been in utero between 0 and 2 months (about 0–7 weeks). 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 significant 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. 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 2 and Table 3). 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. It seems that prenatal culling is actually driven by the birth cohort with a gestational age between 0 and 3 months (our BC_{II}).[23]

4.3 Parental response behavior

In our analysis of the parental responses we do not impose any assumption on whether the parents become aware of their child being prenatally exposed to radioactive fallout. Our implicit assumption is that parents observe the physiological and/or cognitive deficiencies of their child and may react to these.

Austria has comprehensive (welfare) state arrangements that guarantee, among others, universal access to free high-quality healthcare and a free public school system. Since all major out-of-pocket expenses to build children's human capital are covered by the state, we consider parental time as the key parental response behavior.[24] Del Boca *et al.* (2014) provide evidence that parental time is very important for the cognitive development of children. To quantify time spent with children, we examine families' post-treatment fertility and maternal labor force participation. Both outcome variables should be closely related to parental time investment.[25] Thus, we interpret a lower family size and a reduced maternal labor force participation as compensating investment. In contrast, an increased family size and higher maternal engagement on the labor market is interpreted as reinforcing investment.

Table 5 summarizes the estimations results on post-treatment fertility. Each entry rep-

[23] 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.

[24] Private schools (mostly denominational) are the exception; especially in higher education. State universities charge no or very low (about Euro 760 per year) tuition fees. Students from low-income households (do not have to pay any tuition fee and) receive a study grant.

[25] Ideally, explicit information on time use should be employed. Unfortunately, this information is not available in administrative data sources, and the only available time use survey in the relevant sample period does not include information on the community of birth.

resents the results from a separate regression, where the dependent variable is equal to the number of children a BC_{II} mother has born until the respective year after the birth of the pivotal child. Since the mothers 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 6 summarizes the estimations results on maternal labor market participation.[26] 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.

In the case of families with low socioeconomic status, we perform the estimation for the observed and the corrected samples. The estimates are somewhat larger in absolute terms in the corrected samples. Culling effects seem to be of second-order importance in the case of long-term outcomes. Still, we focus here on the results based on the corrected sample (see Table 5 and Table 6) and relegate the estimation output based on the observed samples to Web Appendix C.4 (see Table C.4.1 and Table C.4.2).

Families with low socioeconomic status Exposed families with low socioeconomic status have significantly fewer children at any point in time (see Table 5). The size of the effect increases in absolute terms with the level of radioactive exposure and over time. Twenty years after birth, the effect amounts to minus 0.18 to minus 0.12 children. This is equivalent to a reduction in completed family size of about 17–26 percent. A closer look at the evolution of the estimated effects over time (i. e., first differences in the estimates) reveals that the reduction is predominantly due to less births in the second, fifth, eight, and eleventh year.

Does this effect necessarily reflect a compensatory investment decision, or are there alternative explanations? One alternative explanation we can think of, is a pure biological effect; i. e. women exposed to radioactive fallout are less fertile subsequently. This explanation, however, seems implausible. First, we are not aware of any medical evidence, suggesting that these levels of radioactive exposure affect fertility. Second, we only find significant effects for exposed BC_{II} mothers, but not for their BC_I counterparts, who were exposed to the *same* levels of radioactivity at a different gestational age (see Web Appendix C.2). Further, the fertility effects are not related to miscarriages (caused by the Chernobyl accident), since the estimated effects are identified based on treated mothers who did *not* lose their child.

Starting from about four years after birth, exposed women are more likely to be active on the labor market (see Table 6). However, 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 eight percentage points. We suggest *not* to interpret this labor market response as reinforcing investment, but as a direct consequence of the reduced

[26] Due to an 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.

family size.[27] 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 the fifth and twelfth years) 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 5). The estimates are all statistically not significantly different from zero and the coefficients are quite close to zero. This means, that there is neither evidence for any impact in terms of the 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 6). 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 eighth year, and dissipates over time. After the tenth year, the estimates are basically all zero. Notably, the estimated coefficients for the years 1 to 14 are jointly statistically significant. At the peak, the effect is between minus 4.0 and minus 6.9 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 *early tracking* in the Austrian education system, grades in primary school have far-reaching consequences for later educational choice (which we will discuss in more detail in Section 4.4). Therefore, involved parents will take different measures to solve any learning difficulties at this stage. Given the basic content of the curriculum, professional tutoring is uncommon for pupils of this age and typically, parents teach their children after school to overcome any problems.

