

WORKING PAPERS

N° TSE -1019

June 2019

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# **The Effect of Prenatal Exposure to Radiation on Birth Outcomes: Exploiting a Natural Experiment in Taiwan**

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May 2016

## **Abstract**

We estimate the effect of prenatal exposure to radiation on infant health. By exploiting the 1983 Taiwanese radiation-contaminated buildings (RCBs) accident as a natural experiment, we compare birth outcomes between siblings and cousins exposed to different radiation levels. Given the 1983 accident was unanticipated and exposed cohorts were unaware of the risk until 1992, our design isolates the effect of radiation exposure during pregnancy from other effects. We provide the first evidence that prenatal exposure to a continuous low-level dose of radiation significantly reduces gestational length and increases the probabilities of prematurity and low birth weight.

JEL: I10, I18, J13

## **Abstract**

This paper estimates the causal effect of *in utero* exposure to radiation on infant health. By exploiting the Taiwanese radiation-contaminated buildings (RCBs) accident in 1983 as a natural experiment, we compare birth outcomes between siblings and cousins who were exposed to different levels of radiation, before and after the accident. The identification strategy relies on exogenous variation in doses of radiation and timing while *in utero* as well as different birth cohorts. Given the 1983 accident was unanticipated and the exposed cohorts were completely unaware of the risk until 1992, our design successfully isolates the impact of radiation exposure during pregnancy from effects due to endogenous migration, maternal stress and other avoidance behaviors. We provide direct evidence that prenatal exposure to a higher level of radiation significantly reduces gestational length and increases the probabilities of prematurity and low birth weight. The effects are stronger for boys than for girls, which is in line with the literature on “fragile males”.

## 1. Introduction

A growing literature has highlighted that endowments at birth are influenced by environmental shocks. One line of research focuses on the impacts caused by natural disasters, such as influenza epidemics (Almond et al, 2006), famine (Chen and Zhou, 2007; Almond et al., 2010), earthquakes (Torche, 2011), hurricanes (Currie and Rossin-Slater, 2013), and extreme weather (Fuller, 2014). More recently, an emerging literature emphasizes the threats posed by environmental toxins and pollution, such as exposure to dust, air pollution, water pollution, and radioactive fallout (Almond et al., 2009; Almond, et al., 2013; Black et al., 2013; Currie and Schmieder, 2009; Currie and Schwandt, 2014; Currie et al., 2014). Other studies focus on stressful events, including armed conflict (Akresh et al., 2012), terrorist attacks (Berrebi and Ostwald, 2015), and economic crises (Bozzoli and Quintana-Domeque, 2014). One key challenge in these studies is the difficulty of identifying the causal relationship between exposure to shocks during pregnancy and fetal health. Among potential underlying mechanisms, nutritional deficits and especially maternal stress are the channels most often argued to affect birth outcomes.<sup>1</sup>

We use the radiation-contaminated buildings (RCBs) accident in Taiwan as a natural experiment to explore how infant health responds to *in utero* exposure to radiation. The RCBs accident occurred more than 30 years ago. In 1982, some cobalt-60 (Co-60) contaminated steel bars were used in construction, leading to radiation contamination of more than 180 buildings. Most of these buildings were completed in 1983 and located in northern Taiwan. Ultimately, more than 1600 radiation-contaminated apartments were documented by the Taiwanese Atomic Energy Council (AEC) and approximately 10,000 residents were exposed to elevated doses of radiation.<sup>2</sup> This event was not disclosed until 1992 and subsequently raised widespread public concern regarding the possible health consequences of long-term exposure to radiation. A small medical literature on the RCBs cohort has demonstrated that exposure to radiation is associated with reduced fertility, higher probability of depression, and increased risk of ocular lens opacity and certain cancers (Hwang et al., 2006; Hwang et al., 2008; Lin et al., 2010; Hsieh et al., 2010; Yen et al. 2014). To date, the question of whether *in utero* exposure has any adverse effects on the next generation has not been investigated.

There are only two studies in the economics literature that use quasi-experimental techniques to study the short-term and long-term consequences of *in utero* exposure to radiation. In pioneering work, Almond et al. (2009) studied the

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<sup>1</sup> There is evidence that maternal stress tends to affect birth weight especially during the first trimester of pregnancy (Torche, 2011), while nutritional changes have their greatest effect during the third trimester (Almond et al., 2011).

