The Effect of Maternal Stress on Birth Outcomes: Exploiting a Natural Experiment[1].

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[1] *Preliminary draft, please do not cite or quote without the author's permission.* The motivation for this paper emerged from conversations with Donald Treiman, to whom I am greatly indebted. I am grateful to Rajeev Dehejia, Carolina Echevarria MD, Catterina Ferreccio MD, Juho Harkonen, Eric Klinenberg, Gina Lovasi, Nicole Marwell, Catherine Monk, Patrick Sharkey, and Julien Teitler for helpful comments and suggestions. I would like to thank Danuta Raj MD, head of the Department of Statistics at the Ministry of Health Chile and her team for providing the Chilean birth registry data, and for kindly addressing my multiple questions. I am also grateful to Manuel Dinamarca, from the Chilean National Emergency Office (ONEMI) for providing information about earthquake magnitude by county. Mauricio Bucca and Emily Rauscher provided excellent research assistance.

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Abstract: *A growing literature highlights that in-utero conditions are consequential for individual outcomes throughout the life cycle, but research assessing causal processes is scarce. This paper examines the effect of one such condition – prenatal maternal stress – on birthweight. Birthweight is an early outcome shown to affect cognitive, educational, and socioeconomic attainment later in life. Exploiting a major earthquake as a natural experiment and a difference-in-difference methodology, I show that maternal stress has a substantial detrimental effect on birthweight. This effect is focused on the first trimester of gestation, and it is mediated by reduced gestational age rather than intra-uterine growth restriction. Several robustness and sensitivity tests confirm the causal influence of prenatal maternal stress and reject the hypothesis that the association is driven by unobserved selectivity of mothers. The findings highlight the relevance of understanding the early emergence of unequal opportunity and of investing in maternal wellbeing since the onset of pregnancy.*

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"And surely we are all out of the computation of our age, and every man is some months older than he bethinks him, for we live, move, have a being, and are subject to the action of the elements and the malice of diseases, in that other World, the truest Microcosm, the Womb of our Mother" (Sir Thomas Browne 1642: XXXIX).

1. Introduction. A growing body of evidence shows that early conditions have important consequences for health, educational, and socioeconomic outcomes. Recent research highlights the interaction between genes and environment during the first years of life and has made a case for investment in early human capital development (e.g. Shonkoff and Phillips 2000, Heckman 2006, Palloni 2006). While some research restricts this concern to determinants after birth, there is no good reason to do so. If, as the epidemiological literature on "fetal origins" suggests, *in-utero* conditions start a cascading of events that affect individual outcomes throughout their life cycle, attention should be placed on this early critical period. This paper focuses on one such condition –maternal stress– and its effect on infants' gestational age and birth weight.

I focus on stress because it is both highly prevalent and unequally distributed along socioeconomic and racial lines. Both chronic stress emerging from enduring economic strain and discriminatory experiences, and acute stress emerging from severe life events are more prevalent among the disadvantaged (Turner et al. 1995, Pearlin et al. 2005). I focus on birth outcomes because they are a major social problem in developed and developing societies alike. Low birthweight is the leading cause of infant mortality and it affects physical and mental health, cognitive development, educational and socioeconomic attainment. As a result, policies that increase birthweight may have large social benefits (Behrman and Rosenzweig 2004).

A substantial literature has explored the relationship between maternal stress and birth outcomes. Establishing a causal effect has proven difficult, however. The main limitation is the

"selectivity problem" – women who experience or report higher stress levels may have different genetic endowments, engage in different behaviors, have access to different resources and networks, or be selected by any other unobserved attribute correlated with birth outcomes, making it impossible to identify the effect of stress. The non-random allocation of stress is, to date, the major barrier in the evaluation of its consequences.

To address the selectivity problem I exploit a natural experiment within a counterfactual model. I use a major earthquake -- the Tarapaca earthquake that struck Northern Chile in 2005-- as an exogenous source of maternal stress to evaluate the outcomes at different gestational ages of infants *in-utero* during the earthquake.

Natural experiments use naturally occurring events as source of exogenous variation in presumed causes (treatments), and they are increasingly used in the social sciences (e.g. Meyer 1995). To the extent that these events are uncorrelated with the characteristics of those receiving the treatment, they reduce the risk of omitted variable bias and allow causal evaluation even if not created in a laboratory. As an example, a recent study uses the 1918 influenza pandemic to examine the long-term consequences of *in-utero* exposure to the flu (Almond 2006). The influenza epidemic can be seen as a random treatment. It struck without warning and dissipated quickly; as a result cohorts born only months apart experienced radically different conditions, reducing the influence of omitted variables affecting who got the flu.

This example highlights an important characteristic of population-level analysis of causal effects when *not all* subjects change treatment status as a result of treatment assignment. In these circumstances, treatment assignment (assignment to an area hardly stricken by the flu epidemic) can be seen as an instrumental variable for estimating the causal effect of an endogenous causal factor

(being infected by the flu). The causal effect estimated is the intent to treat (ITT), which measures the aggregate impact of being assigned to the treatment, under the assumption that some, but probably not all, individuals will receive the actual treatment.

The "Tarapaca" earthquake struck the northernmost region of Chile on June 13, 2005. It registered an intensity of 7.9 on the moment-magnitude (MM) scale, which replaces the obsolete Richter scale. Based on its MM score, the Tarapaca earthquake is classified as "disastrous." As a reference, the Northridge earthquake that affected Los Angeles, California in 1994 had magnitude of 6.7, and the devastating 2008 Sichuan earthquake in China reached 7.9. The areas most affected were the Chilean cities of Iquique and Alto Hospicio, and the surrounding towns, with a population of approximately 272,000.

In spite of its violence, the Tarapaca earthquake had few spillover effects, considerably limiting the scope for paths other than stress to affect birth outcomes. The earthquake's toll in terms of lives and property damage was small – 11 people died, 130 were injured, and 180 residences were destroyed. This limited damage is the result of seismic preparedness and low population density. Because Chile is located at the convergence of the South American and Nazca tectonic plates, earthquakes are not unusual, and much infrastructure uses seismic-proof technology. Low population density minimized problems associated with human concentration in disaster-affected areas. The economic consequences were more noticeable. Following the earthquake, the regional index of economic activity dropped by 20.7% compared to the same quarter one year before, and recovery did not begin until early 2006 (INE 2005, 2006). However, the economic dislocation mostly affected the mining industry. Given that mining is capital-intensive, the earthquake had little impact on labor force participation and employment. The unemployment rate in the June-August trimester increased

from 11% to 12% compared to the same trimester a year earlier, a trend that does not substantially depart from the rest of the country. Because the earthquake most heavily damaged sparsely populated rural villages, which accounted for only 2% of the population in this affected region, displacement was reduced. The consequences for human health in terms of acute respiratory infections and other diseases appear to have been modest (Chiu 2006).

