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Prenatal Exposure to an Acute Stressor and Children's Cognitive Outcomes

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Abstract Exposure to environmental stressors is highly prevalent and unequally distributed along socioeconomic lines and may have enduring negative consequences, even when experienced before birth. Yet, estimating the consequences of prenatal stress on children's outcomes is complicated by the issue of confounding (i.e., unobserved factors correlated with stress exposure and with children's outcomes). I combine a natural experiment—a strong earthquake in Chile—with a panel survey to capture the effect of prenatal exposure on acute stress and children's cognitive ability. I find that stress exposure in early pregnancy has no effect on children's cognition among middle-class families, but it has a strong negative influence among disadvantaged families. I then examine possible pathways accounting for the socioeconomic stratification in the effect of stress, including differential exposure across socioeconomic status, differential sensitivity, and parental responses. Findings suggest that the interaction between prenatal exposures and socioeconomic advantage provides a powerful mechanism for the intergenerational transmission of disadvantage.

Keywords Prenatal stress · Cognitive ability · Natural experiment

Introduction

A growing body of research has indicated that *in utero* exposures matter for individual outcomes later in life. The seminal fetal programming hypothesis suggests that developments that enable the fetus to adapt to an adverse uterine environment may result in

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permanent programming of developmental patterns, leading to illness and early death (Barker 1990; Barker et al. 1993). Research has shown that the prenatal period has critical developmental stages that affect later cognitive and emotional development (Nijland et al. 2008; Tomalski and Johnson 2010), which in turn could shape educational attainment and socioeconomic well-being (Knudsen et al. 2006; Palloni 2006). Although Barker's (1990:1111) conjecture that "the womb may be more important than the home" may be an overstatement, it demands an empirical investigation. This study addresses two main questions about the consequences of prenatal exposures. First, does *in utero* exposure to acute stressors shape children's cognitive ability? And second, does the effect of acute prenatal stress vary by socioeconomic status (SES)?

In this study, I focus on prenatal exposure to stress because prior research suggests, but does not prove, that stress is a key factor in explaining the negative effects of poverty and disadvantage, particularly among children (Aber et al. 1997). Poverty is associated with multiple stressors, including noise, crowding, poor housing, economic instability, higher levels of family turmoil, and violence. The continuous exposure to these stressors is claimed to put pressure on children's adaptive capacities and to be toxic for the developing brain at a stage during which neurological systems are highly plastic (McEwen and McEwen 2017; Shonkoff 2010). This hypothesis is plausible but impossible to verify using observational data. The main challenge is disentangling exposure to stress from other characteristics that are unobserved and that may affect the outcome of interest, leading to confounding. To address this challenge, I combine a natural experiment—a strong earthquake affecting a region of Chile—with an instrumental variable approach and an original longitudinal survey. By exploiting a stressor arguably allocated at random, I can isolate the effect of stress from its unfortunately common correlates.

Although most research to date has focused on noxious exposures during childhood, I extend this concern to the prenatal period based on the hypothesis that the nine months spent *in utero* are critical for later development as well as highly susceptible to the environment. Most research to date has focused on chronic stress. I shift the attention to acute stress, positing that even discrete and short-term exposure to stressors experienced prenatally can have long-term consequences. I focus on children's cognitive ability for two reasons. First, cognitive ability predicts health, schooling, and earnings in adulthood (Heckman et al. 2006; Murnane et al. 1995), linking prenatal exposures with adult socioeconomic well-being. Second, because cognitive ability is stable since early in the life course and because it is correlated with socioeconomic advantage (Hackman and Farah 2009), it is an easy target for the misleading interpretation that it is determined only by genetic factors passed across generations.

Prenatal Exposure to Environmental Stressors: Why Does it Matter?

Because stress is a broad concept used by experts and laypeople alike, definitions are necessary at the outset. Following Kugelmass and Lynch (2014:1), stressors are defined as a "wide range of conditions, forces, and experiences with the potential to challenge the adaptive capacities of individuals." The term *stress* describes an overarching process that begins with exposure to a stressor and ends with its manifestation, usually termed *distress*. As I discuss in detail later, this analysis examines a specific dimension

of the stress process: the population-level effect of prenatal exposure to an acute environmental stressor.

The stress process is an important concern for social scientists because exposure to stressors is both highly prevalent and unequally distributed. Socioeconomic disadvantage is associated with a higher exposure to stressors and fewer resources to cope successfully (Aneshensel 1992; McLeod and Kessler 1990). Both long-term stressors (such as economic strain and discrimination) and acute stressors (such as adverse life events) have been shown to be more prevalent among the disadvantaged (Pearlin et al. 2005; Turner et al. 1995).

In the stress literature, the distinction between chronic and acute stress is relevant. Early post-war research was based on a biological stimulus response model (Dohrenwend and Dohrenwend 1970; Selye 1956). Stressors—viewed as life events and understood as discrete, directly measurable circumstances with a limited time frame—were termed *acute stressors*; examples are being laid off or being a crime victim. An alternative perspective suggested understanding stress as a long process of excessive load and fatigue, leading systems to collapse without the need of a triggering event (Wheaton and Montazer 2009). Applied to human populations, this perspective highlighted long-term exposures emerging, for example, from persistent economic disadvantage, living in dangerous neighborhoods, or racial discrimination (Pearlin 1999; Thoits 2010).

This type of persistent exposure came to be known as *chronic stressors* (Pearlin et al. 1981). Whereas acute stress came to be regarded as an adaptive and beneficial response, at least in the short term (Schneiderman et al. 2005), chronic stress came to be seen as particularly maladaptive and toxic. Concepts such as cumulative biological risk factors (Evans et al. 2013; King et al. 2011), allostatic load (McEwen 1998; McEwen and Stellar 1993), and weathering (Geronimus 1992) highlight the wear and tear across the multiple systems of the body that result from continuous, repeated, or cumulative exposure to a number of stressors. Social sciences research has further focused attention on chronic stressors' harmfulness by showing that chronic stress has a stronger negative association with physical and mental health than acute stress and that it is more strongly stratified along socioeconomic lines (Pearlin 1989, 1999; Thoits 1983; Turner 2010; Turner and Avison 2003).

A New Understanding of Human Development and the Relevance of Acute Stress

Although a focus on chronic stress is warranted, acute stress may produce long-lasting consequences when experienced in the prenatal period for two reasons. First, as the first years of life, the prenatal period contains sensitive and critical developmental stages (Nijland et al. 2008; Tomalski and Johnson 2010). Second, because human skills develop in a hierarchical and complementary manner, later attainment builds on earlier stages (Cunha et al. 2006; Heckman 2006).

Sensitive periods are limited developmental stages in which the effect of the environment on a certain capability is stronger (Knudsen 2004). Critical stages are particularly brief and discrete sensitive periods in which the environment may have irreversible effects on a certain capability, regardless of subsequent

experience (Brown 2005). The notion of a critical period suggests a window of developmental opportunity, as in the case of imprinting in animals, first and second language acquisition in humans, and critical developments of the central nervous system and the brain during early life (Rice and Barone 2000).

Although these findings suggest that prenatal exposure to acute stressors could have long-lasting consequences, they do not offer a mechanism for their persistent influence over the life course. A new hierarchical understanding of human development as characterized by self-productivity and dynamic complementarity offers such a mechanism (Cunha and Heckman 2007). *Self-productivity* refers to the facts that capabilities produced at one stage augment the skills attained at later stages and that capabilities are self-reinforcing and cross-fertilizing. For example, emotional security fosters better health, which in turn may promote learning. *Dynamic complementarity* means that capabilities acquired at one stage of the life course raise the productivity of investment at subsequent stages: for example, mastering basic math concepts makes learning more complex concepts easier. This recent understanding of human development suggests that stress experienced before birth, even short-term, may produce enduring effects throughout the early life course. This understanding invites an empirical examination of the influence of acute stress experienced *in utero*.