<sup>2</sup> These apartments were contaminated with cobalt-60 at a total activity ranging from 1–140  $\mu\text{Sv}/\text{yr}$ .

impact of prenatal exposure to radioactive fallout from Chernobyl on compulsory schooling performance in Sweden. They demonstrate that exposure to ionizing radiation between weeks 8 and 25 significantly reduces the likelihood of earning qualifying high school and mathematics scores, but detect no health effects measured by birth outcomes and childhood hospitalizations. Following a similar empirical strategy, Black et al. (2013) take advantage of variation in radiation exposure in Norway due to nuclear weapon testing. They find radiation exposure leads to a decline in IQ scores and reductions in years of completed schooling and adult earnings.

This study contributes to the existing literature in several ways. First, given that unexpected radiation exposure is rare, the Taiwanese RCBs accident offers a rare opportunity to estimate the causal impacts of radiation exposure *in utero* on birth outcomes. Second, unlike prior studies using geographical identification of the stressful events, our measure of exposure is at the individual level, which increases precision and provides stronger evidence of causality. Third, as exposed women were completely unaware of the exposure from its beginning in 1983 until 1992, our estimates are not susceptible to potential biases arising from maternal stress or avoidance behaviors. Moreover, endogenous residential sorting is unlikely to create any bias. Finally, comparisons of birth outcomes between siblings and cousins allow us to control for unobserved mother and family characteristics from the same family of origin. To the best of our knowledge, this is the first study that makes a direct causal inference between prenatal exposure to radiation and fetal health, based on a cohort that was unaware of the risk for almost a decade after gestation. Our results verify that children prenatally exposed to more radiation have lower gestation age and are more likely to be born preterm and suffer low birth weight. Moreover, boys are more vulnerable to the exposure than girls.

The remainder of the paper is organized as follows. A description of the related literature is provided in Section 2. Section 3 describes the specification of the empirical models. The empirical results are described in Section 4, with conclusions in Section 5.

## **2. Related Literature**

Whether exposure to radiation *in utero* causes any adverse effects on birth outcomes has long been a subject of interest. The potential effects of radiation exposure *in utero* include spontaneous abortion, intrauterine growth restriction, mental retardation, organ malformation, and childhood cancer, with the impacts depending on the stage of fetal development at the time of exposure and the amount of radiation the fetus is exposed to. It is generally accepted that the fetus is most susceptible to radiation

during organogenesis from 2 to 7 weeks and in the early fetal period from 8 to 15 weeks after conception. During the second period, when germ cell layers start to develop into internal organs, exposure to radiation could impede growth in organs and the brain, resulting in growth retardation or malformation. After the 26<sup>th</sup> week of pregnancy, fetuses are less sensitive to radiation and the likelihood of birth defects is smaller since most of the organs and body functions are fully developed (McCollough, 2007; Williams and Fletcher, 2010). However, exposure to radiation in later stages of pregnancy could still lead to mutations and increase the risk of childhood cancer.

In most epidemiological studies, the source of radiation exposure is a medical procedure such as medical or dental x-rays. A common feature of this line of research is the doses are relatively low and, more importantly, the exposures are unlikely to be exogenous. While some studies show exposure to medical radiation during pregnancy increases the risk of having a low-birth-weight child, others reveal no significant relationship. For instance, Hujoel et al. (2004) found that exposure to dental radiography greater than an extremely low level (0.4 mGy) during gestation is associated with an increased risk of low-birth-weight infants, while Mortazavi et al. (2013) cast doubt on this finding.

With regard to the high-dose exposure from nuclear accidents, an earlier study by Miller and Blot (1972) reports that exposure to the atomic bomb explosion in Hiroshima during early pregnancy is associated with offspring of small stature and head circumference, with the likelihood increasing with dosage above a minimum of 10 rad (100 mSv). A series of studies of the Chernobyl event evaluate the impact of prenatal exposure to smaller doses of radiation on a wide range of birth outcomes, including spontaneous abortion, stillbirth, gestational age, birth weight and neonatal mortality. Here again, the evidence is not conclusive. Some studies find effects of prenatal radiation exposure while others do not; in general the effects are small (Ericson and Källén, 1994; Sperling et al., 1994; Auvinen et al., 2001). Nevertheless, a few papers do suggest that prenatal exposure to radioactive fallout after Chernobyl resulted in fetal death and detectable cognitive damage in childhood (Nyagu et al., 2004; Almond et al., 2009).

### **3. Data**

This study combines three high-quality administrative datasets: the residential record of RCBs, national birth certificate records for 1980-91, and the household register in 2006. The residential record of RCBs contains information on the residential address, along with gender, educational attainment, birth year, and the cumulative radiation dose for each member of the household. Comprehensive details on children at birth are provided in the birth records data, including gender, gestational age, birth weight,

birth order, and multiple birth status. The household register contains multigenerational information about households, including gender, birth year, educational attainment and relationship to the head of household for each household member. Based on unique personal identifiers, we are able to link the three datasets.