These characteristics render the Tarapaca earthquake a unique natural experiment that satisfies several conditions for a valid instrument for maternal stress in the context of a counterfactual model (Angrist et al. 1996, see also Morgan and Winship 2007: 200-210). First and foremost, the allocation of stress is random. Even though Chile is a country where earthquakes are expected, with the current technology it is impossible to predict when or where in the country an earthquake will occur. Secondly, the minimal long-term spillover effects satisfy the "exclusion restriction", i.e. the assumption that no alternative paths of influence other than stress account for the association between the earthquake and birth outcomes. The third condition requires that the earthquake should induce stress in at least some individuals in the population. Research shows that earthquakes are a major source of physiological and psychological distress – measured by an increase in acute cardiac events, stroke, and changes in brain function among the population affected (Leor et al 1996, Dimsdale 2008, Lui et al. 2009). Even if earthquakes are expected in Chile, on average every Chilean will experience one major seism in their lifetime, ruling out habituation effects. Fourthly, the Tarapaca earthquake satisfies the monotonicity condition, i.e. the requirement that exposure to the earthquake cannot induce stress on some individuals but *reduce* stress on others. This assumption has high face value plausibility –no evidence exists suggesting a decline in stress as a response to a natural disaster. Finally, the stable unit treatment value assumption (SUTVA) requires that the stress

response of individual women should not be affected by other individuals' experience of stress. This assumption is likely violated in a context of interactional and collective influences and I will return to it. These assumptions are stringent and relevant. After presenting the analysis I conduct robustness tests to evaluate their plausibility and implications.

The Relevance of Early Origins: Birthweight and Adult Outcomes. A growing literature shows that lower weight babies have worse outcomes throughout their life cycle. Low birthweight is associated with infant mortality (Mathews et al. 2008), and with health outcomes such as cardiovascular disease and diabetes in adulthood (Barker et al. 1993, Hales et al. 1991, Frankel et al. 1996, but see Christensen et al. 1995). Barker and colleagues formulated the "fetal programming" hypothesis which suggested that birth weight is a marker for poor health outcomes because the same processes that cause low birth weight also cause poor subsequent developments. Programming of the fetus occurs when detrimental conditions are experienced at critical periods in early life when the system is plastic and sensitive to the environment, and it has long-lasting consequences.

Low birth weight is correlated with more than health outcomes. Cross-sectional studies show an association with early cognitive performance, educational attainment, employment, earnings, and the birth weight of the next generation (Currie and Hyson 1999, Boardman et al. 2002, Case et al. 2005). The main limitation of this cross-sectional research, however, is that it cannot identify causal influences, as there could be unobserved genetic or environmental factors connected with both birth weight and later outcomes. To address this limitation, a recent literature uses comparisons between siblings, fraternal twins, and identical twins to account for potential omitted variable bias. These studies confirm the detrimental impact of low birth weight on short- and long-term outcomes. Using

siblings, researchers have found the negative influence of birth weight on infant mortality, educational attainment, height, occupational status, income, and the birth weight of children (Conley and Bennett 2000, Currie and Moretti 2005, Black et al. 2007, Oreopoulos et al. 2008). Twin studies have also found substantial effects on these outcomes (Almond et al. 2005, Conley et al. 2006, Behrman and Rosenzweig 2004, Black et al. 2007, Royer 2009).

Sibling and twin comparisons have opened new avenues in the research on the consequences of birth weight and the identification of its mechanisms. Sibling fixed effects are robust to any time-invariant mother-specific factors, but not to pregnancy-specific factors, which are only controlled for by twin studies. Only identical twin studies fully control for genetic traits. Furthermore, different mechanisms affect outcomes of singleton siblings compared to twins. Sibling studies capture variation based on gestational age and intrauterine growth. In contrast, twins have identical gestational ages, so variation in birth weight emerges only from different rates of intra-uterine development. The fact that sibling studies find birth weight effects as high as those based on twin comparisons – or even higher for some outcomes such as infant mortality (see Black et al. 2007) – suggests that both, reduced gestational age and intra-uterine growth restriction matter for later outcomes. Based on this suggestive evidence, this analysis will examine the effect of maternal stress on both birth outcomes.

Maternal stress and Birth Outcomes: Accounting for Mechanisms. A voluminous literature documents the association between maternal stress and birth outcomes. Existing studies focus on four types of stress: Major life events; short-term state and long-term trait anxiety; stress associated with discrimination and racism; and pregnancy-specific anxiety (Austin and Leade 2000 and Hobel 2004, for reviews of the literature). Most studies find that maternal stress affects birthweight by shortening

gestational age (Copper et al. 1996, Nordentoft 1996). However, the evidence is not yet conclusive, and some studies find effects on intrauterine growth restriction as well (Wadhwa et al. 2004).

The mechanisms linking stress to birth outcomes are physiological and endocrine. Maternal stress has been implicated in the production of corticotrophin releasing hormone (CRH), adrenocorticotrophic hormone (ACTH), and cortisol in both the mother and the fetus, which are in turn related to premature delivery (Glynn et al 2001, Hobel et al. 1999, Hobel 2004, Lockwood 1999). While conclusive evidence exists on the link between stress and cortisol-producing hormones, the important question about the time during pregnancy when stress is more detrimental is still unresolved.

One approach argues that maternal stress later in the pregnancy – particularly in the third trimester– is more influential. Researchers claim that stress-induced CRH alters the physiology of parturition, which produces uterine contractions resulting in early delivery (Majzoub 1999, Mancuso et al. 2004, Wadhwa et al. 1998, 2004). An alternative approach suggests that stress early in the pregnancy has greater consequences. As pregnancy advances, physiological changes lead to dampened responses to stress (Glynn et al. 2001, 2004, de Weerth and Buitelaar 2005). These changes protect women from the consequences of stress later in the pregnancy but leave them vulnerable early on. This hypothesis suggests that early maternal stress initiates a chain of events leading to preterm labor. Specifically, early stress triggers CRH gene expression in the placenta, which initiates a biological clock for early delivery (McLean et al. 1995, 1999, Hobel et al. 1999, Hobel 2004, Sandman et al. 2006).

Given the impact of early circumstances on later outcomes, many socioepidemiological studies have attempted to evaluate the impact of maternal stress on birthweight. Over time, these

studies have moved from retrospective to prospective, using larger sample sizes and adding controls for potential confounders, such as smoking or socioeconomic status. However, most of them cannot rule out the non-random allocation of stress. In fact, the very findings of high-quality studies suggest that the selectivity problem may be a major limitation. For example, one study finds no association between stressful life events and birth outcomes independent of women's appraisals (Hedegaard et al. 1996). In contrast, life events *assessed by women as highly stressful* were associated with low birth weight, suggesting the influence of omitted variables related to maternal personality. Furthermore, to the extent that unobserved factors confound the association between stress and birth outcomes, the important question of when in the pregnancy stress is more detrimental cannot be properly addressed.

A few recent studies use natural experiments to evaluate the influence of stress on birth outcomes. The landmark study by Glynn et al. (2001) uses the 1994 Northridge earthquake and was the first to suggest that the consequences of maternal stress exposure depend on the gestational age of exposure. However, with a sample of only 40 women and no controls for trends in other parts of the country, the evidence is only suggestive. Other studies have utilized the assassination of a Prime Minister and the sinking of a ferry in Sweden (Catalano and Hartig 2001), the September 11th attacks in New York (Eskenazi et al. 2007), and, in an exemplary analysis using a large dataset, the harassment of Arab and Arab-American women after the September 11th attacks in California (Lauderdale 2006) as potential sources of maternal stress. This study builds on this emerging literature and makes three contributions. First, by exploiting an undoubtedly random exogenous shock, I control for unobserved selectivity associated with stress. Second, by using an acute stressor with few long-term spillover effects, I can examine the effect of the timing of stress on birth outcomes. Finally,

the rich dataset allows me to evaluate the alternative mechanisms –reduced gestational age and intra-uterine growth restriction– linking maternal stress and low birth weight.