The Effect of Prenatal Stressor Exposure on Childhood Outcomes.

To date, the literature on prenatal stress has focused on its effects on birth outcomes, such as low birth weight and prematurity. This focus is understandable. Birth outcomes have consequences for later health, development, and well-being (Alderman and Behrman 2006; Conley et al. 2003), and they identify the infants who are most at risk for mortality, morbidity, and developmental problems (Kline et al. 1989). Also, examining birth outcomes requires birth record data, which are easy to obtain and of high quality. In contrast, examining outcomes during childhood requires longitudinal data, a much taller order. However, birth outcomes provide only a rough indicator of a child's resources at the beginning of life. Recent studies have suggested that the influence of *in utero* stress may extend into childhood, affecting motor skills, cognitive ability, and early educational achievement and causing emotional problems (Beydoun and Saftlas 2008; Huizink et al. 2003; Van den Bergh et al. 2005; Weinstock 2008). Given the relevance of these developmental outcomes for socioeconomic well-being, these findings suggest that prenatal stress can have long-lasting consequences. To date, however, this evidence is mostly associational and invites the question about the effect of prenatal stress.

The literature highlights several mechanisms linking prenatal stress with children's cognitive development, including neuroendocrine, immune/inflammatory, vascular, behavioral, and epigenetic (Beijers et al. 2014; Beydoun and Saftlas 2008; Hobel et al. 2008). Maternal stress has been implicated in the production of so-called stress hormones, including corticotrophin-releasing hormone (CRH), adrenocorticotrophic hormone (ACTH), and cortisol in the mother, the placenta, and the fetus (Charil et al. 2010; Gutteling et al. 2006; Van den Bergh et al. 2005). Elevated levels of these stress hormones may delay nervous system maturation, diminish gray matter volume, and impair brain development (Davis and Sandman 2010; Sandman et al. 2011). Although

evidence in humans is still limited, research on nonhuman primates has suggested that stress depletes the placenta's ability to protect the fetus against noxious maternal cortisol, increasing the risk of neurological impairments (Avishai-Eliner et al. 2002; Uno et al. 1994). Stress has also been found to be correlated with decreased blood flow in the uterine artery that is crucial for fetal development (Sjostrom et al. 1997); a decline in immune function and inflammation in the mother (Beijers et al. 2014); and higher-risk health behaviors, such as cigarette smoking and unhealthy eating (Dunkel Schetter and Glynn 2011; Lobel et al. 2008; Umberson et al. 2008). Recent research has highlighted the importance of epigenetic changes as a potential mechanism linking prenatal stress exposure to cognitive function in childhood. A growing literature suggests that epigenetic transformations—molecular transformations in gene expression that do not involve changes in underlying DNA sequence—may account for the influence of early-life environmental exposures on later health and development (Benyshek 2013; Kuzawa and Sweet 2009; Non et al. 2016; Thayer and Kuzawa 2011). Findings from animal models and, increasingly, human studies suggest that the epigenetic regulation of gene expression through processes such as methylation and histone modification could play a critical role in in the programming effects of early-life stress on neural circuitry and brain development (Bock et al. 2015; Monk et al. 2012).

The timing of stressor exposure is important. Early- and mid-pregnancy stress appear to predict children's cognitive outcomes, such as cognitive ability and attention disorders. During early pregnancy, the developing brain is susceptible to alteration in its programming because neurons are still immature (Welberg and Seckl 2001), and many brain areas undergo a cascade of timed processes of neuron proliferation, migration, early differentiation, and sometimes death (Van den Bergh et al. 2005; Weinstock 2008). Subtle alterations in these processes may disturb brain development. In contrast, children's emotional problems—such as anxiety, fearfulness, aggression, and depression—appear to be more sensitive to late pregnancy exposures because the developing brain undergoes rapid growth and is particularly susceptible to a reduction in oxygen and nutrients (O'Connor et al. 2002).

Socioeconomic Stratification: Does the Effect of Prenatal Exposure to Acute Stressors Vary by Socioeconomic Advantage?

The sociological stress model situates personal experiences of stress in the broader social context, examining how position in the stratified social structure shapes exposures and responses to stressors (McLeod et al. 2014; Pearlin et al. 1981). A central implication from this model is that characteristics of the social context could moderate—that is, reduce or intensify—the influence of early exposure to stressors on later outcomes.

Although some scholars have warned us that "a child who falls behind may never catch up" (Heckman 2006:1900), individuals exposed to early insults show substantial variation. Strong evidence comes from studies of epigenetic transformations showing that postnatal experiences may have a critical moderating influence on prenatal effects (Monk et al. 2012) and from studies of orphanage-rearing showing that orphans exposed to severe adversity and deprivation experience remarkable cognitive and emotional recovery once adopted into families (Duyme et al. 1999; Rutter 1998).

These insights highlight the crucial role that the social context could play in shaping the effect of early exposures.

In this analysis, I consider whether family SES moderates the effect of prenatal exposure to an acute stressor. I hypothesize that the effect of exposure will be stronger among disadvantaged families than among their more advantaged counterparts. The literature suggests at least three mechanisms that could account for a stronger effect among poor families: differential exposure, differential sensitivity, and differential parental responses.

Differential Exposure

Socioeconomic advantage may shape the ability of families and communities to protect themselves from environmental stressors such as natural disasters, armed conflict, pollution, and violence. Many factors could result in poor families' greater exposure to environmental stressors. As examples, poor families are likely to reside in more vulnerable areas and in worse-quality dwellings, and to be unable to afford temporary relocation or supplies. This variation will result in differential dosages of stress across SES, even if the stressor is nominally the same for the entire population. This variation has been conceptualized as a methodological nuisance because it violates the assumption of a homogeneous treatment effect that is necessary for causal inference (Rubin 1990; VanderWeele and Hernan 2013). However, the variation is substantively relevant, and it invites the question about differential exposure depending on individual, family, or community resources.

Differential Sensitivity

Even if exposure does not vary along socioeconomic lines, disadvantage may heighten the sensitivity to environmental stressors (McLeod and Kessler 1990; Turner et al. 1995). The theoretical approaches of allostatic load, weathering, and cumulative risk factors suggest that chronic stress emerging from socioeconomic disadvantage may act as a predisposing factor for the influence of acute stressors: that is, a novel stressor will cause more damage to an individual already debilitated by chronic exposures (McEwan and Stellar 1993). However, the opposite hypothesis is also plausible. Exposure to socioeconomic disadvantage may result in reduced reactivity to novel stressors through a protective mechanism variedly termed habituation, inoculation, and adaptation (Eysenck 1983; Feder et al. 2009; Gump and Matthews 1999; Kirshbaum et al. 1995).

Differential Parental Responses

An emergent literature suggests that parental responses may compensate for the consequences of early-life shocks and that compensatory responses among advantaged families provide a strong mechanism for transmitting advantage across generations (Almond and Mazumder 2013; Bernardi 2014). Ethnographic research has shown that parenting approaches and practices are rooted in differential access to economic, social, and cultural resources as well as in a class-based sense of entitlement and familiarity with institutional dynamics, resulting in stratified parenting styles (Lareau 2011). As a result, upper- and middle-class parents may be more able to mobilize resources to

mitigate the effect of adverse exposures. In contrast, disadvantaged parents may be more likely to concentrate on providing basic material and emotional support but have limited resources to relate to relevant institutions (such as schools) and devote less time to the explicit development of cognitive ability. Empirical evidence has provided indirect support for this claim, showing that children's birth weight has a stronger effect on later cognitive and educational outcomes among poor families than among better-off families (Torche and Echevarria 2011), and suggesting that advantaged parents compensate for the negative consequences of an earlier adverse outcome (Bernardi 2014; Conley 2004; Hsin 2012).