We limit our observations to single births and construct two unique samples for analysis. First, we focus on a sample of RCBs mothers who had at least one child who was prenatally exposed to radiation (after 1983) and one child who was not (before 1983). The resulting siblings sample consists of 191 births to 80 exposed mothers. As the sample is small and restricted to exposed mothers only, it may limit the generalizability of our results. Therefore, we extend our analysis by including births to the non-exposed sisters of RCBs women and including exposed births to RCBs women who did not bear any non-exposed children (i.e., bore children in 1983 or later but not before). Specifically, we merge the residential records of RCBs with the household register to identify the non-exposed sisters and trace their fertility history by linking to birth registers. This yields 402 births to 213 exposed mothers and 451 births to their 262 non-exposed sisters, a total of 853 births.

### **Measurement of Radiation Exposure**

To assess the health consequences of exposure to radiation for RCBs residents, a comprehensive epidemiological study was begun in 1995 by the National Health Research Institute (NHRI) in Taiwan. The residential record of RCBs contains an indicator of cumulative excess radiation dose taken from the Taiwan Cumulative Dose (TCD) exposure assessment system. The assessment program incorporates the concept of “highly occupied zone” into the model proposed by the US National Institute for Occupational Safety and Health Advisory Committee for contaminated buildings with multiple radioactive sources. The RCB residents were required to wear thermoluminescent dosimeter (TLD)-embedded chains, belts and badges to measure the external dose equivalent, adjusted by their height and their daily activity in every living space such as the bedroom, living room, and kitchen. The annual excess dose equivalent for every resident by year was estimated by multiplying the estimated daily mean dose by days and months living in the RCB, accounting for the decay rate of Cobalt-60 (a half-life of 5.27 years) and subtracting the annual natural background dose in Taiwan (2 mSv/yr). The cumulative above-background radiation dose for each resident was estimated by totaling the annual excess dose through years. Epidemiological and biomedical studies have indicated that TCD is a reliable method of assessing radiation exposure (Hwang et al., 2006; Hwang et al., 2008).

## **4. Empirical Strategies**

We adopt two empirical strategies. Restricting attention to the births to RCBs mothers, our first strategy combines difference-in-differences methods with mother fixed effects, which controls for any fixed unobservable differences between mothers (such as genetics). With variation in dosage of radiation exposure, we identify the impact of radiation exposure on health outcomes at birth by using the sample of births to mothers having at least one child prenatally exposed to radiation in 1983 or after and one child born earlier, who was not exposed. In such a fixed-effect framework, identification derives from the between-sibling difference in gestational exposure associated with the mother living or not living in an RCB during the prenatal period. We estimate the following model:

$$Birth_{ijt} = \beta_0 + \beta_1 Exposure_{ijt} + \beta_2 TCD_{ijt} + \beta_3 TCD_{ijt} \times Exposure_{ijt} + \pi' X_{ijt} + \alpha_j + \varepsilon_{ijt} \quad (1)$$

where  $Birth_{ijt}$  represents alternatively outcomes such as birth weight, gestational age, prematurity, and low birth weight for child  $i$  born to mother  $j$  at time  $t$ .  $Exposure_{ijt}$  is an indicator variable that takes the value 1 for the cohort born in or after 1983 and 0 otherwise.  $TCD_{ijt}$  is a continuous variable measuring the cumulative dose of radiation exposure for exposed mothers. The main effect of interest is the coefficient of the interaction between  $Exposure$  and  $TCD$ .  $X_{ijt}$  is a vector of control variables that includes parents' age and years of education, the child's gender, birth order, and dummies for birth year, birth month, and Taipei residence. The  $\alpha_j$  are mother fixed effects. As the effect of radiation might be nonlinear, we alternatively replace the continuous variable  $TCD_{ijt}$  with two categorical variables:  $TCD_{low}$  is a dummy variable for exposure levels  $1 \leq TCD \leq 10$  mSV and  $TCD_{high}$  is a dummy for exposure levels  $TCD > 10$  mSV. The reference group is exposures  $TCD < 1$  mSV, which is the accepted limit for the general public. In this specification, our interest is in the coefficients of the interactions  $TCD_{low} \times Exposure$  and  $TCD_{high} \times Exposure$ :

$$Birth_{ijt} = \beta_0 + \beta_1 Exposure_{ijt} + \beta_2 TCD_{low_{ijt}} + \beta_3 TCD_{high_{ijt}} + \beta_4 TCD_{low_{ijt}} \times Exposure_{ijt} + \beta_5 TCD_{high_{ijt}} \times Exposure_{ijt} + \pi' X_{ijt} + \alpha_j + \varepsilon_{ijt} \quad (2)$$