Robustness of results Our identifying assumption requires that families in exposed and non-exposed communities would have followed the same trend in outcome variables over time in the absence of the accident. First it can be noted, that if exposed and non-exposed communities would have followed differential trends (for other reasons), this should have applied to all birth cohorts. However, our results show only significant effects for BC_{II} (the cohort which is expected to be most vulnerable to radioactive exposure). Still, in order to relax this assumption, we augment our specification by community-specific trends based on consecutive conception months. This very rich specification provides basically unchanged point estimates with somewhat larger standard errors. In the case of maternal labor force participation, we further test the robustness of our results to the inclusion of measures of the regional labor market situation. The best available measure is the unemployment rate on a district level. This additional covariate changes our point estimates and standard errors only marginally. The same holds true when we interact this covariate with the treatment indicator. Detailed estimation output is available upon request.

[27] Nevertheless, this indirect effect on maternal labor force participation counteracts the positive effect of reduced fertility on parental time; but also increases available resources.

In sum, we find for both socioeconomic groups statistically significant evidence for increased parental time spent with treated children. 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 adapt their behavior, such that they can devote more time to their children.[28]

How do these results compare to other findings in the literature? The existing design-based literature on the parental response to child endowments is rather scarce (see footnote 1), especially with respect to *developed* countries. Bharadwaj *et al.* (2013) 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.

4.3.1 Non-radiation effects

The local level of ground deposition was not known at the time of the accident. Therefore, parents (or potential parents) in exposed and non-exposed communities may have been stressed and anxious in the aftermath of the accident. This may have changed their behavior immediately after the accident and caused what we term *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 may have been stressed and anxious. This stress *per se* may have detrimental effects on the embryo or fetus, or even lead to miscarriage.[29] A very direct culling effect is given if pregnant women decide to have an induced abortion.[30] Less drastically, expecting mothers could have tried to reduce exposure to radiation. While it is unlikely that such an avoidance behavior (e. g., change of diet) is effective in reducing exposure to radiation, it could have affected the child in some way.

The scope of early parental response behavior differs across birth cohorts. Parents of a child from BC_I had the smallest scope for action. An induced abortion was not possible anymore, since the pregnancy had advanced past the first trimester (the legal time limit).

[28] We have also examined the length of parental leave spells: no difference between exposed and non-exposed families is discernible.

[29] In animal studies there is experimental evidence on the negative effects of in utero exposure to maternal stress on offspring outcomes (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) report 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.

[30] 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).

However, their unborn child could have been exposed to maternal stress or a modified maternal diet during the last two trimesters of pregnancy.[31] In comparison, parents of a child from BC_{II} could have reacted quite drastically and have an abortion.

Table C.5.1 in Web Appendix C.5 summarizes the effects for prenatal culling and health at birth. In the case of 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 a significant negative effect on the likelihood of male birth. The effect is almost identical across different levels of exposure.[32] This does not affect the outcomes gestational age and birth weight at the critical margins (i. e., preterm birth or low birth weight). The parameter ρ_2 gives the estimated non-radiation effects (for BC_{II}) due to miscarriages and induced abortions. No significant effects are found. 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 of induced abortions. 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. Still, we find statistically significant evidence (not tabulated) that induced abortions have decreased the fetal death rate between 3.8–4.3 percentage points (depending on the level of radiation).

Among mothers with low socioeconomic status, we do not find any significant non-radiation effects. This suggests 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

We now assess the long-term effects on exposed children. Any outcome measured after birth (e.g., test scores) is affected by parental response behavior. Since we observe that parents try to compensate for the early-life shock, we can interpret the estimates in this section as a lower bound of the biological effect. We examine human capital outcomes between the ages of 15 and 23.[33] 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 (either as an apprentice or as a regular employee). This applies in particular for children from low socioeconomic backgrounds (see Table B.3 in Web Appendix B).

[31] There is some evidence for a high responsiveness of birth weight to nutritional changes in the third trimester of pregnancy (Painter *et al.*, 2005; Almond *et al.*, 2011).

[32] While in the case of radiation effects, we 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 had not been made aware of the local level of ground deposition.

[33] This analysis is based on a sub-sample (as compared to the analysis of children's health at birth). We are able to link 70 percent of the children in the *Austrian Birth Register* with the ASSD. 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 these 30 percent of children.

