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The Intergenerational Impact of Terror: Did the 9/11 Tragedy Impact the Initial Human Capital of the Next Generation?

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Abstract

Given the unexpected nature of the terrorist attacks of September 11, 2001, a specific cohort of children were exogenously exposed to increased maternal psychological stress *in utero*. Rich administrative data and the precise timing of the event allow this study to uniquely provide insights into the health effects of exposure to maternal psychological stress across gestation. Results suggest that children exposed *in utero* were born significantly smaller and earlier than previous cohorts. The timing of the effect provides evidence that intrauterine growth is specifically restricted by first trimester exposure to stress; reductions in gestational age and increases in the likelihood of being born at low (<2,500 grams) or very low (<1,500 grams) birth weight are induced by increased maternal psychological stress mid-pregnancy. This study also documents a positively selected post-attack fertility response, which would bias an evaluation that includes cohorts conceived after September 11, 2001, in the control group.

Keywords Birth weight · Maternal health · Fetal health · Psychological distress

Introduction

The September 11, 2001 (hereafter, 9/11) tragedies in New York City; Arlington, VA; and Shanksville, PA, extinguished nearly 3,000 lives. The unanticipated nature of the attacks, along with the devastating imagery of the event, produced high levels of psychological stress throughout the nation (Knudsen et al. 2005; Schuster et al. 2001). This event generated elevated levels of stress for several weeks after the attacks

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and weighed particularly heavily on women (Silver et al. 2002; Stein et al. 2004). In addition, as hypothesized in Becker and Rubinstein's (2011) theory of responses to terrorism, the fear generated by the event was not limited to those in assaulted areas. In a nationally representative survey, Schuster et al. (2001) found that more than 40% of adults reported stress-related symptoms after the 9/11 attacks. One particularly troubling aspect of this widespread "terror" shock is that it may cause the impact of the 9/11 event to spread into the next generation.

Using theoretical models, animal experiments, and small-sample human research, the current literature has biologically mechanized and repeatedly correlated maternal stress with, among other birth outcomes, restricted intrauterine growth and shortened gestational length (Aizer et al. 2016; de Catanzaro and Macniven 1992; Mulder et al. 2002; Wadhwa et al. 1993, 2001, 2004). Further, recent and consistent findings have connected birth outcomes to later-life human capital accumulation (Behrman and Rosenzweig 2004; Black et al. 2007; Case et al. 2005). These two lines of research have motivated social scientists to reassess the full negative effect on society of psychologically distressing events, such as discrimination, violence, natural disasters, death of a family member, and even unexpected results in major sporting events by evaluating their impact on the birth outcomes of the exposed pregnant women (Black et al. 2016; Brown 2018; Camacho 2008; Currie and Rossin-Slater 2013; Duncan et al. 2017; El-Sayed et al. 2008, Lauderdale 2006; Mansour and Rees 2012; Novak et al. 2017; Quintana-Domeque and Rodenas-Serrano 2017; Torche 2011). This study adds to this emerging literature by using the 9/11 tragedy as an exogenous stress shock to estimate the response in birth outcomes from the psychological fallout caused by terrorism.

Although this study is able to make use of an event that was completely unanticipated and that for most of the population increased psychological stress as the primary negative externality, the adverse effects for individuals living in areas directly affected by the attack—New York City (NYC) and the Washington, DC primary metropolitan statistical area (DC)—are not limited to psychological stress. Specifically, residents of NYC and DC also faced a pollution-related adverse health shock and/or a negative resource shock due to loss of economic activity (Bram et al. 2002; Currie and Schwandt 2016; Landrigan et al. 2004). Individuals from these areas are thus excluded from this study's analytical sample because these regions are vulnerable to misallocation of stress as the central contributor to poor birth outcomes.