To evaluate whether the effect is temporary or persistent during the post-accident period, we also use two period dummies:  $Exposure1$  (1983-1986) and  $Exposure2$  (1987-1991) and their interactions with TCD measures as an alternative specification:



$$\begin{aligned}
Birth_{ijt} = & \beta_0 + \beta_1 Exposure1_{ijt} + \beta_2 Exposure2_{ijt} + \beta_3 TCD\_low_{ijt} \\
& + \beta_4 TCD\_high_{ijt} + \beta_5 TCD\_low_{ijt} \times Exposure1_{ijt} \\
& + \beta_6 TCD\_low_{ijt} \times Exposure2_{ijt} + \beta_7 TCD\_high_{ijt} \times Exposure1_{ijt} \\
& + \beta_8 TCD\_high_{ijt} \times Exposure2_{ijt} + \pi' X_{ijt} + \alpha_j \\
& + \varepsilon_{ijt} \qquad (3)
\end{aligned}$$

A limitation of this design is that only RCBs mothers are included, the exposed child is born after the unexposed child, and the sample size is relatively small. To provide a valid and homogeneous control group, our second strategy is to apply the difference-in-differences methodology to an extended sample that includes births to RCBs women and to their non-exposed sisters. By including fixed effects for the grandmother of the children, we control for some unobservable characteristics across families and compare children who were exposed to radiation *in utero* to their non-exposed siblings and cousins. It is worth noting that the RCBs mothers may differ in important, unobserved ways from their non-exposed sisters and these unobserved factors may be responsible for the observed effects on offspring.

## 5. Results

Table 1 presents summary statistics on characteristics of the RCBs mothers and birth outcomes of their children for our first sample. Outcomes of interest are birth weight, gestational age, and incidence of prematurity and low birth weight. As the table shows, the mean birth weight was 3290 g and the gestational period was 39.6 weeks. Only 2.6% of births were classified as preterm (< 37 weeks) or low birth weight (< 2500 g). On average, the mothers' age at birth is 27 years and their husbands are two years older. Mothers had on average 10.7 years of education, while their husbands had slightly more, 11.4 years. The average cumulative excess dose measured as TCD for RCBs women is about 64 mSv. The distribution of excess dose is: TCD < 1 mSV, 28%;  $1 \leq TCD \leq 10$  mSV, 19%; and TCD > 10 mSV, 53%.

Table 2 compares the characteristics and outcomes of the exposed mothers and their children with those of their non-exposed sisters and their children. The average birth weight of newborns for RCBs women (3255g) was significantly smaller than that of their non-exposed sisters' children (3304g). In each sample, only 3.5% of births were classified as low birth weight. The gestational period was slightly shorter for children of RCBs mothers (39.6 weeks) than those of their non-exposed sisters (39.7 weeks) and the proportion of preterm births is one percentage point higher for births of RCBs mothers (2%) than those of their non-exposed sisters (1%). Given the majority of RCBs were located in Taipei area, it is not surprising to find that about 74% of the exposed cohort are Taipei residents; the corresponding figure for their

non-exposed sisters is much smaller, about 47%. The RCBs women are also more educated than their non-exposed sisters.

We start our investigation on the sample of births to RCBs women and compare children exposed to radiation *in utero* with their non-exposed siblings. Table 3 estimates the effects using OLS with mother fixed effects. To account for potential confounding factors, we estimate the model with adjustment for child's sex, birth order, birth year, and birth month as well as socioeconomic characteristics of the parents including the mother's and father's education, age at child's birth, and Taipei residence. The inclusion of mother fixed effects allows us to control for time-invariant unobservable characteristics of the mother that do not vary across siblings. Identification is thus driven by comparing children of the same mother, each of whom was exposed to different levels of radiation exposure.

As shown in Table 3, the estimated effects depend on the measure of radiation exposure. Using a continuous measure of TCD reveals no significant effect for any of the measures of birth outcomes. In contrast, using the categorical measure shows that children exposed to a higher level of radiation *in utero* (TCD > 10 mSV) have significantly shorter length of gestation and a higher chance to be preterm and low birth weight, while those exposed to a lower level of radiation were not significantly affected.<sup>3</sup> The estimated magnitudes are non-trivial: fetuses exposed to a higher level of radiation *in utero* have: 1 week shorter gestation, 10 percentage points higher probability of prematurity and 12 percentage points higher probability of low birth weight. Distinguishing births by the time since the contamination accident (Table 4) reveals the effects on birth outcomes are evident only in the first post-accident period (1983-1986) and not in the second period (1987-1991). This may be driven in part by the decay of cobalt-60.