Data, Variables and Analytical Strategy. The main data source is the file of all Chilean birth certificates for 2004, 2005, and 2006; there are over 200,000 births a year. Each record includes information on gestational age at delivery, sex, weight, and height of the newborn, type of delivery (single, multiple), and medical attention. It also includes information on maternal age, marital status, number of previous deliveries, and education. Crucial for this analysis, the birth record also includes the mother's county of regular residence. Chile has a total population of 16 million and an area of 285,860 square miles, divided into 350 counties. Accordingly, counties have on average an area of 816 square miles and a population of 45,000. As a result, the county-level provides a very precise indication of geographical location. I merge information about maternal county of residence during pregnancy with information about the intensity of the earthquake across locations provided by the Chilean National Emergency Office to produce a measure of the treatment.

Variables: The dependent variables are birth weight measured in grams, gestational age measured in weeks, and weight-by-gestational age. In an alternative specification, these variables are dichotomized to produce the standard measures of low birth weight (<2,500 grams), pre-term delivery (< 37 weeks of gestation), and intrauterine growth restriction (birth weight below the 10th percentile of the weight distribution by gestational age[1]). These dichotomous measures identify thresholds claimed to be consequential for later outcomes and they are widely used by the medical and research community.

[1] Given international and temporal variation in weight-by-gestational age distributions, I use updated birthweight curves for the Chilean population (Gonzalez et al. [2004]) to calculate IUGR.

Birth weight is recorded by the professional (MD or certified midwife) who attends the delivery (99.8% of deliveries are attended by a professional in Chile) and measured with little error (Mardones et al. 2008). Gestational age is more difficult to ascertain than birth weight (e.g. Reichman and Hade 2001, Roohan et al. 2003). In the Chilean case, the health care provider estimates gestational age based on last menstrual period information provided by the mother, and early-pregnancy sonogram. The information is considered reliable for women with at least one pregnancy checkup. Based on a large probability survey, I estimate these to be 87.9% of Chilean pregnant women (this compares with 96.4% in the US [OMS 2007, CASEN 2006]). For women who have not had a pregnancy checkup, the attending professional estimates gestational age based on an interview with the mother. This estimation procedure reduces the proportion of missing data for gestational age, which reaches only 0.13% in the Chilean dataset.

The probable cost of reducing missing data for the gestational age variable is the loss in reliability[2]. The consequences of mis-estimation for the models are difficult to assess. If mis-estimation is random, measurement error in the dependent variables does not induce bias. However, if there is a systematic association between under- or over-estimation and treatment allocation (a very unlikely occurrence), then bias in the parameter estimates of interest could result. In order to test the robustness of the findings and to address potential bias I use an additional strategy to identify small preterm babies that are most vulnerable to detrimental outcomes without the need for gestational age information (Wilcox 2001), as explained in the methods subsection.

Treatment: The Mercalli scale is used to quantify the intensity of the earthquake in different regions of the country. While the moment-magnitude scale measures magnitude in terms of energy

[2] Ancillary analysis of the reliability of the gestational age data is available from the author upon request.

released, Mercalli quantifies the effect of the earthquake on the earth's surface, humans, objects of nature, and human-made structures on a scale from I to XII. The ordered categories of the Mercalli scale are the following: Instrumental (I), Feeble (II), Slight (III), Moderate (IV), Rather strong (V), Strong (VI), Very Strong (VII), Destructive (VII), Ruinous (IX), Disastrous (X), Very Disastrous (XI), Catastrophic (XII), with this last category identifying total damage. Given that the Mercalli scale captures how people feel the earthquake and the observed damage, it is a preferred metric for the earthquake as a stressor *as experienced* by the population. I measure the intensity of the Tarapaca earthquake across counties, which varies from I (instrumental) to IX (ruinous), and define a dichotomous version of the treatment. Treated areas (T_1) are those where the earthquake intensity was very strong (VII), Destructive (VIII), and Ruinous (IX), control areas (T_0) are those where intensity was lower. Dichotomization is based on the fact that the VII "very strong" level represents a categorical threshold at which the earthquake is felt by the entire population and damage starts occurring (Ramirez and Peek-Asa 2005). I then test for the robustness of this definition by utilizing an ordinal measure of intensity.

I distinguish five time points in order to address the impact of the earthquake at different gestational ages. In chronological order, t_0 identifies babies conceived after December 2003 and born before the earthquake (June 13, 2005). t_1, t_2 and t_3 identify, respectively, infants who experienced the earthquake during the third, second, and first trimester of gestation; t_4 identifies those conceived after the earthquake and no later than March 2006[3]. Only infants still *in utero* at the time of the earthquake were included in the analysis in both treatment and control groups. This criterion controls for the fact that women who experienced the earthquake early in the pregnancy had more

[3] Given that we are interested in examining the causal effect of the earthquake on gestational age, we define the time of the treatment on the basis of estimated conception date rather than "counting back" from birth date.

time at risk for preterm delivery (e.g. Glynn et al. 2001). Because multiple births are significantly lower-weight, analysis is restricted to singletons, which represent 98.2% of the births in the period considered. After these specifications, the analytical sample sizes for the treatment group across the five time periods are the following: t_0=3,610, t_1=1,066, t_2=1,087, t_3=956, and t_4=3,467; while the comparable figures for the control group are t_0= 170,410, t_1= 51,077, t_2=55,965, t_3=47,408, t_4=165,937.

Statistical Method: I employ a difference-in-differences (DID) methodology, a simple yet powerful approach to assess the influence of a treatment on a population (for instance, Meyer 1995, Cameron and Trivedi 2005: 55-57, 760-70, 878-879). Using the earthquake as the treatment, two groups are distinguished based on treatment status T=0, 1, where 1 identifies pregnant women living in areas where the intensity of the earthquake was very strong to ruinous and 0 identifies the control group living in other areas of the country. I observe individuals in five time periods, where t=0 indicates infants born before the earthquake, and the subsequent values refer to the four time points described in the previous section. This formulation is a straightforward extension of the simple case with two groups and two time points, expressed as follows. $\overline{Y}_0^T$ and $\overline{Y}_1^T$ are the sample averages of the outcome for the treatment group before and after the treatment, and $\overline{Y}_0^C$ and $\overline{Y}_1^C$, the corresponding averages for the control group (subscripts identify time periods, and superscripts identify treatment status). The outcome Y_i is modeled by the following equation:

$$Y_i = \alpha + \beta T_i + \gamma t_i + \delta (T_i^* t_i) + \varepsilon_i$$

where β is the treatment group-specific effect, which accounts for average permanent differences between treatment and control groups, γ is a time-trend common to control and treatment groups. The coefficient for the interaction term δ captures the pre-post difference in average outcome in the treatment group minus the pre-post difference in the average outcome in the control group, i.e. $\widehat{\delta}_{DD}$

$= ([\overline{Y}_1^T] - [\overline{Y}_0^T]) - ([\overline{Y}_1^C] - [\overline{Y}_0^C])$, and it is the true effect of the treatment, net of treatment-group specific factors such as high altitude (e.g. Wilcox 2001) and time-trends common to both groups, such as seasonal effect on birthweight (e.g. McGrath et al. 2007).

The unbiasedness of the DID estimator relies on the assumption that there are not unobserved factors correlated with the treatment-group specific temporal trend, called the "parallel trend" assumption" [in formal terms, $\text{cov}(\varepsilon_i, T_i * t_i)=0$]. Even if an earthquake is a random occurrence, the response to the treatment may not be random. In particular, selective out-migration from the treated area could alter the composition of the treated population. I evaluate this potential source of bias in detail in the robustness checks section.