In addition, an issue that must be carefully considered in any study of *in utero* health is selective fertility. When using the 9/11 attacks as the quasi-random shock of interest, it is reasonable to assume that its unexpected nature makes selective fertility related to the terrorist event unlikely. Cohorts conceived after 9/11, though, are not shielded from potential nonrandom selection. For example, it is quite plausible that family planning decisions made after the catastrophe could be endogenously related to parental characteristics correlated with birth outcomes. Unique to the literature on environmental stressors and birth outcomes, this study directly examines selective fertility after the focal event, finding that cohorts conceived after the attacks have mothers who were significantly more educated and less likely to be African American. This finding indicates that in this case, the post-event cohorts are nonrandomly selected based on characteristics correlated with higher birth weight, and thus their inclusion would severely hinder identification. This study attempts to mitigate concerns over

endogenous fertility by testing for differences in the maternal characteristics of the exposed and unexposed cohorts and excluding all cohorts conceived after 9/11.

I use the Vital Statistics Natality Birth Data, which include all U.S. live births that received a birth certificate, to carry out this analysis. The power available from the data set's large sample sizes and its detailed pregnancy information provides this analysis with the opportunity to make two additional contributions. First, aside from the recent exception of Bruckner et al. (2019), all the studies that have examined the impact of crime/conflict/terrorism on birth outcomes have solely used birth month to determine *in utero* exposure timing. The concern with this approach is that if, as predicted by the medical literature, gestational age is affected by exposure to maternal psychological stress, then the exposure timing assignment will be endogenously related to the level of psychological stress experienced by that mother. This study uses gestational age information in order to break this link between exposure intensity and assignment of exposure timing. Second, despite a growing set of studies connecting stressful events to worsened birth outcomes, information about the importance and differential impact of exposure timing during gestation is still limited. In this study, because of the exact timing and short-lived nature of the exposure paired with information about gestational age, the monthly temporal path of *in utero* exposure to the event on the distinct outcomes of intrauterine growth and gestational age can be examined.

The results of this analysis indicate that infants *in utero* during the 9/11 attacks are significantly smaller (on average, up to 15 grams smaller) and much more likely to be defined as at high risk for future health problems (up to a 14% increased risk of being born weighing less than 1,500 grams and a 9% increased risk of being born at <37 gestational weeks). Further, intrauterine growth is most sensitive to exposure in the first trimester, but gestational age is most reactive to exposure mid-pregnancy. A rich set of health behavior responses are also investigated to rule out these reactions as the mechanism behind the relationship between maternal exposure to 9/11 and fetal health.

Literature Review

Stress and Birth Outcomes: Biological Mechanisms

The biological evidence regarding a link between a pregnant mother's experience of a stressful event and their subsequent pregnancy outcome is suggestive but limited. Although physiological-level responses to a stressful event vary across individuals, all humans use certain biological feedbacks to regulate psychological stress. In particular, the body generates cortisol, norepinephrine, and epinephrine in elevated levels in reaction to acute stress as well as "worry, anxiety, and cognitive preparation for a threat" (McEwen 1998:175). These chemicals then stimulate the supply of corticotropin-releasing hormone (CRH). Linking maternal stress to birth outcomes, various studies have indicated that the level of CRH is strongly related to intrauterine growth and parturition timing (e.g., Mancuso et al. 2004; Wadhwa et al. 1993, 2004). Additionally, Mulder et al. (2002) suggested that arousal of the sympathetic nervous system—a symptom of increased stress—can cause restricted blood flow to the fetus and thus result in decreased intrauterine growth. Moreover, relationships may vary with the child's gender (Torche and Kleinhaus 2012) and the mother's socioeconomic status

(Brown 2018). Infection sensitivity is also increased by stress exposure, and reaction to this inflammation could trigger earlier labor (Wadhwa et al. 2001).

Multiple medical studies have shown that the release of the hormones associated with a reaction to stress is attenuated during pregnancy, and this chemical insulation increases throughout pregnancy (de Weerth and Buitelaar 2005; Schulte et al. 1990). Although this suggests that the adverse effects of maternal psychological stress on birth outcomes should be most prevalent in early gestation, not all studies have supported this claim, and some have even come to the opposite conclusion (Hedegaard et al. 1993; Schneider et al. 1999). As it stands, the medical literature advocates that the timing of *in utero* stress exposure is important to the biological path of birth outcome damage, but the specific pattern is still without strong empirical support.

September 11, 2001, and Birth Outcomes: Prior Evidence

In the years following 9/11, many researchers have expressed concern over the possible negative effects it may have had on infants exposed *in utero* to these events. Such studies have focused on three areas: environmental fallout, discrimination, and stress.