Stratification of stress exposure, sensitivity, and parental responses provide alternative mechanisms that would result in a stronger effect of prenatal stress exposure on children's outcomes among disadvantaged families. These factors are not mutually exclusive or exhaustive, and to the best of my knowledge, no empirical study has tested their relative importance. Although I cannot fully verify these mechanisms with the data at hand, I offer these hypotheses as an initial attempt to examine the multiple ways in which the effect of early exposures could vary by socioeconomic advantage.

Capturing the Effect of Prenatal Exposure to Acute Stressors: The Challenge of Confounding

Many studies have documented the association between prenatal stress and children's cognitive outcomes (Beydoun and Saftlas 2008; Entringer et al. 2015; King and Laplante 2005; Tarabulsy et al. 2014; Weinstock 2008). However, these studies cannot rule out the role of unobserved factors that are correlated with both stress exposure and children's outcomes. As a review of the literature candidly states, "there could be other . . . effects that account for these associations, ranging from shared genetic variance to indirect behavioral mechanisms . . . a number of potential third variables (remain) that might explain the apparent association" (Talge et al. 2007:252).

A strategy for addressing the challenge of confounding is the use of natural experiments—events occurring in the physical or social world that are allocated as "at random" within a particular population and thus are not correlated with unobserved maternal or ecological characteristics; a sudden economic decline, a natural disaster, or a drastic change in social policy are examples (Dunning 2012). Natural experiments have been used to study the effect of prenatal stress on birth outcomes, using stressors such as economic contraction (Margerison-Zilko et al. 2011), a natural disaster (Torche 2011), a terrorist attack (Eskenazi et al. 2015), racial discrimination (Lauderdale 2006), and an immigration raid (Novak et al. 2017). A seminal attempt to examine the effect of prenatal stress over the early life course is the Project Ice Storm, which exploits a major winter storm affecting Quebec in 1998 to assess outcomes of children exposed *in utero*. The project recruited approximately 200 exposed pregnant women and has followed their children since birth up to early adolescence (King and Laplante 2005; Laplante et al. 2008). The ice storm study provides a blueprint for further research, but it has limitations that highlight the methodological challenges of this type of study, including a small sample size, the lack of a control group, and the nonrandom nature of the sample. By combining a natural experiment with a panel survey, this study addresses these limitations and offers a study of the effect of prenatal stress over the early life course.

Methods

I exploit a strong earthquake as a natural experiment. The Tarapacá earthquake hit the northernmost region of Chile on June 13, 2005, reaching a magnitude of 7.9 on the moment-magnitude (MM) scale (considered extremely high). The use of this natural disaster as an instrumental variable for stress requires satisfying several assumptions (Angrist et al. 1996). First, the earthquake needs to occur randomly. Although the entire Chilean territory is prone to earthquakes, current technology does not predict when and where a tremor will occur. As a result, the earthquake was essentially a random exposure within the Chilean population.

Second, the earthquake needs to be correlated with stress. It is well documented that earthquakes are a source of acute anxiety, signaled by such health indicators as acute cardiac events and stroke (Dimsdale 2008; Leor et al. 1996), changes in brain function (Lui et al. 2009), and population reports of distress and anxiety following the disaster (Siegel 2000).

Third, the exclusion restriction assumption requires that the earthquake's effect on children's cognitive outcomes occur entirely through stress, without alternative pathways of influence. The exclusion restriction is the main reason for selecting this event from the many disasters that have occurred around the world in the recent past. The consequences of most natural disasters are devastating and wide. They include loss of life and property, displacement, destruction of infrastructure, pollution, and creating a public health emergency, among other corollaries: think Hurricane Katrina in the United States in 2005, the 2010 Haiti earthquake, or the 2011 Tōhoku earthquake and tsunami in Japan. In contrast, the Tarapacá earthquake had few spillover effects, despite its violence. In terms of lives and property damage, the earthquake's toll was small: 11 people died, 130 were injured, 180 residences were destroyed, and 0.035 % of the population had to temporarily relocate to shelters (ONEMI 2005). This limited damage was the result of seismic preparedness and low population density. Chile has enforced a stringent building code for decades, and much of its infrastructure uses earthquake-proof technology (Hidalgo and Arias 1990). Low population density minimized problems associated with human concentration in disaster-affected areas. The earthquake also had little impact on employment. The unemployment rate in the June–August quarter of 2005 increased to 12 % compared with 11 % for the same quarter a year earlier; this trend was not distinct from the rest of the country. Displacement was minimal because the earthquake damaged sparsely populated rural villages most heavily, which accounted for less than 8 % of the population and less than 3 % of the births in the affected region (Earthquake Engineering Research Institute 2005). The consequences for population health in terms of acute respiratory infections and other communicable diseases were also minor. These limited spillover effects suggest that the main corollary of earthquake exposure was heightened levels of stress and anxiety.

The fourth assumption for using the Tarapacá earthquake as an instrument for stress is monotonicity: that is, the earthquake should increase stress (or have no effect) among those exposed, but it cannot increase stress for some while reducing stress for others. Despite no formal test of this assumption, it would be extremely unlikely for a strong earthquake to have reduced the amount of stress among exposed individuals. Finally, the stable unit treatment value assumption (SUTVA) states that the effect of being exposed to the earthquake or experiencing stress on any particular individual should not depend on others

being exposed or experiencing stress. This "no interference" requirement is most likely violated in this setting, as it is in most social settings in which individuals are embedded in social networks of influence and interaction that could ameliorate or exacerbate the individual-level effect of exposure. I return to this assumption in the conclusions.

The instrument is defined as the intensity of the earthquake in the county where mothers resided during their pregnancy. The moment-magnitude scale measures energy released, but the modified Mercalli intensity scale evaluates the effect of the earthquake on the earth's surface, humans, objects of nature, and human-made structures; that is, the scale evaluates the earthquake as experienced by the population. It uses a 12-point ordinal scale with the following categories: instrumental (I), feeble (II), slight (III), moderate (IV), rather strong (V), strong (VI), very strong (VII), destructive (VIII), ruinous (IX), disastrous (X), very disastrous (XI), and catastrophic (XII). This last category, which has never been observed, denotes total destruction. The Tarapacá earthquake intensity ranged from I (instrumental) to IX (ruinous). Counties with an intensity of very strong to ruinous (VII–IX) are defined as the treated area because its lower bound defines a categorical boundary at which the earthquake is felt by the entire population and damage starts occurring (Ramirez and Peek-Asa 2005). The control group is defined as counties with similar demographic and socioeconomic characteristics as the treated counties but with an earthquake intensity that is moderate or less. The control counties selected were located in the center-north region of the country.

Data

I combine data from two sources. The first source is the file of Chilean birth certificates for 2004, 2005, and 2006 established by the Chilean Ministry of Health. Each record includes information on infant and mother's characteristics as well as the mother's county of regular residence when children were born. The second data source is the Children of the Earthquake panel survey, fielded in 2013/2014, when the children were approximately age 7. Using Chilean birth registry data as a sampling frame, the Ministry of Education supplied information from the schools that the children born in the treated and control counties attended, with children's individual identities masked. A multistage sampling design was used. In the first stage, schools were randomly selected with probability proportional to size. A varying number of students within schools was then randomly selected in order to achieve a self-weighted sample. The sample size is 1,149 with 591 cases in the treated area and 558 in the control area. Importantly, randomly selected children were located and included in the sample regardless of whether they had moved since birth, thus avoiding producing a selected sample. The data collection instruments consisted of a questionnaire given to the mothers or primary caregivers and an assessment of the children's cognitive and executive function. All instruments were used with the entire sample, including both the treatment and control groups.

Dependent Variable

The main dependent variable is the children's cognitive ability, measured by the Wechsler Intelligence Scale for Children-Third Edition (WISC-III), adjusted for the Chilean context (Ramirez and Rosas 2007). The WISC-III contains 13 subtests. Given time constraints, I use eight subtests: similarities, arithmetic, vocabulary, digit span,

coding, block design, symbol search, and mazes. This subset has good properties in terms of validity and reliability (Campbell 1998; Kaufman et al. 1996). These items are combined to obtain measures of overall IQ, verbal IQ (first four subtests), and performance IQ (latter four subtests). Verbal and performance ability measures capture different domains, which are potentially affected differentially by prenatal exposures. The verbal scale focuses on language, reasoning, and memory skills, and the performance scale measures spatial, sequencing, and problem-solving skills.