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 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 a university entrance exam.[34]

Low track career path The majority of the students (about 72 percent) are allocated to the *low track*. This share is higher among children from parents with low socioeconomic status (87 percent) than among those from parents with higher socioeconomic status (66 percent).[35] Approximately 82 percent of students from the low track enter the work force at around 15 years of age, ideally via the dual education system or as an unskilled worker. The dual education system combines 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 are allocated to 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, do not include any information on educational attainment, it comprises detailed information on all workers in Austria regarding their labor market status in employment, 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 of birth). Table 7 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. Each entry represents the coefficient for exposed children from BC_{II} interacted with their age. In Table 8, we further estimate the

[34] A further institutional detail of the Austrian education system impedes an analysis of test scores. Tests in either track are decentralized. This means that 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.

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

impact on the likelihood of being an apprentice at ages 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 corrected samples. As in the case of parental response behavior, the sample correction has little impact. This means that 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 here the estimates based on the corrected sample and relegate the estimation output for the observed sample to Web Appendix C.4 (see Table C.4.3 and Table C.4.4).

Put simply, due to high intergenerational educational persistence in Austria (OECD, 2010), there are two realistic successful career paths for children from low socioeconomic backgrounds. They 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 8). Second, exposed children are less likely to be employed throughout the whole time period under consideration (see Table 7). While not each coefficient is individually significant, they are consistently negative and jointly significant. The effect is on average minus 7 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.[36] This means that exposed children from low socioeconomic backgrounds have worse educational and labor market outcomes and have (at least until the age of 23 years) accumulated less human capital. We conclude that for this group prenatal exposure to radiation has — despite compensating behavior of their parents — substantial long-term scarring effects. Based on wage regressions (42 kBq), the estimated loss in annual before-tax income amounts to 8665 Euro or 53 percent. A quantification of the corresponding loss in the counterfactual situation, where parents of exposed children would not have adjusted family size (as a response to the treatment) is hard, since this compensating investment is endogenous.

Families with higher socioeconomic status For this group, we find little evidence for effects on their labor market outcomes. At the age of 16, they are somewhat more likely to be in

[36] 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 less likely 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 farfetched. Only 14 percent of children from low socioeconomic backgrounds complete the high track — this would imply an treatment effect of almost 60 percent. Still, to provide supportive evidence for our interpretation, we have estimated the effect on the 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.

the labor force (about four to five percentage points). A comparison between Table 7 and 8 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, 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 that even if some negative scarring effects are present in adolescence, they seem to vanish over time. Again, it is unclear whether exposed children would have worse outcomes in the case where parents would have not compensated for the shock.

These results conform with AEP, who find that Swedish children in low-educated families 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. 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 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 non-observable dimensions (such as private tutoring or social job-finding networks).

In contrast, Black *et al.* (2013) — who show with their Norwegian data that in utero exposure to radioactive fallout caused by nuclear weapons testing reduced IQ scores, educational attainment, and earnings — find almost no differences between socioeconomic groups.

4.5 Siblings' human capital outcomes

Last, we ascertain whether the compensating parental response has spillover effects onto otherwise unaffected siblings. *A priori*, it is unclear whether the compensatory investments come at their cost or to their benefit. On the one hand, siblings may have suffered if parents reallocate resources (i. e., private goods) from them to the exposed child. We do not have information on this dimension. On the other hand, they may have benefited from an increased supply of local public goods (e. g., via reduced family size). Thus, the net effect can be positive or negative.

Since post-treatment fertility is endogenous we use only families who had at least one child before the pivotal child. Our most extensive sample (using treatment group definition 1) comprises 52, 461 older siblings, of which 26 percent had an exposed sibling and 74 percent had a non-exposed sibling. An equivalent estimation analysis as in the previous section is summarized in Tables C.6.1 and C.6.2 in We Appendix C.6. We find a positive effect on labor force participation for children from either socioeconomic background. In the case of children from low socioeconomic backgrounds, we do not see a different likelihood of apprenticeship training; however, we find positive effects on labor force participation in their early 20s.

Untabulated results show that this is driven by employment (predominantly in white-collar jobs) and not by unemployment. In sum, these siblings seem to have benefited from the reduced family size. This is confirmed by some positive effects on their annual before-tax income.