Studies have suggested that the destruction of the World Trade Center (WTC) was the most severe environmental catastrophe in the history of NYC (Landrigan 2001). After the events of 9/11, a gigantic plume containing a mixture of numerous hazardous materials hovered over and traveled across NYC (Landrigan et al. 2004). Although analysis of the impact of this pollution exposure on birth outcomes had previously led to inconsistent and/or imprecise conclusions, Currie and Schwandt (2016) showed that controlling for unobserved differences in the mother's exposed to the 9/11 dust cloud and their neighbors in lower Manhattan is vital to proper identification. After this nonrandom heterogeneity is removed through the use of within-mother comparisons, Currie and Schwandt showed that mothers living in the pollution-affected parts of NYC were significantly more likely to give birth earlier and to smaller babies. These findings indicate that focusing attention on births outside NYC may be a more accurate way to assess avenues in which the attack affected the exposed gestations beyond direct health shocks from pollution. One interesting line of research to that end has looked at how differential treatment and psychological stress of Arab-named women may have led to poorer birth outcomes.

Diane Lauderdale (2006) and El-Sayed et al. (2008) hypothesized that post 9/11, Arabic-named women would suffer from significant increases in discrimination and that this would negatively affect their birth outcomes. Although these natural experiment studies had very similar data resources and methodologies, the results were quite different. In California, Lauderdale found that children born to Arabic-named women who were pregnant during 9/11 had a significantly higher likelihood of being low birth weight (LBW, <2,500 grams) and preterm (PTB, <37 weeks of gestation) than comparison children from the previous year—a finding that did not hold for any other ethnicities. On the other hand, El-Sayed et al. (2008) found in Michigan that women with Arab American ethnicity who were pregnant during 9/11 were *less* likely to give birth to a LBW or PTB child.

It is difficult to reconcile these conflicting findings other than to speculate that each state may have varying levels of discrimination, distinct patterns of geographic/social ethnic clustering, and/or different magnitudes and selectivity of in-/out-migration, none of which is captured by either analysis. Furthermore, although these studies asked a very intriguing question, they were not able to nail down the mechanism through which discrimination affected birth outcomes. Increased stress is one channel, but another major pathway could be financial. For instance, Kaushal et al. (2007) found that wages for Arab Americans declined after the 9/11 attacks. Further, family incomes could be negatively impacted through reduced transactions with Arab American businesses. Thus, although these studies represent an innovative approach, the discrimination studies have not formed a consensus and are not aimed at identifying the effects of psychological stress specifically.

A host of studies in the medical literature have attempted to make a more clear statement about the effect of 9/11–induced maternal stress on birth outcomes. Several studies used small selected samples of New Yorkers who lived close to the WTC (Berkowitz et al. 2003; Eccleston 2011; Engel et al. 2005; Lederman et al. 2004; Perera et al. 2005). These analyses supported a connection between maternal stress and poor birth outcomes, but geographic proximity to the attack confounds the identification strategy with pollution effects. In addition, by using residents from any part of NYC, the analysis faces the prospect of the exposed cohorts experiencing not just aggravated maternal stress but also a negative resource shock. Multiple studies have shown that NYC employees lost a significant number of labor hours and wages over the next few months following the attacks (Bram et al. 2002; Dolfman and Wasser 2004). Intuitively, loss of income for expecting families can lead to reduced health inputs, causing poorer birth outcomes and thus creating an overstatement of the effect of maternal stress. Moreover, in addition to the income shock faced by the NYC treatment group, this cohort may also be contaminated by selective migration. Following a major health-threatening event, there may be migration out of the affected area by pregnant women trying to insulate themselves from further stressors or other health insults.[1]

The work most in line with the approach found in this paper was conducted by Eskenazi et al. (2007). They used birth certificate data for upstate New York residents in the 40 weeks after the event and compared them with those born during the same period in the preceding two years to shield their analysis from some of the concerns raised previously. The results from this study indicated that very low birth weight births (VLBW, <1,500 grams) increased in upstate New York around the New Year (second trimester exposure) and eight months after 9/11 (first trimester exposure), but moderately low birth weight births (1,500 to <1,999 grams) *decreased* for those born in early December. Results for PTB were also mixed: the authors found that late-December births were more likely to