Analytical Approach

I use a difference-in-differences (DID) methodology to capture the causal effect of exposure to an acute stressor at the population level, using the differences in mean outcomes over time and across treatment groups.[1] I distinguish two groups based on treatment assignment status: t_1 is an indicator variable coded 1 for children who were born in the treatment area (as determined by the Mercalli scale) and 0 for children who were born in the control area and were *in utero* during the same period as the treatment group. Given the potential importance for brain development of the timing of prenatal exposure to an acute stressor, I distinguish exposure by trimester of gestation, where d_1, d_2, and d_3 are indicator variables that identify exposure to the earthquake in the first, second, and third trimester of gestation, respectively.

The outcome Y is modeled by Eq. (1):

$$Y = \beta_0 + \beta_1 t_1 + \beta_2 d_1 + \beta_3 d_2 + \beta_4 (t_1 \times d_1) + \beta_5 (t_1 \times d_2) + \varepsilon, \qquad (1)$$

where Y is the child's cognitive ability; β_1 is the treatment group-specific main effect, which captures differences in the outcome between treatment and control groups; and β_2 and β_3 capture changes over time in the outcome that are common to control and treatment groups. The terms β_4 and β_5 capture the effect of earthquake exposure in the first and second trimester of exposure, respectively. These terms, β_4 and β_5, measure the difference (between treatment and control groups) in a difference (across trimester of exposure), giving the DID estimator its name; and they capture the difference in children's cognitive ability between the treatment and control groups between trimesters of exposure. By including an effect for the treatment group, the model accounts for any systematic differences across treatment groups that emerge, for example, due to the socioeconomic characteristics of the population, altitude, or preschool and educational institutions, which are captured by the parameter β_1. By including an effect for timing of exposure, the model controls for any trends that may affect both treatment groups, including season of birth, the economic cycle, or any historical event affecting the entire country, which are captured by the parameters β_2 and β_3. All models use robust standard errors clustered at the school level to account for the potential correlation of the errors within schools. Seven observations (0.6 % of the sample) contained missing values for one or more variables and were excluded from the analysis, reducing the analytical sample to 1,142.

The DID formulation requires that one of the treatment status groups and one of the timing-of-exposure groups be excluded and used as a baseline for comparing the

[1] For simplicity, I refer to treatment and control groups based on exposure to the earthquake, although it should be kept in mind that the earthquake is the instrument for prenatal stress.

estimation. As Eq. (1) shows, the excluded group based on treatment status is the control group, and the excluded trimester of exposure is the third trimester. As a result, the effect of earthquake exposure in the first and second trimesters is compared with that of the third trimester; I assume that there is no effect—positive or negative—associated with exposure during this period. The rationale for this decision is the aforementioned research indicating that the effect of prenatal stress on cognitive development likely emerges from early-pregnancy (and perhaps also mid-pregnancy) exposure but not from late-pregnancy exposure. If there were a negative effect associated with late-pregnancy exposure to stress, this analysis would underestimate the effect of the stressor because the third trimester of gestation, used as the baseline for comparison, would also be negatively affected by the earthquake. However, if third trimester exposure to the earthquake had a positive effect on cognitive ability, the parameter estimates for first and second trimester exposure would provide an overestimation of the negative effect because it will be compared with a positive-effect baseline. The latter possibility is extremely unlikely. There is no theoretical rationale or empirical evidence in either animal or human studies suggesting a positive effect of late-pregnancy acute stress on cognitive outcomes.[2]

The unbiasedness of the DID approach rests on the parallel trends assumption, which requires that there be no treatment group–specific trends that could bias the estimates of treatment effects. This assumption is highly plausible in this setting because an earthquake is a random occurrence in a national context in which the entire territory is prone to earthquakes. To further account for differences between the treatment or control populations as well as compositional change over time, all models include controls for mothers' and children's characteristics. Mother's characteristics were all obtained from the birth certificate to ensure their exogeneity and include mother's age, education (less than a high school diploma, high school graduate, college or more), and marital status at the time of the child's birth (married, unmarried). Children's attributes include sex, age in months at the time of test-taking, and its square. Table 1 offers descriptive statistics for the treatment and control areas.

In this setting, to bias the observed effect, a systematic difference between treatment and control groups should be (1) correlated with the precise timing of exposure, (2) correlated with the outcome, and (3) uncorrelated or weakly correlated with observed controls. These conditions are implausible. Still, two sources of selectivity could emerge as a result of earthquake exposure. The first one is migration: some women might have left the area in response to the earthquake such that they would not have been observed in the treatment group. The second one is fetal loss: if acute stress had resulted in miscarriages or spontaneous abortions, some pregnancies would not be observed as births. These responses could alter the population composition of births, thus introducing selectivity. For example, healthier women leaving the area, and leaving behind their more vulnerable peers, would induce negative selectivity. Ancillary analyses, included in the online appendix (section A1), indicate that neither source of selectivity is likely to be at play in this case.

[2] The influence of small increases in cortisol within a normal range late in the pregnancy is subject to debate, with some studies reporting small negative effects and others reporting small positive effects (Davis and Sandman 2010; Huinzik et al. 2003).

Table 1 Descriptive statistics: Means (with standard deviations in parentheses) and percentage distribution of analytical variables in the treatment and control areas

	Control Area		Treatment Area	
Mother's Age	27.05	(6.77)	27.04	(6.71)
Mother Is Married	0.34	(0.47)	0.36	(0.48)
Urban Residence	0.97	(0.16)	0.97	(0.17)
Mother's Education (%)				
Less than high school	33.51		32.88	
High school graduate	41.40		39.49	
College	25.09		27.63	
Child Is Male	46.67		50.93	
Child's Age (months)	90.99	(3.55)	91.10	(3.63)
Number of Observations[a]	555		587	

[a] Excludes seven observations with missing data in one or more variables used in the analysis.

Before moving to the analysis, it is important to take stock on the attributes of this design to capture causal effects. In an ideal design, the researcher would randomly allocate stress to some pregnant women but not others in the controlled setting of an experimental laboratory, would wait for several years, and would then evaluate their children's outcomes. This strategy is naturally impossible and undesirable. I rely on a setting that provides an as close as possible parallel to the random allocation of stress in real life. Even if this design departs from the laboratory context, this natural experiment provides a naturalistic setting that overcomes artificiality concerns associated with manipulation and control. Because the treatment is measured for the population of children exposed to the earthquake and the stress response cannot be measured at the individual level, this analysis offers a reduced-form effect at the aggregate level. In instrumental variable parlance, the effect captured is an *intent to treat*—that is, the effect of experiencing the earthquake (instrument) rather than of experiencing stress (treatment). The effect captured is an average that may contain heterogeneity across the population.

The second step of this analysis examines one crucial dimension of heterogeneity, family SES. I measure SES by mother's education, with three categories: less than high school, high school graduate, and some college or more. The effect of the stressor is evaluated separately across the three socioeconomic groups. Finally, the third step of the empirical analysis examines the potential mechanisms, identified previously, for stratifying the effect of prenatal stressor exposure: differential exposure, differential sensitivity, and differential parental responses.

Findings

The Effect of Prenatal Exposure to a Stressor on Children's Cognitive Ability

Table 2 offers models examining the effect of prenatal stressor exposure on children's subsequent cognitive ability. Column 1 shows the model for verbal ability, and columns

2 and 3 show models for performance and overall cognitive ability, respectively. Measures of cognitive ability are standardized so that parameter estimates can be interpreted using the standard deviation metric. As Eq. (1) specifies, the indicator variable identifying the treated area captures the main effect of residing in this area; the indicators for trimester of gestation 1 and 2 capture, respectively, the main effect of being exposed to the stressor in the first or second trimester of gestation (using the third trimester as the reference category). The effects of interest are captured by the interaction of the treatment group with the timing of exposure; these are presented in Fig. 1, along with 95 % confidence intervals.