The analysis using only births to RCBs women suffers from small sample size and the confounding between radiation exposure and birth order (because non-exposed siblings were born before 1983). To overcome these limitations, we utilize an extended sample including births to non-exposed sisters of the RCBs mothers for analysis. We use difference-in-differences models with grandmother fixed effects. The identification strategy rests on the assumption that there are no alternative paths of influence, other than exposure to radiation, that have detrimental effects on birth outcomes. One concern is that if mothers of "higher quality" (who take more precautionary measures before or during pregnancy) are more likely to reside in Taipei and also have favorable birth outcomes, this would partially offset the effect of prenatal exposure to radiation on birth outcomes and our results would be

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<sup>3</sup> Due to a substantial reduction of sample size on same-sex siblings, we cannot precisely estimate whether boys or girls are more affected by radiation exposure in utero.

underestimates. Alternatively, if women in Taipei are more likely to be employed, causing maternal stress during pregnancy, our analysis may overestimate the effect of exposure to radiation. Smaller birth weights among RCBs mothers than their non-exposed sisters prior to the start of exposure suggests that the RCBs mothers do not tend to have more favorable birth outcomes, and so the first hypothesis may be discounted. With regard to stress at work, we have no information on work status and hence cannot control for it. However, we include controls for mother's schooling years and mother fixed effects, which partly capture the socioeconomic status of mothers.

Similar to the analysis using RCBs mothers only, when using TCD as a continuous measure of exposure to radiation, we do not detect any significant effects of radiation exposure on measures of birth outcomes. By using the categorical exposure variables, however, we find evidence that exposure to high levels of radiation *in utero* reduces gestational age, as reported in Table 5.

Some recent work suggests that male fetuses are more fragile than female fetuses under poor intrauterine conditions (Kraemer, 2000; Sanders and Stoecker, 2015). To investigate this possibility, we estimate separate regressions for male and female children in Table 6. We find that boys do suffer more than girls. *In-utero* exposure to radiation leads to a significant reduction in gestational length and an increase the probability of prematurity for boys, but not for girls. Consistent with earlier estimates, the effect is evident only for the period 1983-1986 and not for the period 1987-1991.

As a robustness test, we use 50 mSv as an alternative cutoff to distinguish between high and low levels of radiation exposure. This alternative reduces the fraction of children of RCBs mothers categorized as having high exposure from 53% to 20%.<sup>4</sup> Compared with our previous results, the effects on gestational age and probability of prematurity are smaller. However, the main findings are unaltered. At this higher level of radiation exposure, the effect on incidence of low birth weight remains evident only for boys, confirming the 'fragile male' effect.

There are several limitations and potential caveats to our analysis. First, as the most vulnerable pregnancies could be lost due to radiation exposure, selection bias arising from miscarriages and stillbirths may result in underestimation of the effect of prenatal exposure to radiation. Second, we may still suffer from omitted-variable bias due to lack of indicators on pregnancy-related health problems and time-varying maternal behaviors during pregnancy such as smoking and alcohol consumption. Third, as the exposure is continuous on a daily basis, we are not able to estimate how birth outcomes are affected by exposure during different trimesters. Fourth, a potential concern we cannot address is the possibility of changes in mother's residential

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<sup>4</sup> To save space, we do not report the results in a table. Results are available from authors upon request.

location. Since the accident was not disclosed until 1992, there was no obvious reason to believe that RCBs residents would move out in response to the shock and this bias is likely to be small or negligible. Lastly, as birth weight and gestation are crude measures of health at birth, more sensitive indicators such as birth complications or birth defects should be considered in future analysis.

## 6. Conclusions

The novelty of this study is combining exposure measured at the individual level with a compelling identification strategy. By exploiting the RCBs accident in Taiwan as a natural experiment and comparing birth outcomes between siblings and cousins who were exposed to different levels of radiation, we identify the impacts of *in utero* exposure to radiation on infant health. Given that the accident was unexpected and the lack of awareness about the exposure, our estimates are unlikely to be affected by biases arising from endogenous exposure, residential sorting, or maternal stress and avoidance behaviors.