Low birth weight has been questioned as a marker of increased mortality and other risks. Wilcox (Wilcox and Russell 1983, Wilcox 2001) shows that the birthweight distribution is essentially normal but with additional births in the lower tail. It can therefore be divided into two components: A normal term-birth weight distribution ("predominant distribution") and an excess of observations in the left tail mainly composed by small preterm births ("residual distribution").This distinction is important insofar as populations with lower mean term birth weight do not have higher risk of detrimental outcomes such as infant mortality, but small preterm infants do. I therefore conduct additional analysis to evaluate the magnitude of the residual distribution for infants affected by the earthquake using available software developed by Wilcox (http://eb.niehs.nih.gov/bwt/). By focusing on the size of the residual distribution I avoid using information on gestational age, circumventing measurement limitations, and provide supplementary evidence for the potential effect of maternal stress on early outcomes.

Analysis. The Effect of Maternal Stress on Birth Outcomes. Table 1 presents the analysis for the continuous versions of all variables – birth weight measured in grams, gestational age measured in weeks, and weight-for-gestational age as the ratio between the two – using linear regression models. The effect of the treatment across the five time points distinguished is evaluated.

Table 1 about here

Model 1 predicts mean birthweight. The parameter estimates of interest are the interaction terms between each time period and exposure to the treatment. Model 1 indicates that there is no significant decline in birthweight for the infants exposed to the earthquake in their third and second trimester of gestation[4]. However, infants exposed during their first trimester of gestation experience a large decline in birthweight, of 51 grams on average. This drop is substantial. As a reference, it is close in magnitude to the 63-grams increase in birthweight for pregnant women enrolled in the Supplemental Nutrition Program for Women, Infants and Children (WIC) (Bitler and Currie 2005: 84). But while the effect of the WIC intervention is measured only among treated women, the earthquake-related drop is impressive, as it is a *population-level* effect, which likely includes women who did not suffer from stress.

Model 3 examines the effect of earthquake-induced maternal stress on gestational age. It indicates a decline for those exposed to the earthquake in the first trimester, by one-fifth of a week for the treated infants. As in the case of birth weight, gestational age returns to pre-earthquake levels among infants conceived after the stressful event. Model 3 suggests that the effect of maternal stress

[4] Since I use data for the entire population, I make heuristic use of significance tests, implicitly invoking a super-population.

on birth weight is at least partly driven by a reduction in gestational age. Given that this effect is restricted to the first trimester of gestation, it is consistent with the hypothesis of a biological clock for preterm delivery initiated early in the pregnancy, when the mother is most vulnerable.

Model 4 addresses the effect of stress on weight-for-gestational age. It shows a significant but small decline in weight-for-age for those exposed to the earthquake in the first trimester of gestation. Finally, model 2 adds gestational age – a presumed mediator of low birth weight – to model 1; and it addresses the following question: Does maternal stress have an effect on birthweight net of gestational age? Note that model 2 addresses the same question as model 4, as there are only two alternative paths to low birthweight – reduced gestational age and intra-uterine growth restriction. After controlling for gestational age, the parameter estimate associated with the treatment substantially declines in magnitude and becomes statistically insignificant. This is consistent with the findings from model 4, and it suggests that the influence of maternal stress on birth weight is largely mediated by reduced gestational age.

A graphical display of the findings is presented in figures 1-3. Figure 1 plots mean birth weight for the treatment and control groups, across the five time points distinguished. A substantial decline in weight for women living in the treatment area limited to the first trimester of gestation is apparent. Furthermore, birthweight returns to its pre-treatment level among infants conceived after the earthquake. This trend indicates that the effect of prenatal maternal stress is tightly focused early in the pregnancy, and it is also consistent with the absence of long-term spillover effects, which would have affected those conceived after the earthquake.

Figures 1 - 3 about here

Figure 2 reproduces the analysis for weeks of gestation. The findings are similar to those on birth weight. The mean gestational weeks drops substantially among those who experienced the earthquake in their first trimester of gestation but remained nearly flat throughout the period considered in the control area. Figure 3 adds information on intrauterine growth restriction. It consistently shows a decline in weight-to-gestational age for individuals *in-utero* in the treated area during the first trimester of gestation, and a recovery thereafter.

The analysis so far uses continuous versions of the dependent variables. This implicitly assumes linear relationships and cannot identify thresholds potentially consequential for later outcomes. To address these possible limitations, I utilize standard dichotomous version of the variables – low birthweight, preterm birth, and IUGR. Table 2 presents the analysis. I use linear probability models, because the interaction effects that are the basis of the DID approach are not interpretable in nonlinear models such as probit or logit (Ai and Norton 2003). Robust standard errors account for the built-in heteroskedasticity in linear probability models.

Table 2 about here

The pattern of effects is nearly identical to those presented in table 1. Model 1 indicates that the probability of low birth weight increases from 5.3% to 7.1% among those exposed to the earthquake during the first trimester, but not in other periods of gestation. Model 3 suggests that this effect is mediated by growing chances of being born pre-term. The only departure from specifications using a continuous version of the variables is model 4 – the increase in the probability of IUGR fails to reach significance, while the higher discriminatory power of the continuous variable likely

enhances the ability to capture this small effect. Model 2 consistently shows that the influence of maternal stress on birthweight is largely mediated by increasing the chances of being born pre-term.

These findings provide strong evidence for a detrimental effect of maternal stress on birth weight. However, birthweight has been criticized as a marker for vulnerability. Thus, I use the methodology devised by Wilcox to partition the distribution of birth weight into its dominant and residual components, when the latter identifies small pre-term babies that are most vulnerable. Evidence for a maternal stress effect would emerge from differences between treatment and control groups in the size of the residual component, but not in the mean of the dominant distribution.

Table 3 about here

Table 3 shows that there is no significant change in the residual distribution across the time periods considered in the control area, with values around 3.0% before, during, and after the treatment. In contrast, for the treated area, the residual distribution is substantially larger for those infants who experienced the earthquake in their first trimester of gestation, reaching 9%. Furthermore, the post-treatment size of the residual distribution returns to their pre-treatment values, confirming that the influence of stress is focused on the first trimester (note that there is more variation in the proportion of the residual distribution than in the control group due to smaller sample sizes)[5].

To the extent that the preterm, low-weight infants in the residual distribution are at higher risk of mortality and other negative outcomes, this analysis circumvents the need to use gestational

[5] Graphical display of the dominant and residual distribution across time period and treatment group is available from the author upon request.

age information and confirms that maternal stress may have long-term negative consequences for those who experienced the earthquake during the first trimester of gestation.

Robustness and Sensitivity Analyses. The analysis has shown a substantial association between stress experienced by mothers during the first trimester of gestation and birth weight, but is this relationship causal? In spite of the random allocation of the earthquake, confounding factors may induce bias in the estimation of its effects. First, a stressful event may result in a spontaneous abortion or miscarriage (Nepomnaschy et al. 2006, Mulder et al. 2002). If the most vulnerable pregnancies were lost, yielding a healthier exposed population eligible for live birth, the outcomes of the survivors would be different from the counterfactual outcomes for the entire population. If fetal deaths increased in the area stricken by the earthquake but not in others, evidence would suggest underestimation of the earthquake's effect due to a "culling of the weakest." I examine this possibility by exploiting the records on spontaneous abortions and miscarriages obtained from the Chilean Ministry of Health. The number of reported fetal deaths in the treated area was 3, 13, and 5 for those who experienced the earthquake during their first, second and third trimester of gestation, respectively. These figures compare with an average of 7 fetal deaths per trimester in the treated area over the entire 2004-2006 period excluding the 9 months of in-utero exposure to the earthquake. These figures are not consistent with a "culling of the weakest" hypothesis. Furthermore, if some abortions and miscarriages, particularly early ones, went unnoticed or unreported, this would result in this analysis *underestimating* the negative stress effects on birth outcomes.