Table 2 and Fig. 1 address this study's first question: Does prenatal exposure to an acute stressor have a negative effect on the children's cognitive ability? Exposure to the earthquake in the first trimester of the pregnancy results in a decline in verbal ability by 0.132 standard deviations, a decline in performance ability of 0.053 standard deviations, and a decline of 0.117 standard deviations in overall ability (effects are much smaller for second trimester exposure).[3] As expected, the effect of stress exposure early in the pregnancy is negative when compared with exposure in the third trimester, but it is substantively small and fails to reach significance at the conventional $p < .05$ level. The conclusion from this analysis, then, is that the evidence is not consistent with a substantial detrimental effect of prenatal exposure to an acute stressor on children's cognitive ability.

However, this analysis has proceeded with the likely naïve assumption that the effect is homogenous across the population. The second analytical step relaxes this assumption and raises the question about socioeconomic stratification of the effect of prenatal exposure to stress. To address this question, I stratify the sample by family SES as defined by the three groups outlined earlier: mothers with less than a high school diploma (33 % of the sample), who are high school graduates (40 % of the sample), or who completed some college education or more (27 % of the sample). Table 3 offers parameter estimates, standard errors, and significance tests; Fig. 2 plots the parameter estimates of interest comparing the effect of prenatal stress exposure across family SES (interaction terms between first trimester and treated area across levels of schooling in Table 3).

The results show marked socioeconomic heterogeneity in the effect of prenatal stress. Although second trimester exposure has no effect on children's cognitive ability for any socioeconomic group, the effect of first trimester exposure varies considerably by family SES. Among disadvantaged families—those in which the mother has less than a high school diploma—exposure to the acute stressor in the first trimester of gestation results in a decline in verbal ability by 0.457 standard deviations. The decline in cognitive ability reaches 0.676 standard deviations for performance ability, and 0.622 standard deviations for overall ability when comparing the difference in ability among children in the treated and control region for those exposed in the first trimester of gestation with the same difference for those exposed in the third trimester. Among more advantaged families, by contrast, the effect is indistinguishable from 0 at

[3] There is also a significant overall difference in cognitive performance between treatment and control areas, captured by the parameter estimate associated with the treatment area. Interviews with local experts suggested that regional differences in quality of preschool and early education institutions may play a role in this baseline difference.

Table 2 Difference-in-differences model of the effect of prenatal earthquake exposure on children's cognitive ability at age 7: Verbal ability, performance ability, and overall ability

	Verbal Cognitive Ability	Performance Cognitive Ability	Overall Cognitive Ability
Trimester 1	0.203	0.276	0.264
	(0.193)	(0.182)	(0.188)
Trimester 2	0.055	0.201	0.130
	(0.141)	(0.139)	(0.139)
Trimester 3 (ref.)			
Treated Area	–0.281**	–0.184	–0.276**
	(0.120)	(0.124)	(0.129)
Trimester 1 × Treated	–0.132	–0.053	–0.117
	(0.116)	(0.122)	(0.117)
Trimester 2 × Treated	0.083	–0.031	0.044
	(0.165)	(0.133)	(0.155)
Trimester 3 × Treated (ref.)			
Mother's Age	–0.005	0.001	–0.003
	(0.005)	(0.006)	(0.005)
Mother Is Married	–0.003	0.067	0.031
	(0.055)	(0.078)	(0.068)
Mother's Education Is Less Than High School (ref.)			
Mother's Education Is High School Graduate	0.337***	0.299***	0.364***
	(0.072)	(0.083)	(0.078)
Mother's Education Is College	0.659***	0.479***	0.671***
	(0.089)	(0.097)	(0.098)
Male	0.093*	0.147***	0.136***
	(0.056)	(0.051)	(0.050)
Age in Months	–0.507	2.471	0.797
	(5.740)	(6.099)	(5.928)
Age in Months, Squared	0.000	–0.002	–0.001
	(0.005)	(0.006)	(0.005)
Constant	152.3	–656.4	–200.0
	(1,575.0)	(1,674.7)	(1,626.5)
Number of Observations	1,142	1,142	1,142

Note: Numbers in parentheses are standard errors.

*$p < .05$; **$p < .01$; ***$p < .001$

the $p < .05$ level and is substantively small.[4] Fig. 2 plots the focal parameter estimates and shows that the differences in the effects of prenatal stress exposure between poor families and advantaged families is significantly different from 0 for performance and overall ability (and close to significance at the $p < .05$ level for verbal ability).

[4] Substantive results remain unaltered if models include a larger set of covariates (Table A2.1 in the online appendix).

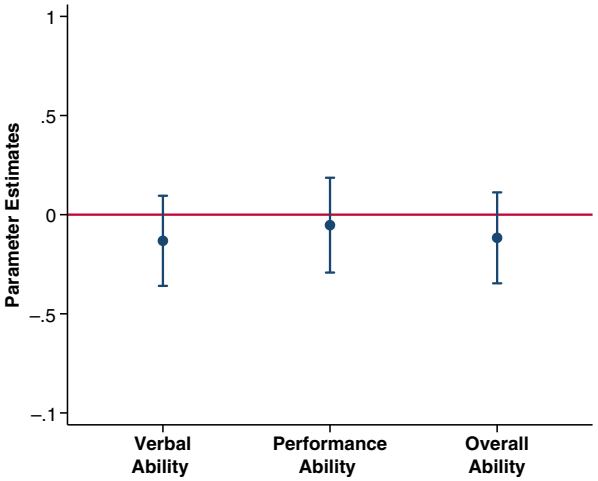

Fig. 1 Effect of prenatal exposure to earthquake in first trimester of gestation on children's cognitive ability at age 7: Verbal ability, performance ability, and total ability (WISC-III). Solid dots are parameter estimates; vertical bars are 95 % confidence intervals. *Source:* Table 2

Moving from statistical to substantive significance, it is important to evaluate the magnitude of the negative effect in cognitive ability among poor families. I use two benchmarking strategies. First, I compare this effect with the association between mother's education and child's cognitive ability reported in Table 2. Based on Table 2, children of mothers with a college degree have, on average, 0.671 standard deviation higher cognitive ability compared with the children of mothers with less than a high school diploma (the reference category). The effect of acute prenatal stress exposure on children's ability among poor families is comparable with this gap in mother's education, which arguably represents a vast distance in terms of socioeconomic and cultural resources. As a second strategy, I compare the effect of prenatal stress exposure with the effect of early childhood interventions intended to boost cognitive ability. The effect of prenatal stress exposure on children's cognitive ability is comparable with the largest measured effect of renowned program interventions, including the Carolina Abecedarian Project (effect size = .620), the Perry Preschool Project (effect size = .970), the Chicago Child-Parent Centers program (effect size = .350), and the Houston Parent-Child Development Center program (effect size = .520) (Karoly et al. 2005). The effect of prenatal stress exposure on children's ability is also larger than conditional cash transfers making sizable payments to poor families in Latin American countries, including Nicaragua (effect size = .100; Macours et al. 2012) and Mexico (effect size = .110; Fernald et al. 2009). Naturally, these studies vary in design, age of measurement, and contents of the interventions. Nevertheless, they provide a general yardstick suggesting that the negative effect of prenatal stress exposure is substantial.

What Accounts for the Stratification in the Effect of Prenatal Stress Exposure on Children's Cognition?