We provide evidence that infants who were exposed to a relatively higher dose of radiation (TCD >10 mSV) *in utero* had a higher probability to be low birth weight, which is mediated by shorter gestational age and higher incidence of prematurity rather than by affecting the intrauterine growth of term infants. This finding is contrary to the evidence in Sweden by Almond et al. (2009), who argue that the effect of radioactive fallout from Chernobyl is subclinical. While few studies have demonstrated that *in utero* radiation exposure causes cognitive damage, we are the first to provide evidence of a causal effect of *in utero* radiation exposure on health at birth. We also find that boys are more vulnerable than girls, which is consistent with the hypothesis of ‘fragile males’ in the literature.

Enhanced knowledge about the effects of radiation exposure on public health will allow countries to clarify the direct and indirect costs of such accidents. While we have identified short-run health consequences of prenatal radiation exposure on infants, future work should investigate the possibility of effects on long-run outcomes such as educational attainment, adult health and earnings. Further understanding of the possible existence and magnitude of a threshold exposure level below which adverse effects are not present is also important to formulate effective policies to shield pregnant women and their offspring from the consequences of radiation exposure.

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Table 1 Summary Statistics of RCBs Mothers and Their Children  
(DD with Mother Fixed Effects)

	Obs	Mean	Std. Dev.
<b><u>Birth Outcomes</u></b>			
Birth weight	191	3289.71	471.21
Low birth weight (birth weight<2500g)	191	0.026	0.16
Gestational age	191	39.59	1.27
Prematurity (gestational age<37 weeks)	191	0.026	0.16
<b><u>Parental Characteristics</u></b>			
Mother's birth year	191	1956.21	3.11
Mother's years of schooling	191	10.70	3.27
Mother's age at child's birth	191	27.10	3.78
Father's years of schooling	191	11.42	3.86
Father's age at child's birth	191	29.32	4.01
<b><u>Measures of radiation exposure</u></b>			
$0 \leq \text{TCD} < 1$	191	0.28	0.45
$1 \leq \text{TCD} < 10$	191	0.19	0.39
$10 \leq \text{TCD}$	191	0.53	0.50
<b><u>Children Characteristics</u></b>			
Boy	191	0.48	0.50
Parity	191	1.93	0.90
Born in Taipei	191	0.75	0.43
Child's birth year	191	1983.31	2.69
Exposure $\geq$ 1983	191	0.54	0.50
$1983 \leq \text{Exposure} \leq 1986$	191	0.40	0.49
$1987 \leq \text{Exposure} \leq 1991$	191	0.14	0.35



Table 2 Summary Statistics of RCBs mothers, Their Non-Exposed Sisters and Children  
(DD with Grandmother Fixed Effects)

	Non-exposed sisters of RCBs			RCBs mothers			T test
	Obs	Mean	Std. Dev.	Obs	Mean	Std. Dev.	RCBs—non-RCBs
<b><u>Birth Outcomes</u></b>							
Birth weight	451	3304.14	432.81	402	3254.65	434.05	-49.49**
Low birth weight (birth weight <2500g)	451	0.035	0.19	402	0.035	0.18	-0.00
Gestational age	451	39.73	1.07	402	39.56	1.18	-0.17**
Prematurity (gestational age<37 weeks)	451	0.011	0.10	402	0.020	0.14	0.01
<b><u>Parental Characteristics</u></b>							
Mother's birth year	451	1958.62	4.24	402	1958.41	4.16	-0.21
Mother's years of schooling	451	9.82	3.11	402	10.57	3.22	0.75***
Mother's age at child's birth	451	26.47	3.57	402	26.85	3.83	0.38*
Father's years of schooling	451	10.19	3.14	402	11.15	3.69	0.96***
Father's age at child's birth	451	29.93	4.70	402	29.72	4.13	-0.21
<b><u>Measures of radiation exposure</u></b>							
$0 \leq \text{TCD} < 1$	451	0.00	0.00	402	0.26	0.44	
$1 \leq \text{TCD} < 10$	451	0.00	0.00	402	0.23	0.42	
$10 \leq \text{TCD}$	451	0.00	0.00	402	0.50	0.50	
<b><u>Children Characteristics</u></b>							
Boy	451	0.54	0.50	402	0.52	0.50	-0.012
Parity	451	2.01	1.11	402	1.75	0.84	-0.26***
Born in Taipei	451	0.47	0.50	402	0.74	0.44	0.26***
Children's birth year	451	1985.09	3.47	402	1985.26	3.24	0.17
Exposure $\geq$ 1983	451	0.70	0.46	402	0.77	0.42	0.07***
$1983 \leq \text{Exposure} \leq 1986$	451	0.33	0.47	402	0.40	0.49	0.07**
$1987 \leq \text{Exposure} \leq 1991$	451	0.37	0.48	402	0.37	0.48	0.00