For children from higher socioeconomic backgrounds, we observe positive effects on labor force participation around the age of 16 and in their early 20s. The early effects are driven by a higher likelihood of apprenticeship training. The effects in their early 20s are more pronounced (and statistically significant) if we look at employment (not shown in table). More detailed regressions reveal that the effects are driven by blue-collar jobs. We find no effect on the wage sum; this is in line with a zero sum of more employment in lower-paying blue collar jobs. Given that apprenticeship training and a blue-collar job do not represent a desirable career path for this group of siblings, we interpret the overall effect as negative. This suggest that in families with higher socioeconomic status the negative effect of the reallocation of private goods dominates the positive externalities of the compensating behavior. One may speculate that the positive spillover effects are larger in the case of reduced family size as compared to reduced maternal labor supply.

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. We study the case of prenatal exposure in the Austrian 1986 cohort to radioactive fallout from the Chernobyl accident. Identification is based on exogenous 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 parental investment that differs in type and effectiveness across families' socioeconomic backgrounds.

Our results urge caution in the interpretation of estimates of the long-term effects of early-life shocks on children. They demonstrate that these estimates can only be interpreted as reduced-form effects, and not as the biological effect of the shock; since parental investment behavior is an empirically relevant phenomenon. To reach a deeper understanding of the relationship between early-life shocks and the formation of human capital, it is indispensable to account for parental response behavior.

In the case of Chernobyl, we find that parents try to compensate for the early-life shock. Families with low socioeconomic status reduce their family size, and families with higher socioeconomic status reduce maternal labor supply temporarily. This observation allows us to interpret the estimated long-term effects on children as a lower bound of the biological effect. Exposed children from low socioeconomic backgrounds still have worse outcomes in young adulthood, whereas we do not find any detrimental long-term effects for children from higher socioeconomic backgrounds. Notably, we find spillover effects on otherwise unaffected older siblings for each group.

These results should also be of interest to policy-makers. It provides a strong argument for providing disadvantaged families with the necessary economic and social resources that allow early childhood investment. It is widely documented that children from low socioeconomic backgrounds typically grow up in less-favorable environments and 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 status — adjust their behavior to invest in their children according to their specific needs. However, parental response behavior of families with higher socioeconomic status seems more effective. Families with low socioeconomic status are most likely to be more restricted in their compensatory investment along pecuniary and non-pecuniary dimensions.

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6 Tables and figures (to be placed in paper)