The finding of a stratified effect in prenatal exposure to an acute stressor raises the question about the factors that account for socioeconomic stratification. Based on the

Table 3 Difference-in-differences model of the effect of prenatal earthquake exposure on cognitive ability at age 7 by family socioeconomic status (measured by mother's education)

	Verbal Cognitive Ability			Performance Cognitive Ability			Overall Cognitive Ability		
Mother's Education:	Less Than High School	High School Graduate	College	Less Than High School	High School Graduate	College	Less Than High School	High School Graduate	College
Trimester 1	–0.302	0.514**	0.233	0.142	0.314	0.324	–0.147	0.499*	0.308
	(0.325)	(0.255)	(0.359)	(0.367)	(0.307)	(0.308)	(0.349)	(0.258)	(0.343)
Trimester 2	–0.179	0.023	0.273	0.141	0.193	0.191	–0.057	0.107	0.268
	(0.255)	(0.198)	(0.198)	(0.249)	(0.227)	(0.226)	(0.262)	(0.192)	(0.191)
Trimester 3 (ref.)									
Treated Area	0.002	–0.401**	–0.472**	0.273	–0.320*	–0.514**	0.123	–0.418**	–0.558***
	(0.176)	(0.167)	(0.187)	(0.198)	(0.162)	(0.207)	(0.195)	(0.174)	(0.206)
Trimester 1 × Treated	–0.457**	0.044	0.021	–0.676***	0.187	0.249	–0.622***	0.117	0.116
	(0.212)	(0.234)	(0.249)	(0.234)	(0.198)	(0.284)	(0.217)	(0.236)	(0.271)
Trimester 2 × Treated	–0.369	0.246	0.372	–0.335	0.188	0.032	–0.389	0.253	0.260
	(0.282)	(0.211)	(0.265)	(0.231)	(0.218)	(0.231)	(0.275)	(0.213)	(0.258)
Trimester 3 × Treated (ref.)									
Mother's Age	0.004	–0.009	–0.008	–0.005	–0.000	0.015*	0.000	–0.006	0.001
	(0.007)	(0.008)	(0.010)	(0.008)	(0.009)	(0.008)	(0.008)	(0.009)	(0.008)
Mother Is Married	–0.092	0.151	–0.132	0.134	0.155	–0.101	–0.000	0.177	–0.135
	(0.108)	(0.099)	(0.093)	(0.134)	(0.134)	(0.102)	(0.118)	(0.117)	(0.098)
Male	0.183	0.077	0.070	0.128	0.246***	0.054	0.190*	0.170**	0.076
	(0.111)	(0.086)	(0.104)	(0.094)	(0.082)	(0.101)	(0.100)	(0.083)	(0.100)
Age in Months	2.234	12.273	–21.101	5.897	–1.125	8.159	3.968	7.776	–9.960
	(10.920)	(8.986)	(13.049)	(12.364)	(8.171)	(12.884)	(12.405)	(9.067)	(12.127)
Age in Months, Squared	–0.002	–0.011	0.019	–0.005	0.001	–0.008	–0.004	–0.007	0.009

Table 3 (continued)

	Verbal Cognitive Ability			Performance Cognitive Ability			Overall Cognitive Ability		
Mother's Education:	Less Than High School	High School Graduate	College	Less Than High School	High School Graduate	College	Less Than High School	High School Graduate	College
	(0.010)	(0.008)	(0.012)	(0.011)	(0.007)	(0.012)	(0.011)	(0.008)	(0.011)
Constant	–628.7	–3,345.0	5,821.1	–1,622.0	344.0	–2,212.4	–1,101.4	–2,101.9	2,766.0
	(2,998.1)	(2,468.7)	(3,584.0)	(3,397.1)	(2,240.8)	(3,540.1)	(3,406.9)	(2,488.5)	(3,331.0)
Number of Observations	378	461	303	378	461	303	378	461	303

Note: Numbers in parentheses are standard errors.

*$p < .05$; **$p < .01$; ***$p < .001$

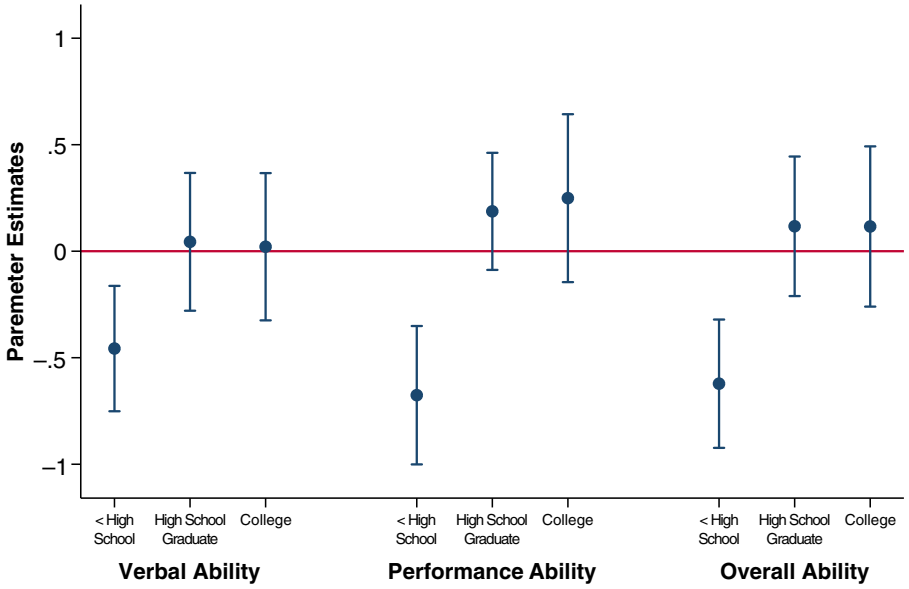

Fig. 2 Effect of prenatal exposure to earthquake in first trimester of gestation on children's cognitive ability at age 7 by family SES. Solid dots are parameter estimates; vertical bars are 95 % confidence intervals based on tests for the null hypothesis that the difference in parameter estimates across SEs is different from 0 at the .05 level (Knol et al. 2011). Chi-square tests for the difference in parameter estimates across family SES are as follows: (1) Performance $ability_{LTHS}$ versus Performance $ability_{HSGrad}$ = 7.53 (p = .006); (2) Performance $ability_{LTHS}$ versus Performance $ability_{College}$ = 5.55 (p = .019); (3) Overall $ability_{LTHS}$ versus Overall $ability_{HSGrad}$ = 4.75 (p = .029); (4) Overall $ability_{LTHS}$ versus Overall $ability_{College}$ = 4.11 (p = .043); (5) Verbal $ability_{LTHS}$ versus Verbal $ability_{HSGrad}$ = 2.30 (p = .130); (6) Verbal $ability_{LTHS}$ versus Verbal $ability_{College}$ = 2.20 (p = .138). *Source:* Table 3

literature, I propose three hypotheses: socioeconomic differences in exposure, sensitivity, and parental responses. The first hypothesis suggests that exposure to the stressor was not homogenous, but rather was more acute among disadvantaged families given the vulnerability of their residences and lack of resources. If that were the case, then what we have called heterogeneity of the effect would be an artifact of heterogeneity of exposure. To examine this possibility, I create an exposure scale. Using principal component analysis, I combine 18 indicators of earthquake impact based on survey questions, including whether the dwelling suffered any damage; electric power was lost; self/family member/neighbor was wounded or injured; and whether the household suffered a drop in income, had to temporally relocate to a shelter, or had to take in other people who were displaced. I use the first component as the measure of exposure. The Cronbach's alpha of the measure reaches .763, signaling acceptable reliability.

Table 4 displays the mean of the exposure scale across the levels of family socioeconomic advantage, showing a substantial difference between treatment and control areas as well as very little socioeconomic variation in the treatment area. Consistent with the high level of seismic preparedness in Chile and the limited spillover effects of this natural disaster, the differences in intensity of exposure by family SES are very minor and do not reach statistical significance. This finding is thus inconsistent with the first hypothesis suggesting that SES variation in the effect of prenatal stress results from stratified exposure.