Table 3 The Effect of Prenatal Radiation Exposure on Birth Outcomes – DD with Mother Fixed Effects

	Birth Weight		Low Birth Weight		Gestational Age		Prematurity	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
TCD × Exposure	-0.0878		0.0004		-0.0032		0.00048	
	[0.954]		[0.0004]		[0.003]		[0.00034]	
TCD_low × Exposure		99.576		0.0628		-0.0401		0.0103
		[232.973]		[0.087]		[0.712]		[0.070]
TCD_high × Exposure		-2.85		0.1204*		-0.9024*		0.1007**
		[144.103]		[0.070]		[0.465]		[0.050]
Exposure	-15.0183	-140.3435	-0.1495	-0.23	1.129	1.199	-0.1602	-0.1673
	[435.335]	[415.353]	[0.122]	[0.145]	[1.189]	[1.266]	[0.139]	[0.137]
Control parents' education, age at birth, child's sex, parity, birth year, month and Taipei residence	v	v	v	v	v	v	v	v
Observations	191	191	191	191	191	191	191	191
R-squared	0.721	0.722	0.495	0.511	0.522	0.54	0.563	0.573

Notes: Figures in parentheses are robust standard errors. \*\*\*significant at 1%; \*\*significant at 5%; \*significant at 10%.

Table 4 The Effect of Prenatal Radiation Exposure on Birth Outcomes – DD with Mother Fixed Effects (high vs. low TCD levels, two periods)

	Birth Weight		Low Birth Weight		Gestational Age		Prematurity	
	(3)	(4)	(3)	(4)	(3)	(4)	(3)	(4)
TCD × Exposure1	-0.6722		0.0008		-0.0058		0.0006	
	[1.327]		[0.001]		[0.005]		[0.0005]	
TCD × Exposure2	0.3086		0.0001		-0.0015		0.0004	
	[0.980]		[0.0003]		[0.003]		[0.0003]	
TCD_low × Exposure1		-118.1414		0.0944		-0.4065		0.0417
		[188.603]		[0.075]		[0.652]		[0.057]
TCD_high × Exposure1		-27.3969		0.1192		-0.9392*		0.1071**
		[145.745]		[0.075]		[0.481]		[0.052]
TCD_low × Exposure2		994.2521*		-0.0513		1.4508		-0.1287
		[528.743]		[0.207]		[1.924]		[0.200]
TCD_high × Exposure2		486.0039		0.0818		-0.1107		0.0099
		[307.377]		[0.102]		[1.033]		[0.102]
Exposure1	27.8583	-22.5407	-0.1035	-0.1832	-0.2827	-0.0129	-0.0679	-0.0965
	[182.189]	[235.084]	[0.077]	[0.119]	[0.550]	[0.747]	[0.074]	[0.090]
Exposure2	-106.2385	-810.1036	-0.0839	-0.1642	0.7219	0.1017	-0.139	-0.0511
	[468.032]	[527.672]	[0.129]	[0.187]	[1.302]	[1.807]	[0.148]	[0.177]
Control parents' education, age at birth, child's sex, parity, birth year, month and Taipei residence	v	v	v	v	v	v	v	v
Observations	191	191	191	191	191	191	191	191
R-squared	0.722	0.746	0.501	0.515	0.526	0.549	0.563	0.578

Notes: Figures in parentheses are robust standard errors. \*\*\*significant at 1%; \*\*significant at 5%; \*significant at 10%.

Table 5 The Effect of Prenatal Radiation Exposure on Birth Outcomes – Grandmother Fixed Effects (high vs. low TCDs, two periods)