Secondly, the dichotomous formulation of the treatment needs further testing. I therefore implement an ordinal specification, obtained by dividing the control group into a "moderate

intensity" segment where the intensity was moderate to strong (IV-VI in the Mercalli scale), and a "low intensity" segment, where the intensity was less than moderate (less than IV in the Mercalli scale). Results are presented in table 4. Model 1 evaluates the effect of the treatment on birth weight, while model 2 assesses the probability of low birthweight. Figure 4 plots the predicted birthweight across treatment groups and time obtained from model 1. It reproduces the substantial decline in birthweight among infants who experienced the treatment during the first trimester of gestation already reported in previous analysis; it shows a smaller decline in the "moderate intensity" area but no such decline in the "low intensity" control area. Similar findings are obtained for the probability of low birthweight. Although this drop in the moderate intensity area is insignificant at conventional statistical levels, it is a nontrivial population-level decline consistent with a dose-response influence of an acute environmental stressor.

Table 4 and figure 4 about here

Thirdly, the DID analysis rests on the "parallel trend" assumption, requiring that there are not treatment-group specific trends that could bias the coefficient. Although this assumption is plausible given the random allocation of the earthquake, there is one potential source of bias that needs to be explored. The earthquake may have been followed by selected out-migration from the treated region, altering the composition of the babies at risk. If, for example, healthier women were overrepresented among the out-migrants, the increase in low birth weight may be due to the selective reduction of the population.

To assess this possibility, I use the large-scale probability 2006 CASEN survey to identify

women aged 15-50 with children born between mid-June 2005 and March 2006 (i.e. who are likely to have been pregnant during the earthquake). I examine the proportion of these women who changed county of residence after the earthquake, by relying on the survey question "Were you living in [your current county of residence] in 2002? If the woman was not living in her current county of residence four years ago, the survey asked: In which county were you living in 2002?[6]

Even if the 2002-2006 comparison captures migratory flows taking place before the earthquake, it will provide a close approximation to earthquake-induced migration. Overall, only 10.5% of women changed counties between 2002 and 2006 in Chile. This proportion was slightly higher in the treated area, reaching 11.5% of the population. This suggests very minor differences in the rate of out-migration from the treated area. Furthermore, detailed analysis of the migrants' counties of destination indicates that 38% of those who emigrated from a county in the treated area moved to another county within the treated area. As a result, only 7.1% of the population living in the treated area in 2002 had moved to a non-treated area by 2006. Even if the influence of selective out-migration cannot be fully ruled out, these small percentages suggest that, if it exists, it should be minor. As an additional test of compositional changes in the treated area, I exploit the sociodemographic information contained in the birth records, and replicate the analysis for birthweight and the probability of low birthweight adding controls for maternal age, education, parity, marital status, and rural residence. In an alternative specification, I add county fixed effects to account for unobserved factors at the community level, and use propensity score methods to weight the regression by the propensity score of living in the treated area to improve the balance between treated and untreated women (Imbens 2004). In all these specifications, reported in Appendix 1, the

[6] The question was asked of all family members, except, naturally, for those who had not been born in 2002. Survey question allowed for residence in a foreign country in 2002. Individuals living in a foreign country in 2002 are only 0.4% of the population.

effect of the earthquake is nearly identical to the original findings, providing further evidence against compositional-level confounders.

Finally, the use of an earthquake as an instrumental variable relies on the assumption that no alternative paths of influence other than stress account for the association between the earthquake and birth outcomes. Limited spillover effects of the earthquake gives credence to this exclusion restriction. As an additional test, I analyze the effect of stress on birthweight across maternal level of schooling, distinguishing women with primary and secondary education from those with college education. Results are presented in table 5.

Table 5 about here

Model 1 models mean birthweight and model 2 reproduces the analysis using the probability of low birthweight as the dependent variable. Before moving to the core of the analysis note that – as indicated by the regressions' intercepts in model 1— infants born to women with college education weigh less, on average, than those born to women with less schooling. This gap is accounted for by the well-documented sharp increase in obesity among lower-education women, which raises the likelihood of high-weight (macrosomic) births (Mardones 2003, Atalah et al 2004). Interestingly, the higher proportion of heavy babies does not translate into lower rates of low birthweight, indicating that the increase in birthweight is focused on the upper extreme of the birthweight distribution among lower-education women.

Moving to the core of the analysis, table 5 shows that the detrimental effect of the acute stressor is *greater* among highly-educated women. Infants exposed to the earthquake in the first

trimester of gestation experience a decline in weight of 118 grams and the increase in the proportion low birth is 3.7% if their mothers are college-educated. The comparable figures among low-education women are 33 grams and 1.4%, significant only at the $p<.10$ level. Under the assumption that a college degree is correlated with the ability of women to prevent or reduce her vulnerability to the long-term effects of an earthquake – for example, economic displacement and health outcomes – this evidence is consistent with reduced spillover effects and supports the assumption that the main path linking the earthquake to birth outcomes was prenatal maternal stress, on which the exclusion restriction is based. It also raises the question: Why is the effect of stress more detrimental among highly educated women?, which I return to in the next section.

Conclusions and Discussion. There is growing awareness in the social sciences that individuals are sensitive to the external environment even before birth. An emerging literature suggests that *in-utero* conditions may have consequences not only for mortality and health, but also for cognitive and socioeconomic outcomes throughout the life-cycle. This hypothesis has intuitive appeal. Intrauterine life is a critical period, in which the organism is highly susceptible to alterations in its programming, so that environmental impacts can have long-lasting consequences. However, measuring the influence of specific characteristics of the environment while *in utero* has been hampered by the difficulty of isolating these factors.

This paper analyzes influence of one such condition – prenatal maternal stress – on birthweight. I focus on birthweight because it is an early marker for cognitive, educational and socioeconomic outcomes, thus shaping the human capital and wellbeing of subsequent generations. I focus on maternal stress because it is both highly prevalent and unequally distributed in

contemporary societies.

Given its importance, much research has attempted to establish a causal relation between prenatal maternal stress and birth outcomes. The main limitation this literature faces is the potential for selectivity bias –women who experience or report more stress may have different genetic endowments, engage in different behaviors, have access to different resources and networks, or be selected by another unobserved attribute correlated with birth outcomes – preventing the evaluation of the causal effect of stress. I address this limitation by exploiting a natural experiment, the occurrence of an earthquake while *in-utero,* and a counterfactual approach to evaluate the influence of maternal stress on birth weight, gestational age, and intra-uterine growth restriction. Drawing on all birth certificate records from 2004 to 2006 in Chile, I examine not only *whether* maternal stress has an effect but *when* in the pregnancy is stress more influential.

The findings are robust and consistent. They indicate a substantial increase in the probability of low birthweight for women who experienced the acute stressor in the first trimester of gestation but *not later* in the pregnancy. I also find that this effect is largely mediated by a reduction in gestational age, rather than by factors affecting the intra-uterine growth of term infants. The findings suggest that the mechanism plausibly at play is the trigger of a placental clock for premature delivery by early-pregnancy stress. Although this hypothesis is new, it has received growing support in recent research and is consistent with the notion of other biological clocks applied to aspects of maturation in extra-uterine life, such as the timing of puberty of senescence.