Table 4 Mean exposure and sensitivity to stressor scales by family socioeconomic status (measured by mother's education) in treated and control areas[a]

	Treated Area		Control Area	
	Mean	SD	Mean	SD
Exposure Scale				
Mother's education				
Less than high school diploma	0.762	(1.013)	–0.684	(0.396)
High school graduate	0.757	(0.865)	–0.665	(0.414)
Some college or more	0.568	(0.949)	–0.699	(0.375)
Sensitivity Scale				
Mother's education				
Less than high school diploma	0.897	(0.612)	–0.856	(0.418)
High school graduate	0.848	(0.579)	–0.850	(0.450)
Some college or more	0.645	(0.696)	–0.915	(0.323)

[a] Exposure and sensitivity scales based on principal component analysis of several indicators, with the first component extracted. Scales have a mean of 0 and variance of 1 by construction.

The second hypothesis indicates that the poor may be more sensitive to an acute environmental stressor because the chronic stress associated with poverty heightens their sensitivity to the novel stressor and exhausts their coping resources. To address this hypothesis, I create a stress sensitivity scale using 10 indicators based on such survey items as, "Everything brought back memories of the earthquake," "I had difficulty sleeping as a result of the earthquake," "I felt irritable and edgy," and "I felt the earth was shaking permanently." As with the exposure scale, the indicators are combined using a principal component analysis, and the first component is extracted as a measure of sensitivity (Cronbach's alpha = .899, indicating good reliability). The rationale for using this scale is that individuals who carry a greater bodily burden because of disadvantage will display stronger sensitivity to a novel stressor. As in the case of earthquake exposure, vast differences between treatment and control areas exist in the sensitivity scale, but socioeconomic differences in sensitivity to the stressor in the treatment area are slight (Table 4). Therefore, the observed pattern of effects makes it unlikely that either heightened exposure or sensitivity accounts for the stronger effect among the poor.

To test the role that intensity of exposure and sensitivity to exposure play as pathways for the effect of prenatal stressor exposure on children's cognition, I conduct a mediation analysis, with the caveat that this analysis cannot be given a causal interpretation because the putative mediators are not randomly allocated, violating the sequential ignorability assumption (Imai et al. 2011). Table 5 shows the result of the mediation analysis by level of family SES. The first set of parameter estimates for each level of family SES does not include any mediators and simply replicates the results of Table 3 for overall cognitive ability. Model 2 controls for the intensity of exposure using the exposure scale, and Model 3 adds a control for sensitivity to Model 2 using the sensitivity scale. As is clear from Table 5, the parameter estimates capturing the effect of prenatal stress on children's cognitive development across family SES remain

virtually unchanged after controlling for these factors. This indicates that none of these factors plays a significant role the stratification of the effect of prenatal stress exposure.[5]

Having established that the stratified effect of stress exposure is not due to socioeconomic differences in exposure or sensitivity, I offer an alternative hypothesis. Sociological and economic research has suggested that parental responses may play a role in the unequal effect of prenatal exposures if parents compensate for or reinforce what they perceive as children's early handicaps or adversity, and if such responses are stratified by SES (Almond and Mazumder 2013; Bernardi 2014; Conley 2004; Hsin 2012). Because my quantitative data do not allow testing the role of parental responses, I conduct a qualitative investigation to assess the plausibility of this potential mechanism. Given the objective of capturing socioeconomic differences, I select samples of advantaged and disadvantaged mothers who were exposed to the environmental stressor during the first trimester of gestation. A trained interviewer and I conducted 38 interviews (18 with mothers/primary caregivers with less than a high school diploma, and 20 with mothers with a high school diploma or more) in the respondents' residence or place of employment between January and November 2015, when the children were, on average, 9 years old and attending fourth grade.

Findings from the qualitative interviews are reported in the online appendix (section A3). They show that advantaged parents continuously assess their children's strengths and weaknesses, mobilizing resources to mitigate the effect of what they perceive as their children's weaknesses and limitations (regardless of their attribution of the cause of these weaknesses), including time, money, assistance from professionals and experts, enrollment in organized activities, and close interaction with teachers and schools. Although some disadvantaged families have also resorted to the assistance of experts and educators and have requested institutional support, they face substantial barriers in terms of time, economic resources, and—equally important—access to social networks and mastery of cultural resources to effectively negotiate with institutions for advantages for their children.

These qualitative findings are consistent with prior literature (e.g., Lareau 2011). Their value is not their novelty, but the fact that they offer the hypothesis that class-based parental responses provide a mechanism for the stratified effect of prenatal stress exposure on children's later development and achievement. Although this hypothesis cannot be formally tested with the data at hand, its plausibility based on prior research and the qualitative evidence invites further investigation of this potentially critical channel of stratification during the early life course.

Conclusion and Discussion

Recent scholarship has shown that the prenatal period is both consequential for later attainment and well-being and vulnerable to the environment, and suggests that in terms of individual development and attainment, "the womb may be more important than the home" (Barker 1990:1111). Prompted by these new findings, this study

[5] I also examined birth weight as a potential mediator of the relationship between stress exposure and children's cognitive outcomes, finding that it plays no mediating role (online appendix, Table A2.2).

Table 5 Difference-in-differences model of the effect of prenatal earthquake exposure on children's overall cognitive ability by family socioeconomic status: No potential mediators (Model 1), controlling for earthquake exposure (Model 2), and exposure and sensitivity (Model 3)

	Model 1: No Controls			Model 2: Model 1 + Control for Earthquake Exposure			Model 3: Model 2 + Control for Earthquake Sensitivity		
Mother's Education:	Less Than High School	High School Graduate	College	Less Than High School	High School Graduate	College	Less Than High School	High School Graduate	College
Trimester 1	–0.147	0.499*	0.308	–0.013	0.564**	0.289	–0.009	0.551**	0.324
	(0.349)	(0.258)	(0.343)	(0.358)	(0.269)	(0.341)	(0.353)	(0.269)	(0.334)
Trimester 2	–0.057	0.107	0.268	0.023	0.152	0.229	0.028	0.143	0.252
	(0.262)	(0.192)	(0.191)	(0.280)	(0.201)	(0.195)	(0.279)	(0.201)	(0.188)
Trimester 3 (ref.)									
Treated Area	0.123	–0.418**	–0.558***	0.231	–0.338	–0.479**	0.330	–0.257	–0.310
	(0.195)	(0.174)	(0.206)	(0.283)	(0.208)	(0.223)	(0.377)	(0.213)	(0.232)
Trimester 1 × Treated	–0.622***	0.117	0.116	–0.557**	0.062	0.170	–0.554**	0.081	0.207
	(0.217)	(0.236)	(0.271)	(0.211)	(0.236)	(0.278)	(0.218)	(0.238)	(0.286)
Trimester 2 × Treated	–0.389	0.253	0.260	–0.449	0.330	0.324	–0.463*	0.309	0.315
	(0.275)	(0.213)	(0.258)	(0.274)	(0.232)	(0.268)	(0.275)	(0.230)	(0.269)
Trimester 3 × Treated (ref.)									
Mother's Age	0.000	–0.006	0.001	0.002	–0.008	0.003	0.003	–0.008	0.003
	(0.008)	(0.009)	(0.008)	(0.009)	(0.010)	(0.009)	(0.009)	(0.010)	(0.009)
Mother Is Married	–0.000	0.177	–0.135	–0.030	0.173	–0.138	–0.034	0.175	–0.144
	(0.118)	(0.117)	(0.098)	(0.120)	(0.129)	(0.101)	(0.113)	(0.128)	(0.102)
Male	0.190*	0.170**	0.076	0.165*	0.182**	0.104	0.168*	0.185**	0.091
	(0.100)	(0.083)	(0.100)	(0.097)	(0.089)	(0.096)	(0.097)	(0.091)	(0.095)
Age in Months	3.968	7.776	–9.960	0.013	7.392	–7.883	0.116	7.576	–6.645
	(12.405)	(9.067)	(12.127)	(14.061)	(10.222)	(11.827)	(13.961)	(10.234)	(11.693)