	<b>Birth Weight</b>	<b>Low Birth Weight</b>	<b>Gestational Age</b>	<b>Prematurity</b>
TCD_r × Exposure1	12.7854 [106.696]	-0.0288 [0.058]	0.3868 [0.245]	-0.0186 [0.019]
TCD_r × Exposure2	-135.07 [118.242]	0.0518 [0.067]	-0.2081 [0.340]	0.0259 [0.040]
TCD_low × Exposure1	-6.1402 [152.899]	-0.0176 [0.048]	0.2861 [0.445]	-0.0262 [0.046]
TCD_high × Exposure1	78.6418 [85.744]	0.0712 [0.044]	-0.4306* [0.246]	0.029 [0.035]
TCD_low × Exposure2	12.5558 [177.967]	-0.0068 [0.058]	0.1602 [0.521]	-0.0592 [0.053]
TCD_high × Exposure2	59.2693 [95.892]	-0.0211 [0.050]	-0.1278 [0.268]	-0.0051 [0.034]
TCD_r	88.9138 [101.128]	-0.0422 [0.056]	-0.2854 [0.245]	0.0065 [0.015]
TCD_low	-24.055 [159.031]	0.0129 [0.051]	-0.5578 [0.449]	0.0627 [0.047]
TCD_high	-198.4316** [77.514]	0.0126 [0.044]	0.1992 [0.221]	0.0116 [0.030]
Exposure1	22.1495 [90.603]	-0.0549 [0.045]	-0.0783 [0.225]	0.0114 [0.027]
Exposure2	-35.572 [95.014]	0.0185 [0.059]	-0.3053 [0.300]	0.0278 [0.038]
Control parents' education, age at birth, child's sex, parity, birth year, birth month and Taipei residence	V	V	V	V
Observations	853	853	853	853
R-squared	0.481	0.339	0.371	0.261

Notes: TCD\_r represents a dummy for RCBs mothers TCD level lower than 1 mSV. Figures in parentheses are robust standard errors. \*\*\*significant at 1%; \*\*significant at 5%; \*significant at 10%.

Table 6 The Effect of Prenatal Radiation Exposure on Birth Outcomes –Grandmother Fixed Effects, by Gender (different TDC levels, different periods)

	Birth Weight		Low Birth Weight		Gestational Age		Prematurity	
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
TCD_r × Exposure1	-2.0062 [161.599]	-193.4598 [235.638]	-0.1307 [0.119]	-0.0848 [0.095]	-0.2492 [0.481]	0.6404* [0.382]	0.0206 [0.037]	-0.0172 [0.025]
TCD_r × Exposure2	-124.5453 [220.717]	-189.5549 [260.029]	-0.1345 [0.121]	0.0814 [0.117]	-0.3167 [0.667]	-0.7772 [0.614]	-0.0001 [0.070]	0.1043 [0.092]
TCD_low × Exposure1	267.1282 [437.902]	83.1182 [185.744]	-0.1348 [0.146]	0.0307 [0.054]	0.5474 [1.776]	0.6647* [0.397]	-0.1189 [0.168]	0.0024 [0.016]
TCD_high × Exposure1	-50.2016 [179.939]	13.033 [158.409]	0.107 [0.066]	0.051 [0.054]	-1.3422** [0.641]	-0.1084 [0.355]	0.1808* [0.096]	0.0045 [0.015]
TCD_low × Exposure2	728.5825 [476.486]	-311.0185 [220.274]	-0.2437 [0.166]	0.1161 [0.073]	1.6042 [1.958]	0.7445 [0.523]	-0.3359* [0.192]	-0.0075 [0.022]
TCD_high × Exposure2	85.9855 [183.556]	155.6869 [207.879]	-0.0525 [0.062]	0.0048 [0.070]	-0.3586 [0.584]	0.0095 [0.520]	0.0605 [0.069]	0.0104 [0.019]
TCD_r	76.9636 [179.936]	214.9931 [214.199]	0.0825 [0.112]	-0.0231 [0.085]	0.2794 [0.515]	-0.375 [0.421]	-0.05 [0.045]	0.0123 [0.036]
TCD_low	-479.6908 [446.318]	91.1026 [162.141]	0.1385 [0.153]	-0.0242 [0.046]	-1.6942 [1.816]	-0.7651* [0.405]	0.2478 [0.180]	0.0053 [0.014]
TCD_high	-229.5858 [157.609]	-215.2236 [155.300]	0.0114 [0.046]	-0.0013 [0.057]	0.7865 [0.550]	-0.0561 [0.385]	-0.0905 [0.068]	-0.0198 [0.020]
Exposure1	123.4591 [168.536]	103.8036 [157.060]	-0.0019 [0.046]	-0.0759 [0.084]	0.4689 [0.384]	-0.4344 [0.381]	-0.0111 [0.041]	-0.0096 [0.022]
Exposure2	-199.5031 [175.896]	84.568 [209.349]	0.1580* [0.093]	-0.0785 [0.105]	-0.6036 [0.524]	0.0183 [0.538]	0.0946 [0.069]	-0.0116 [0.022]
Control parents' education, age at birth, children's sex, parity, birth year, month, location	v	v	v	v	v	v	v	v
Observations	332	263	332	263	332	263	332	263
R-squared	0.583	0.595	0.467	0.547	0.499	0.632	0.425	0.46

Notes: Figures in parentheses are robust standard errors. \*\*\*significant at 1%; \*\*significant at 5%; \*significant at 10%