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

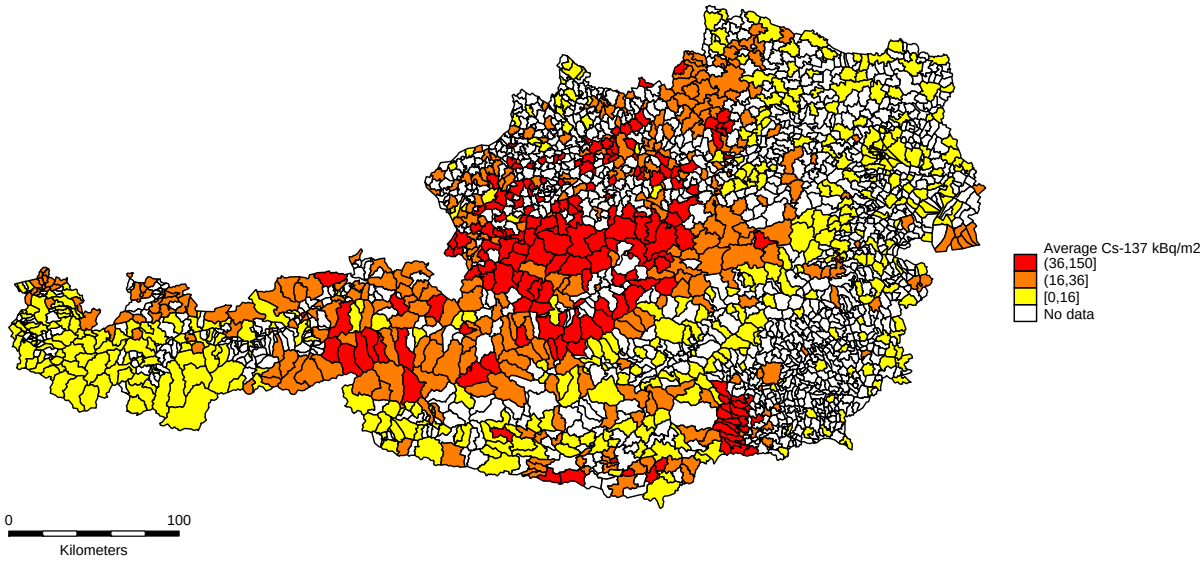
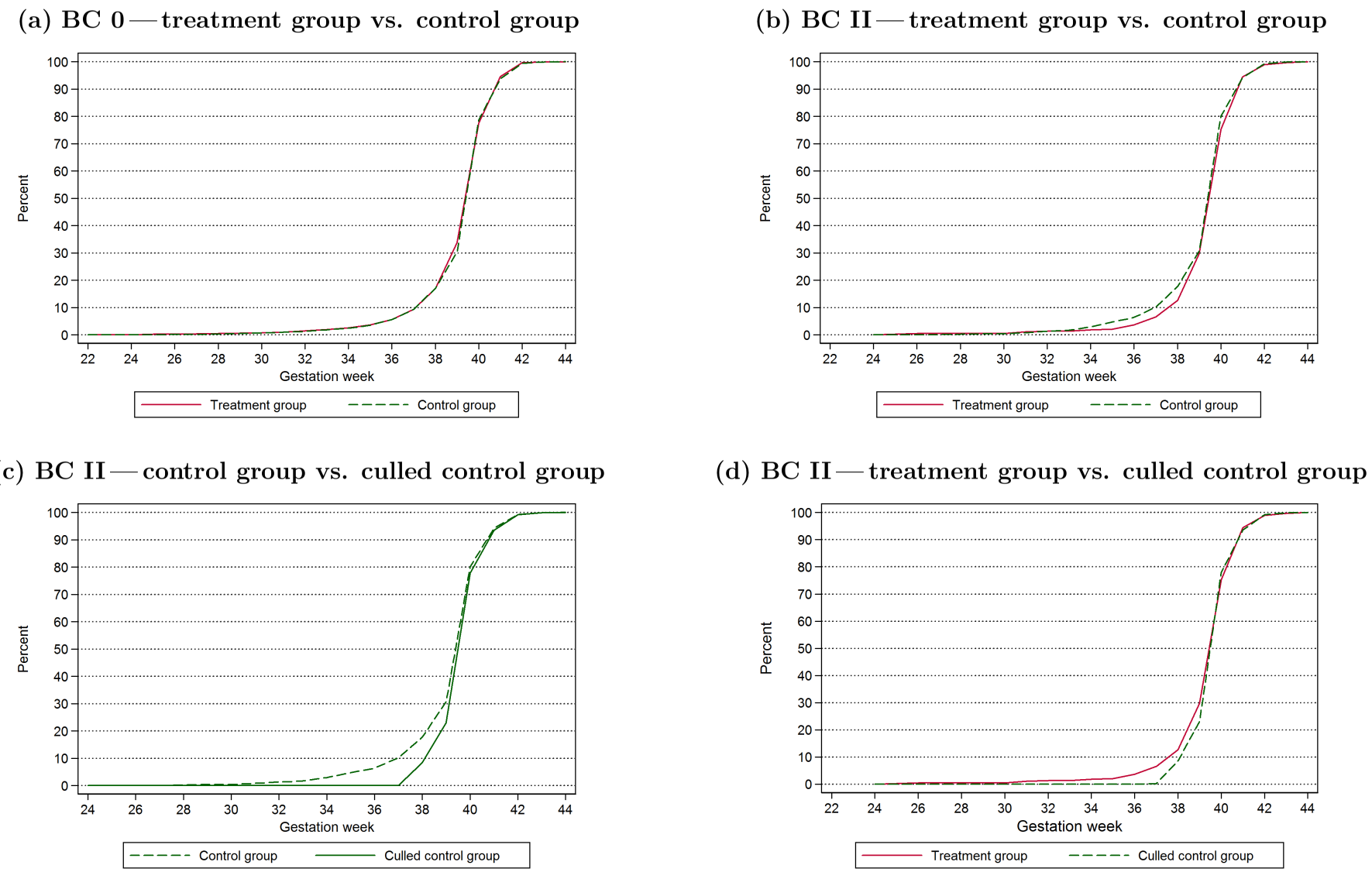


Table 1: Definition of treatment and control groups

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)	72,607
Treatment group 1	$T1$	37 ore more	175	49.1	(12.5)	22,496
Treatment group 2	$T2$	42 ore more	130	54.4	(12.3)	14,812
Treatment group 3	$T3$	47 ore more	93	59.2	(12.5)	9,986
Excluded[a]		between 17 and 36	322	27.3	(5.5)	37,335
		between 17 and 41	367	29.3	(6.6)	45,019
		between 17 and 46	404	30.7	(7.8)	49,845

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



Birth cohort 0 (BC_0) includes all children who were conceived and born before the Chernobyl accident. Birth cohort II (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.