Table 5 (continued)

	Model 1: No Controls			Model 2: Model 1 + Control for Earthquake Exposure			Model 3: Model 2 + Control for Earthquake Sensitivity		
Mother's Education:	Less Than High School	High School Graduate	College	Less Than High School	High School Graduate	College	Less Than High School	High School Graduate	College
Age in Months, Squared	–0.004	–0.007	0.009	0.000	–0.007	0.007	–0.000	–0.007	0.006
	(0.011)	(0.008)	(0.011)	(0.013)	(0.009)	(0.011)	(0.013)	(0.009)	(0.011)
Exposure				–0.074	–0.050	–0.089	–0.052	–0.003	0.013
				(0.081)	(0.071)	(0.067)	(0.068)	(0.091)	(0.077)
Sensitivity							–0.049	–0.065	–0.136*
							(0.108)	(0.069)	(0.080)
Constant	–1,101.4	–2,101.9	2,766.0	–8.467	–1,993.2	2,194.5	–36.604	–2,043.9	1,855.7
	(3,406.9)	(2,488.5)	(3,331.1)	(3,863.3)	(2,805.4)	(3,249.5)	(3,835.9)	(2,808.8)	(3,211.8)

*$p < .05$; **$p < .01$; ***$p < .001$

examines the effect of prenatal exposure to an acute environmental stressor on children's cognitive ability as well as the stratification of that effect. To address the challenge of unobserved selectivity among women more likely to be exposed to stress, I combine a natural experiment—a strong earthquake—with a panel survey of children prenatally exposed to the earthquake and observationally similar but unexposed children constituting a control group. Findings show that prenatal exposure to an acute environmental stressor has no effect on children's subsequent cognitive ability among advantaged families, but it has a negative effect among poor families.

This finding speaks to two alternative approaches to the effect of early-life shocks on individual development and well-being. The first approach indicates that an early shock may have lasting cumulative consequences over the life cycle, such that "a child who falls behind may never catch up" (Heckman 2006:1900). An alternative approach suggests that the effect of an initial shock will fade and disappear over time (Grossman 1972). This study indicates that both hypotheses are correct, with their plausibility dependent on family SES. Among poor families, children prenatally exposed to an environmental stressor had much lower levels of cognitive ability than comparable control children. In contrast, no effect was observed among socioeconomically advantaged families. Although it is not necessarily true that a child who falls behind never catches up, the ability to catch up depends on the family's socioeconomic resources.

This finding raises a subsequent question about the factors accounting for the stratification of the prenatal exposure effect. I examine several mechanisms potentially accounting for stratification and find that the effect's stratification does not emerge from stronger exposure or from heightened sensitivity among poor families. Having ruled out these hypotheses, I offer the hypothesis that stratification emerges from socioeconomic differences in parental responses. Advantaged parents might mobilize diverse resources to compensate for their children's early disadvantage, including parental time, professional experts (such as developmental psychologists and neurologists), and private tutors. In contrast, poor parents face severe constraints in terms of time, material, cultural, and social resources, which make compensation difficult. Although I cannot test this hypothesis at this point, this is a verifiable premise. Specifically, I invite future research to test whether class-based parental resources and styles interact with early exposure to adversity, shaping life chances in early childhood.

This analysis highlights a potentially powerful mechanism for the transmission of disadvantage across generations: poor children are more likely to be exposed to environmental stressors, and such exposure has a stronger effect on their cognitive development compared with their advantaged peers. The combination of these factors may result in cognitive gaps between poor and advantaged children very early in life. Because prenatal exposures are hard to observe, it would be easy to interpret these socioeconomic gaps as emerging from genetic or other innate attributes rather than from structural factors rooted in different access to resources. The findings reported here are especially important because stressors such as natural disasters are only one of the many forms of toxic exposures faced disproportionately by the poor. It is now well documented that disadvantaged populations are more exposed to multiple environmental risks, including violence (Harrell et al. 2014), lead toxicity (Tong et al. 2000), and pollution (Bell and Ebisu 2012), all of which may induce largely invisible but cumulative damage very early in life. Whereas the literature has focused on the noxious

effect of chronic stress resulting from persistent, continuous, or repeated exposure to stressors, this analysis highlights the potentially toxic effect of acute stress. The findings show empirically that exposure to acute stress, even if short-term, can have persistent consequences when experienced before birth.

Even if the combination of a quasi-experiment with a DID approach provides a safeguard against confounding, this study has a number of limitations, including the retrospective nature of the survey information, the lack of biomarkers or other direct measures of the stress response immediately after exposure to the earthquake, and the lack of additional control groups including (for example) children born before the earthquake. This study also raises additional questions. The focus is explicitly on an acute stressor rather than a chronic one. This decision was guided by the hypothesis that discrete exposures may have long-term effects when experienced during critical developmental periods. This raises the important question about interactions between acute and chronic sources of stress. Socioeconomic disadvantage, experiences of discrimination, work-related demands, or persistent family conflict are chronic stressors that can modify the effects of exposure to a novel acute stressor, either exacerbating or reducing its impact. This study offers an initial foray into this question by examining the stratification of the effect of a prenatal acute stressor by family disadvantage, representing a source of chronic stress. However, there are multiple factors that can potentially shape responses to acute stressors and their consequences over the life course.

A further question refers to the relevant unit of analysis for the effect observed. I estimate a reduced-form effect among all women exposed to a natural disaster. This effect is likely shaped by social networks of interaction and influence connecting individuals. For example, interactions between friends or neighbors in the wake of the disaster could exacerbate fear and anxiety, magnifying the effect of stressor exposure. Alternatively, they could be the source of social support and relief, ameliorating the consequences of exposure. In methodological terms, the inability to isolate the individual-level from the community-level components of the effects is a limitation described as the violation of the stable unit treatment value assumption (SUTVA), and this assumption is required for causal inference. SUTVA requires that there should be no interference between subjects; that is, the potential outcomes of any exposed individual should not depend on other individuals being exposed to the stressor or experiencing stress (Rubin 1980, 1986). However, each individual's potential outcome due to experiencing the acute stressor is likely affected by other members of their social networks being exposed to or experiencing stress. This assumption is, therefore, virtually guaranteed to be violated in this case and in all social settings in which individuals are connected to each other through networks of interaction and influence.

From one perspective, the impossibility of separating individual- from collective-level mechanisms for the effect is irrelevant. I am not interested in the effect that an acute stressor would have on individuals in the fictitious isolation that an experimental laboratory could provide. However, this feature raises the question about the generalizability of the results. The treatment effect captured in any particular social setting might differ from that in other settings depending on the type of social networks prevalent in each case. This invites more research on the role that social networks and other macro-level processes play in accounting for the consequences of stress exposure at the population level. Researchers have traditionally considered SUTVA a

nuisance: the notion of interference explicitly suggests something that needs to be eradicated to capture a true causal effect. However, interference through social networks is an important substantive phenomenon in its own right, and social scientists are particularly qualified to examine the scope and dynamics of social influence contributing to observed causal effects.

The final, and perhaps the most important, question this study raises returns to a life course perspective. In an attempt to provide a dynamic account of the influence of prenatal exposures, this analysis examines children's cognitive outcomes at age 7, still early in their life course. Although this expands a literature that is largely focused on effects of prenatal exposures either at the very beginning of life (that is, birth outcomes) or very late in life (that is, later-life health and mortality), this is just a starting point. Given that cognitive ability in childhood predicts schooling, health, and economic well-being later in life, the findings offered here call for additional research following up individual trajectories into adulthood, as the children affected engage institutions and settings beyond their families of origin, such as schools and neighborhoods. Such research is essential to further illuminate the dynamics underlying persistence and change of early disadvantage over the life course.

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