It is important to highlight that the causal effect estimated here corresponds to the "intent to treat" of stress, the population-level effect of being exposed to the stressor, rather than the individual-level experience of stress. Under the reasonable assumption that some, but not all pregnant women

exposed to the earthquake suffered from stress, the ITT estimate is lower, perhaps substantially so, than consequences among the subgroup of women who did experience stress as a result of the earthquake. Given the limited resources and large costs of policies targeted to reduce low birthweight, this study suggests the possibility for considerable social benefits from investments to identify and reduce maternal stress and to provide health, psychological, and economic support to pregnant women. The fact that effects are focused very early in the pregnancy, when many women may not be aware of their condition, suggests that early detection may be particularly important.

This study also raises important further questions. I have exploited an unusual natural disaster to examine the causal effect of maternal stress. A relevant question is the extent to which findings can be generalized to other sources of stress. The answer is a qualified yes. Unfortunately, acute stress such as elicited by an earthquake is not an uncommon occurrence in contemporary societies. Events such as heat waves, storms, tornadoes, floods and other 'natural' disasters, industrial and technological accidents, violence associated with terrorism and war, and sudden macroeconomic crises are prevalent in contemporary societies. They have all increased in recent decades in industrial and developing countries alike, and no diminution is foreseeable. Furthermore, most of these events are not allocated randomly and tend to affect the most deprived nations and groups within nations (Perrow 2007, Wisner et al 2006). The findings hereby presented likely generalize to these situations. Furthermore, the limited long-term spillover effects associated with this event helps isolate the influence of the physiological and psychological stress response, but it is the exception rather than the rule. It is likely, then, that the effects hereby estimated are a *lower bound* of the detrimental influence of prevalent sources of acute stress. More studies, exploiting natural experiments when possible, could address this question.

But much stress in contemporary societies is not acute but chronic, as emerging from continuous economic strain and discrimination experiences. Although research suggests similar physiological and endocrine responses to both sources of stress (Sapolsky 2004), the generalizability of these results to chronic stress is very much an empirical question. My unexpected finding that earthquake-induced stress appears to be *more* detrimental among the college-educated (see table 5) cautions against easy generalizability. This finding is consistent with the "inoculation effect" hypothesis emerging from psychology. According to this hypothesis, given the enormous resilience of human beings, chronic stress emerging, for instance, from economic strain reduces the reactivity to an acute novel stressor (Eysenck 1983, Gump and Matthews 1999). While testing this hypothesis is beyond the scope of this paper, it cautions against easy extrapolation of the findings. It also highlights the need to examine the complex interactions regulating the association between socioeconomic advantage, exposure to stress, and birth (or individual) outcomes (Tierney 2000, McLeod and Lively 2007, Thoits 2006).

As mentioned in the introduction, a source of concern for the causal interpretation of the findings is the potential violation of the stable unit treatment value assumption (SUTVA), the assumption that the effect of the treatment is independent of others receiving the treatment. Insofar as individual stress levels depend on interactional or collective dynamics and support networks (Aneshensel 1992, Thoits 2006) the aggregate of individual-level effects may be mis-estimated. At one level, this is not a limitation. To the extent that in real societies individuals are embedded in social networks and necessarily interact with others, the effect captured by this analysis is relevant in terms of the real consequences of stressful collective phenomena. The question remains, however, about the effect of circumstances that induce distress among specific individuals without social

ramifications.

This analysis has provided robust evidence for a causal effect of maternal stress on birth outcomes, with potentially long-lasting consequences into adulthood. However, initial disadvantage is not deterministic. Developmental trajectories are crucially shaped by resources such as access to networks and institutions, neighborhood contexts, and parenting styles. I hope this study has motivated more research of the environmental factors that shape the early origins of advantage and disadvantage, and on the factors that reduce or exacerbate such disadvantage during the initial stages of the life course.

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Table 1. Difference-in-Difference Analysis. Effect of Maternal Stress on Birthweight, Gestational Age and Weight-by-Gestational Age. Chilean births 2004-2006.[1]

	Model 1 Birth weight[2]		Model 2 Birth weight[2]		Model 3 Gestational age[3]		Model 4 Weight by gestational age[4]	
	Coeff.	S.E.	Coeff.	S.E.	Coeff.	S.E.	Coeff.	S.E.
Earthquake strong-ruinous (T=1)	46.302***	(8.655)	17.264*	(6.705)	.154***	(.028)	0.824***	(.198)
Born before earthquake (t=0)								
Third trimester gestation (t=1)	8.881***	(2.596)	-3.731	(2.011)	.067***	(.008)	0.168**	(.059)
Second trimester gestation (t=2)	-2.870	(2.507)	-4.545	(1.942)	.009	(.008)	-0.085	(.057)
First trimester of gestation (t=3)	-10.330***	(2.672)	0.426	(2.070)	-.056***	(.008)	-0.143*	(.061)
Conceived after earthquake (t=4)	-5.172**	(1.775)	5.495***	(1.375)	-.056***	(.005)	-0.022	(.040)
t0 (born before earthquake) * T1								
t1 (third trimester) * T1	-0.148	(18.111)	-2.800	(14.030)	.014	(.060)	-0.006	(.416)
t2 (second trimester) * T1	16.656	(17.985)	15.788	(13.932)	.004	(.060)	0.505	(.413)
t3 (first trimester) * T1	-51.202**	(18.898)	-14.222	(14.640)	-.195**	(.063)	-0.954	(.434)
t4 (conceived after earthquake)*T1	6.956	(12.360)	1.078	(9.575)	.031	(.041)	0.140	(.284)
Gestational age			189.01***	(.327)				
Intercept	3345.4***	(1.246)	-3962.5***	(12.684)	38.663***	(.004)	86.273***	(.028)
N	500,983		500,983		500,983		500,983	

[1] Linear regression model with robust standard errors. Sample includes all singleton infants conceived between December 2003 and March 2006 and born in Chile. * p<.05, ** p<.01, ***p<.001

[2] Birthweight measured in grams.

[3] Gestational age measured in weeks.

[4] Birthweight (grams) divided by gestational age (weeks).

Table 2. Difference-in-Difference Analysis. Effect of Maternal Stress on Low Birthweight, Preterm Birth and IUGR. Chilean births 2004-2006.[1]

	Model 1		Model 2		Model 3		Model 4	
	Low birth weight[2]		Low birth weight[3]		Preterm delivery[4]		IUGR[5]	
	Coeff.	S.E.	Coeff.	S.E.	Coeff.	S.E.	Coeff.	S.E.
Earthquake strong-ruinous (T=1)	-.007	(.004)	-.002	(.003)	-.008*	(.003)	-.006	(.003)
Born before earthquake (t=0)								
Third trimester gestation (t=1)	-.007***	(.001)	-.002**	(.001)	-.008***	(.001)	-.0003	(.001)
Second trimester gestation (t=2)	-.0001	(.001)	.001	(.001)	-.002*	(.001)	.001	(.001)
First trimester of gestation (t=3)	.001	(.001)	-.0003	(.001)	.002	(.001)	.001	(.001)
Conceived after earthquake (t=4)	.003***	(.001)	.002*	(.001)	.002**	(.001)	-.0001	(.001)
t0 (born before earthquake) * T1								
t1 (third trimester) * T1	.003	(.008)	.004	(.007)	-.001	(.008)	.006	(.007)
t2 (second trimester) * T1	-.007	(.008)	-.003	(.006)	-.006	(.008)	.008	(.007)
t3 (first trimester) * T1	.018*	(.008)	.002	(.007)	.026**	(.008)	-.005	(.007)
t4 (conceived after earthquake)*T1	-.001	(.005)	-.003	(.004)	.002	(.005)	-.0004	(.005)
Preterm delivery			.594***	(.001)				
Intercept	.053***	(.001)	.017***	(.001)	.060***	(.001)	.048***	(.001)
N	500,983		500,983		500,983		500,983	

[1] Linear probability models with robust standard errors. Sample includes all singleton infants conceived between December 2003 and March 2006 and born in Chile. * p<.05, ** p<.01, ***p<.001

[2] Birth weight < 2,500 grams.