Table 2: Radiation effects on prenatal and postnatal culling

	Low SES				Higher SES			
	Mean	37 kBq	42 kBq	47 kBq	Mean	37 kBq	42 kBq	47 kBq
Prenatal culling								
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	−0.015	−0.005	−0.016
		(0.036)	(0.039)	(0.040)		(0.019)	(0.022)	(0.027)
Postnatal culling								
Alive after 24 hours[d]	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[d]	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[d]	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[d]	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 community-level data (first row) and individual-level data (second and third row) from the *Austrian Birth Register* and the *Austrian Death Register* (fourth to seventh row) 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 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. [d] The dependent variable is equal to one if the child is still alive after the respective time period after birth.

Table 3: 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
Preterm birth[a]								
Culling & scarring	0.056	−0.029**	−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	0.067	−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	0.065	0.028**	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	0.019	0.023	0.021
		(0.047)	(0.053)	(0.069)		(0.018)	(0.022)	(0.028)
Scarring	9.873	−0.022	−0.042	−0.065				
		(0.044)	(0.051)	(0.067)				
Maternity leave (post)[d]								
Culling & scarring	62.79	−1.807**	−1.543	−1.570	62.59	−0.807**	−0.801	−0.917*
		(0.860)	(1.059)	(1.199)		(0.400)	(0.519)	(0.537)
Scarring	62.71	−0.256	0.007	−0.016				
		(0.831)	(1.037)	(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 4: 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								
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)								
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)
Preterm birth								
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								
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								
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* 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. See notes to Tables 2 and 3 for a description of outcome variables.

Table 5: 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	0.009	0.010*	0.008	0.005	0.006	−0.001	−0.001	0.004
		(0.007)	(0.008)	(0.009)		(0.003)	(0.004)	(0.005)
2 yrs after	0.110	−0.031	−0.058***	−0.065**	0.113	−0.007	−0.004	−0.003
		(0.029)	(0.022)	(0.025)		(0.010)	(0.014)	(0.017)
3 yrs after	0.210	−0.010	−0.019	−0.026	0.233	0.004	−0.005	−0.004
		(0.031)	(0.032)	(0.036)		(0.016)	(0.018)	(0.021)
4 yrs after	0.296	−0.035	−0.051	−0.067	0.329	0.023	0.007	−0.001
		(0.034)	(0.036)	(0.042)		(0.019)	(0.020)	(0.024)
5 yrs after	0.368	−0.051	−0.071	−0.103**	0.401	0.018	−0.007	−0.019
		(0.038)	(0.044)	(0.051)		(0.021)	(0.021)	(0.026)
6 yrs after	0.429	−0.077*	−0.099**	−0.112**	0.460	0.027	−0.007	−0.017
		(0.043)	(0.048)	(0.054)		(0.026)	(0.024)	(0.029)
7 yrs after	0.485	−0.090*	−0.104**	−0.123**	0.510	0.040*	0.016	0.010
		(0.046)	(0.053)	(0.058)		(0.024)	(0.027)	(0.033)
8 yrs after	0.528	−0.099**	−0.119**	−0.162***	0.544	0.035	−0.003	−0.007
		(0.049)	(0.054)	(0.061)		(0.028)	(0.028)	(0.035)
9 yrs after	0.565	−0.085*	−0.110*	−0.149**	0.572	0.032	−0.005	−0.011
		(0.051)	(0.058)	(0.064)		(0.027)	(0.028)	(0.034)
10 yrs after	0.595	−0.087	−0.111*	−0.150**	0.597	0.027	−0.016	−0.013
		(0.056)	(0.063)	(0.071)		(0.030)	(0.030)	(0.038)
11 yrs after	0.619	−0.103*	−0.132**	−0.167**	0.619	0.026	−0.016	−0.014
		(0.056)	(0.062)	(0.070)		(0.032)	(0.031)	(0.038)
12 yrs after	0.639	−0.106*	−0.137**	−0.172**	0.632	0.020	−0.022	−0.027
		(0.056)	(0.063)	(0.070)		(0.032)	(0.030)	(0.038)
13 yrs after	0.655	−0.099*	−0.125*	−0.156**	0.643	0.018	−0.027	−0.034
		(0.058)	(0.066)	(0.076)		(0.035)	(0.031)	(0.039)
14 yrs after	0.668	−0.104*	−0.138**	−0.168**	0.652	0.019	−0.026	−0.032
		(0.059)	(0.066)	(0.077)		(0.035)	(0.032)	(0.041)
15 yrs after	0.677	−0.117**	−0.148**	−0.178**	0.660	0.023	−0.024	−0.036
		(0.059)	(0.067)	(0.078)		(0.035)	(0.032)	(0.041)
16 yrs after	0.685	−0.118*	−0.155**	−0.181**	0.665	0.022	−0.026	−0.036
		(0.060)	(0.067)	(0.078)		(0.036)	(0.033)	(0.042)
17 yrs after	0.692	−0.118*	−0.155**	−0.176**	0.669	0.025	−0.021	−0.029
		(0.062)	(0.069)	(0.081)		(0.037)	(0.034)	(0.043)
18 yrs after	0.697	−0.120*	−0.157**	−0.177**	0.672	0.030	−0.017	−0.028
		(0.062)	(0.069)	(0.081)		(0.037)	(0.034)	(0.043)
19 yrs after	0.701	−0.120*	−0.160**	−0.180**	0.675	0.032	−0.018	−0.028
		(0.062)	(0.069)	(0.081)		(0.037)	(0.034)	(0.043)
20 yrs after	0.703	−0.123**	−0.162**	−0.179**	0.676	0.030	−0.020	−0.030
		(0.062)	(0.070)	(0.082)		(0.037)	(0.034)	(0.043)
Obs.		24,554	22,824	21,635		68,544	62,742	59,206

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 a mother has born until 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. (The results are robust to including indicators for maternal age.) 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 6: 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	0.011	0.106	−0.001	−0.011	−0.015
		(0.019)	(0.022)	(0.025)		(0.012)	(0.014)	(0.016)
2 yrs after	0.355	0.002	0.014	0.000	0.386	−0.039**	−0.030	−0.028
		(0.032)	(0.034)	(0.043)		(0.017)	(0.022)	(0.025)
3 yrs after	0.372	0.011	0.009	−0.003	0.411	−0.021	−0.010	−0.003
		(0.028)	(0.033)	(0.040)		(0.017)	(0.021)	(0.025)
4 yrs after	0.400	0.026	0.036	0.041	0.449	−0.011	−0.026	−0.023
		(0.034)	(0.039)	(0.049)		(0.018)	(0.020)	(0.026)
5 yrs after	0.449	0.078**	0.087**	0.083*	0.489	−0.018	0.006	0.026
		(0.038)	(0.039)	(0.048)		(0.020)	(0.020)	(0.025)
6 yrs after	0.470	0.067*	0.085**	0.071*	0.516	−0.018	0.010	0.017
		(0.040)	(0.036)	(0.042)		(0.019)	(0.020)	(0.025)
7 yrs after	0.487	0.060*	0.064*	0.051	0.536	−0.040***	−0.040**	−0.044*
		(0.036)	(0.038)	(0.044)		(0.015)	(0.018)	(0.023)
8 yrs after	0.508	0.027	0.035	0.041	0.560	−0.039**	−0.054***	−0.069***
		(0.035)	(0.035)	(0.042)		(0.018)	(0.020)	(0.023)
9 yrs after	0.531	0.020	0.030	0.050	0.590	−0.018	−0.031	−0.035
		(0.035)	(0.034)	(0.038)		(0.017)	(0.021)	(0.025)
10 yrs after	0.557	−0.003	0.011	0.010	0.619	−0.014	−0.007	−0.016
		(0.038)	(0.039)	(0.047)		(0.018)	(0.025)	(0.029)
11 yrs after	0.581	0.046	0.062	0.051	0.645	−0.007	0.002	−0.009
		(0.035)	(0.040)	(0.050)		(0.018)	(0.023)	(0.027)
12 yrs after	0.602	0.078**	0.101***	0.087*	0.669	−0.001	−0.004	−0.006
		(0.038)	(0.039)	(0.045)		(0.016)	(0.021)	(0.025)
13 yrs after	0.620	0.049	0.069*	0.068	0.690	−0.003	−0.010	−0.022
		(0.032)	(0.038)	(0.044)		(0.017)	(0.022)	(0.026)
14 yrs after	0.635	0.026	0.048	0.046	0.710	−0.002	0.001	0.003
		(0.030)	(0.036)	(0.040)		(0.016)	(0.020)	(0.024)
15 yrs after	0.653	0.033	0.053	0.062	0.733	0.001	0.001	−0.000
		(0.030)	(0.036)	(0.042)		(0.016)	(0.021)	(0.024)
16 yrs after	0.666	0.016	0.025	0.033	0.750	−0.000	−0.003	−0.011
		(0.028)	(0.034)	(0.038)		(0.016)	(0.021)	(0.025)
17 yrs after	0.671	0.041	0.035	0.038	0.761	0.014	0.016	0.016
		(0.029)	(0.037)	(0.044)		(0.014)	(0.019)	(0.021)
18 yrs after	0.671	0.056*	0.066*	0.069	0.766	0.010	0.013	0.011
		(0.031)	(0.037)	(0.045)		(0.013)	(0.017)	(0.020)
19 yrs after	0.669	0.066**	0.074*	0.078*	0.768	−0.005	0.002	0.001
		(0.031)	(0.038)	(0.046)		(0.014)	(0.018)	(0.020)
20 yrs after	0.663	0.060**	0.054*	0.066*	0.769	0.004	0.006	0.001
		(0.027)	(0.033)	(0.039)		(0.014)	(0.018)	(0.021)
Obs.		719,541	668,484	632,803		2,053,657	1,879,809	1,773,200
No. mothers		23,211	21,564	20,413		66,247	60,639	57,200