[3] Gestational age < 37 weeks.

[4] Below 10th percentile of birth weight for gestational age.

Table 3. Birth Weight Analysis of all Chilean Births, Dominant Normal Distribution and Residual Percent for Treated and Control Groups.[1]

	Dominant Birthweight Distribution (grams)		Residual Distribution	N
	Mean	s.d.	%	
Control group				
Pre-treatment (born before earthquake)	3401	437	2.8	170,410
Treatment 3rd trimester of gestation	3395	432	2.2	51,077
Treatment 2nd trimester of gestation	3399	429	3.0	55,965
Treatment 1st trimester of gestation	3387	443	2.5	47,408
Post-treatment (conceived after the earthquake)	3398	439	3.0	165,937
Treatment group				
Pre-treatment (born before earthquake)	3450	445	2.9	3,610
Treatment 3rd trimester of gestation	3461	429	5.5	1,066
Treatment 2nd trimester of gestation	3470	420	4.9	1,087
Treatment 1st trimester of gestation	3449	415	9.0	956
Post-treatment (conceived after the earthquake)	3457	431	4.1	3,467

[1] Sample includes singleton infants conceived between December 2003 and March 2006 and born in Chile.

Table 4. Difference-in-Difference Analysis. Effect of Maternal Stress on Birthweight with Ordinal Specification of Treatment. Chilean births 2004-2006.[1]

	Model 1 Birthweight		Model 2 Low birthweight[2]	
	Coeff.	S.E.	Coeff.	S.E.
Earthquake moderate (T=1)	16.228	(9.496)	-.006	(.004)
Earthquake strong (T=2)	44.105***	(8.653)	-.006	(.004)
Born before earthquake (t=0)				
Third trimester of gestation (t=1)	8.289**	(2.635)	-.007***	(.001)
Second trimester gestation (t=2)	-3.436	(2.542)	-.0003	(.001)
First trimester gestation (t=3)	-10.540***	(2.712)	.001	(.001)
Conceived after earthquake (t=4)	-5.333**	(1.800)	.002***	(.001)
t0 (born before earthquake) * T1				
t1 (third trimester) * T1	9.090	(16.576)	-.002	(.009)
t2 (second trimester) * T1	8.763	(16.524)	-.004	(.009)
t3 (first trimester) * T1	-13.757	(15.137)	.009	(.009)
t4 (conceived after earthquake) * T1	12.942	(11.555)	.002	(.006)
t0 (born before earthquake) * T1				
t1 (third trimester) * T1	0.445	(18.106)	.003	(.008)
t2 (second trimester) * T1	17.221	(17.979)	-.006	(.008)
t3 (first trimester) * T1	-50.991**	(18.893)	.018*	(.008)
t4 (conceived after earthquake) * T1	7.118	(12.357)	-.001	(.005)
Intercept	3344.5***	(1.264)	.053***	(.001)
N	500,983		500,983	

[1] Linear regression models with robust standard errors. Sample includes all singleton infants conceived between December 2003 and March 2006 and born in Chile.

[2] Linear probability model with robust standard errors, dependent variable is probability of low birthweight (<2,500 grs.).

Table 5. Difference-in-Difference Analysis. Effect of Maternal Stress during different gestational periods on birth weight by maternal education. Chilean births 2004-2006.[1]

	Model 1[2] Mother's Education				Model 2[3] Mother's education			
	Primary and Secondary		College		Primary and Secondary		College	
	Coeff.	S.E.	Coeff.	S.E.	Coeff.	S.E.	Coeff.	S.E.
Earthquake strong-ruinous (T=1)	35.332***	(9.745)	87.123***	(18.659)	-.004	(.004)	.020*	(.009)
Born before earthquake (t=0)								
Third trimester gestation (t=1)	10.277**	(2.966)	8.279	(5.282)	-.007***	(.001)	-.008***	(.002)
Second trimester gestation (t=2)	-2.186	(2.871)	-0.994	(5.062)	.0005	(.001)	-.002	(.002)
First trimester of gestation (t=3)	-8.362**	(3.071)	-12.606*	(5.329)	.002	(.001)	-.001	(.002)
Conceived after earthquake (t=4)	-2.781	(2.040)	-2.922	(3.540)	.003***	(.001)	.001	(.001)
t0 (born before earthquake) * T1								
t1 (third trimester) * T1	8.656	(20.586)	-21.217	(37.711)	.0002	(.009)	.005	(.018)
t2 (second trimester) * T1	26.147	(20.535)	-14.771	(36.746)	-.010	(.009)	.005	(.018)
t3 (first trimester) * T1	-33.908	(21.019)	-118.09**	(38.424)	.014	(.009)	.037*	(.018)
t4 (conceived after earthquake) * T1	15.502	(14.051)	-26.638	(25.768)	-.004	(.006)	.007	(.012)
Intercept	3316.4***	(1.755)	3278.7***	(4.394)	.053***	(.001)	.053***	(.002)
N	388,575		112,408		388,575		112,408	

[1] Linear regression model with robust standard errors. Sample includes all singleton infants conceived between December 2003 and March 2006 and born in Chile. Dependent variable is birthweight measured in grams. Controls included for maternal age (19 years old, 20-29, 30-34, 35 and older), marital status (married, non-married), parity (1 birth, 2-3, 4 or more) and urban residence.

[2] Linear regression models with robust standard errors, dependent variable is birthweight measured in grams.

[3] Linear probability models with robust standard errors, dependent variable is probability of low birthweight (<2,500 grams).

Appendix Table 1. Difference-in-Difference Analysis. Effect of Maternal Stress on Birthweight, Gestational Age and Weight-by-Gestational Age with individual and county-level controls. Chilean births 2004-2006.[1]