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, 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 7: 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	−0.016	−0.028**	−0.028**	0.025	−0.002	−0.007	−0.007
		(0.011)	(0.011)	(0.013)		(0.005)	(0.006)	(0.007)
Age 16	0.475	−0.069**	−0.079*	−0.082*	0.241	0.040	0.041*	0.056*
		(0.033)	(0.041)	(0.046)		(0.028)	(0.024)	(0.030)
Age 17	0.591	−0.063*	−0.099**	−0.097**	0.309	0.027	0.039	0.042
		(0.037)	(0.040)	(0.044)		(0.028)	(0.024)	(0.031)
Age 18	0.613	−0.063	−0.087**	−0.077*	0.325	0.023	0.030	0.037
		(0.042)	(0.041)	(0.046)		(0.025)	(0.025)	(0.034)
Age 19	0.585	−0.053	−0.073*	−0.050	0.319	−0.001	0.000	0.000
		(0.037)	(0.039)	(0.043)		(0.021)	(0.023)	(0.029)
Age 20	0.581	−0.095**	−0.131***	−0.106**	0.358	0.000	−0.013	0.013
		(0.038)	(0.040)	(0.044)		(0.021)	(0.024)	(0.031)
Age 21	0.671	−0.019	−0.030	−0.014	0.458	0.012	0.027	0.032
		(0.035)	(0.041)	(0.047)		(0.026)	(0.026)	(0.027)
Age 22	0.711	−0.066*	−0.086**	−0.096**	0.513	0.007	0.009	0.015
		(0.035)	(0.041)	(0.048)		(0.024)	(0.027)	(0.026)
Age 23	0.727	−0.050	−0.059	−0.080*	0.552	0.008	−0.001	0.017
		(0.033)	(0.037)	(0.044)		(0.021)	(0.027)	(0.031)
Obs.		143,370	133,119	125,766		452,160	413,316	389,538

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. 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. 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 8: 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	−0.039	−0.035	−0.050	0.191	0.043	0.047*	0.062**
		(0.033)	(0.040)	(0.044)		(0.029)	(0.024)	(0.029)
Age 17	0.487	−0.076**	−0.076*	−0.088**	0.248	−0.003	0.014	0.024
		(0.034)	(0.040)	(0.044)		(0.030)	(0.023)	(0.028)
Age 18	0.489	−0.033	−0.033	−0.021	0.257	0.013	0.027	0.032
		(0.037)	(0.042)	(0.046)		(0.028)	(0.024)	(0.033)
Obs.		143,370	133,119	125,766		452,160	413,316	389,538
Income								
Age 15-23 (wage sum)[b]	9.534	−0.509	−0.722**	−0.913**	7.729	−0.052	−0.110	0.005
		(0.321)	(0.324)	(0.387)		(0.205)	(0.253)	(0.261)
Age 15-23 (total wage sum)[c]	9.702	−0.534*	−0.755**	−0.947**	7.588	−0.044	−0.099	0.034
		(0.323)	(0.325)	(0.388)		(0.210)	(0.258)	(0.263)
Obs.		15,930	14,791	13,974		50,240	45,924	43,282

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 15-23). 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.