	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	Birth weight[2,4]		Low BW[3,4]		Birthweight[2,5]		Low BW[3,5]		Birthweight[2,6]		Low BW[3,6]	
	Coeff.	S.E.	Coeff.	S.E.	Coeff.	S.E.	Coeff.	S.E.	Coeff.	S.E.	Coeff.	S.E.
Earthquake strong/ruinous (T=1)	43.72***	(8.63)	-.007*	(.004)					48.43***	(8.95)	-.007	(.003)
Born before earthquake (t=0)												
Third trimester gestation (t=1)	-8.32**	(2.66)	.008	(.001)	10.60***	(2.58)	-.007***	(.001)	-10.03***	(2.81)	-.008***	(.001)
Second trimester gestation (t=2)	-1.33	(2.50)	-.0003	(.001)	-1.60	(2.49)	-.0002	(.001)	-0.85	(2.60)	-.0004	(.001)
First trimester gestation (t=3)	10.65***	(2.59)	-.007***	(.001)	-8.30**	(2.66)	.001	(.001)	10.73***	(2.58)	.001	(.001)
Conceived after earthquake (t=4)	-1.31	(1.77)	.003**	(.001)	-1.70	(1.77)	.003**	(.001)	-3.67*	(1.87)	.003**	(.001)
t0 * T1												
t1 * T1	4.02	(18.05)	.002	(.008)	1.57	(18.03)	.002	(.008)	-2.00	(17.44)	.003	(.007)
t2 * T1	16.92	(17.92)	-.006	(.008)	15.02	(17.88)	-.006	(.008)	14.64	(17.62)	-.007	(.008)
t3 * T1	-50.95**	(18.83)	.018*	(.008)	-54.22**	(18.81)	.019*	(.008)	-51.50**	(20.06)	.018*	(.009)
t4 *T1	8.61	(12.32)	-.001	(.005)	5.32	(12.32)	-.001	(.005)	5.46	(12.64)	-.001	(.005)
Intercept	3311.3***	(3.11)	.052***	(.001)	3295.4***	(3.452)	.056***	(.001)	3343.2***	(1.31)	.049	(.001)
N	500,983		500,983		500,983		500,983		500,983		500,983	

[1] Sample includes all singleton infants conceived between December 2003 and March 2006 and born in Chile. * p<.05, ** p<.01, ***p<.001

[2] Linear regression model of birthweight (grams) with robust standard errors.

[3] Linear probability model of low birthweight (<2500 grams) with robust standard errors.

[4] Control for the following covariates added: Maternal age (<19 years old, 20-29, 30-34, 35 and older), education (less than college/some college or more), parity (1 birth, 2-3, 4 or more), marital status (married/non-married) and urban residence.

[5] County fixed effects added to models 1 and 2. Coefficients associated with Treatment=1 not estimated because they are perfectly collinear with county fixed-effects.

[6] Model with propensity score weights. Let PSi be the propensity score associated with the probability that a woman lives in the treated area, as predicted by observed covariates. The weight for control area women is [PSi/(1-PSi)] whereas the weight for treatment area women is 1.

Figure 1. Mean birth weight of all Chilean births by treatment status and timing. 2004-2006.

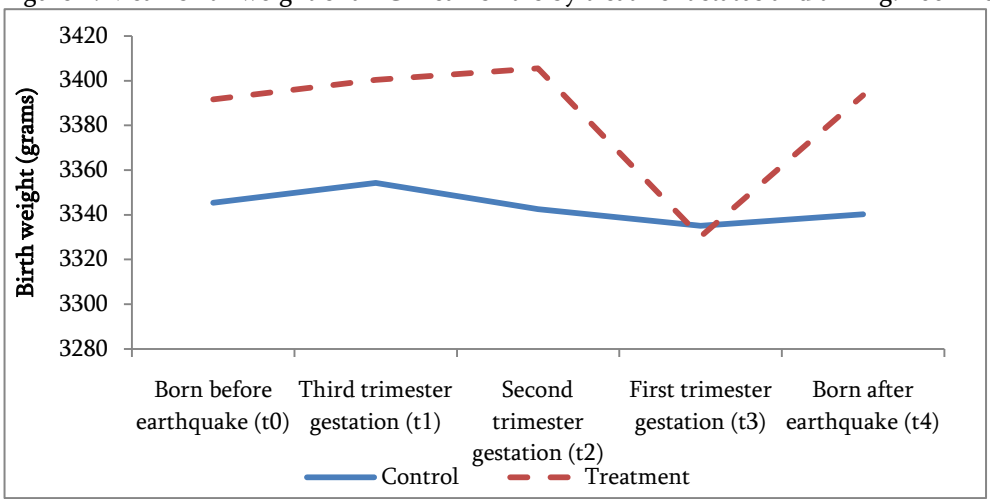

Figure 2. Mean Weeks of Gestation. Chilean births by treatment status and timing. 2004-2006.

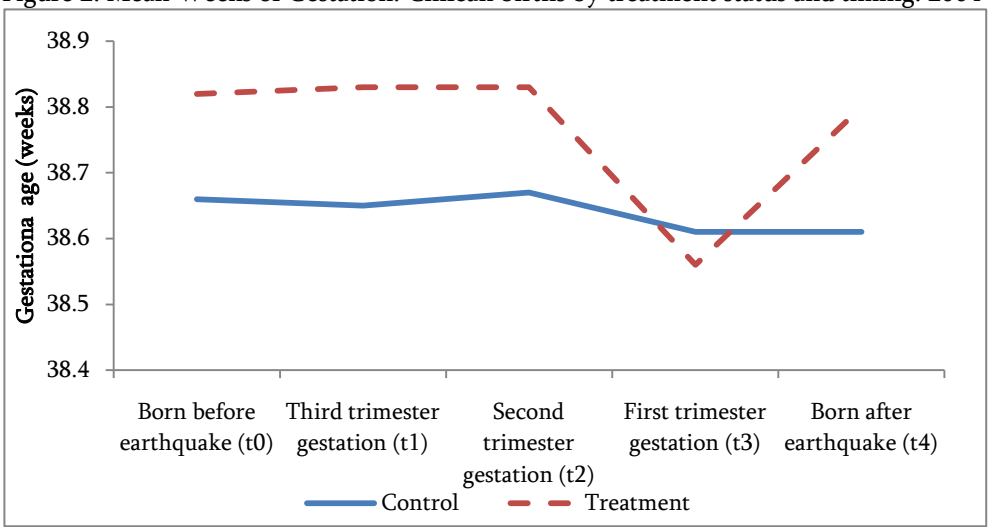

Figure 3. Birth Weight by Gestational Age. Chilean births by treatment status and timing. 2004-2006.

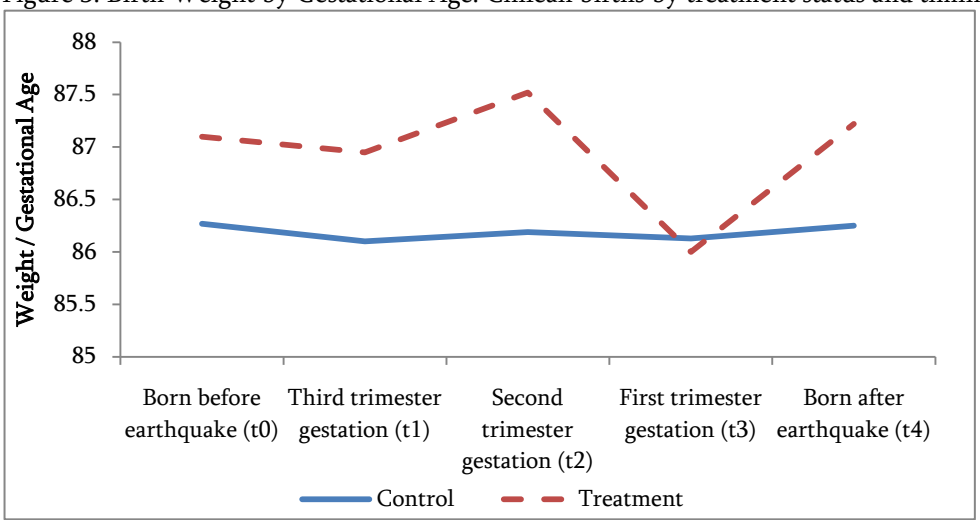

Figure 4. Birthweight by treatment status and timing with ordinal definition of treatment. Chilean Births 2004-2006.