[1] Eccleston (2011) provided evidence of this phenomenon for the specific case of NYC residents after 9/11. In Eccleston's study, she showed that NYC and NY state income tax filings indicate that from 2001 to 2002, NYC experienced more, and higher income, emigration than the rest of NY state. In addition, she showed that the composition of exposed births in NYC had significantly more non-White mothers than previous cohorts.

be moderate PTB (32 to <37 weeks), but those exposed late in pregnancy living in upstate New York were significantly *less* likely to have a moderate PTB. Eskenazi et al. (2007) represented an important improvement over examining births to residents of lower Manhattan. The analysis presented here builds on Eskenazi et al. (2007) but differs most notably in the following ways: (1) the sample is national, (2) births conceived after 9/11 are explored for evidence of selective fertility and excluded, (3) conception date is used rather than birth date to assign exposure, (4) birth weight is assessed based on its measurement in grams rather than by 500-gram intervals, and (5) the mothers' health behaviors are examined as potential mechanisms.

To avoid the difficulty of identifying maternal stress's relation to birth outcomes using residents from cities that were attacked, a few studies have looked elsewhere for confirmation of the link but have provided contradictory conclusions. Smits et al. (2006) looked at more than 3,000 Dutch infants *in utero* during and one year after 9/11, finding that those exposed while in their second and third trimester had significantly smaller birth weight. Alternatively, a study by Endara et al. (2009), using a large data set of infants born to active-duty military families, found no *in utero* effect during the attacks. Both studies, though, relied on the use of the post–9/11 conception cohort as the control group and thus lost part of their identification given that fertility rates and parental characteristics have been found to change after catastrophic events (Evans et al. 2010). Further, Rich-Edwards et al. (2005), studying 1,184 Boston-area women, estimated that those pregnant during 9/11 were *less* likely to have a PTB, but a failure to control for time trends may have driven this counterintuitive result. The study presented here pairs the advantageous strategy used by these analyses of examining birth outcomes of mothers from locations not explicitly attacked during the events of 9/11 with a careful examination and accounting of selective fertility and the inclusion of rich temporal controls.

A final important distinction between this study and prior analyses of this topic is the use of conception date rather than birth date to assign exposure. This choice provides several advantages. First, when relying solely on date of birth, the analysis may include individuals who were born within nine months of the event but were conceived after the event took place. To the extent that these post-event conceived births are potentially nonrandom, which has been shown to be the case in other settings (Evans et al. 2010) and will be shown in the subsequent analysis to be relevant to this context, they would lead to biased estimates. By using conception date information, we can restrict the sample to only those births conceived prior to the focal event. Second, even if there is no evidence of selective fertility, those births that occurred within nine months of the event but were conceived after the event will be misassigned to the treatment group and lead to an underestimate of the effect. This issue will be most pronounced for those thought to be exposed early in gestation and is alleviated when using conception date to assign exposure. Third, using date of birth makes it more difficult to assess how exposure in different parts of the *in utero* period may have varying degrees of impact on the birth outcomes. Specifically, if length of gestation is impacted by maternal psychological

distress, then the calculated timing of exposure using birth date will be endogenous and incorrect.[2] Using date of conception combined with the date of the focal event allows for a more precise description of the gestational age of the fetus when exposure to the shock occurs.[3]

Data and Methodology

The data used for this study are the 35,809,694 birth certificates for children born between January 1, 1995, and December 31, 2003, collected by the National Center for Health Statistics available in the Vital Statistics Natality Birth Data (VSNB). In addition to providing a large sample, the data contains several birth outcome variables as well as demographic and medical data on the mother and the birth.[4] For the reasons discussed previously, births to NYC and DC residents are excluded from the analytical sample.[5]

To determine a birth's prenatal exposure to the 9/11 attacks, I use the VSNB information on gestational age.[6] When gestational age (in weeks) is used along with

[2] For example, if exposure to an environmental stressor in the fourth month of pregnancy leads to an increase in births that occur at least one month early, using only date of birth to determine exposure timing will erroneously assign these poor birth outcome births to first trimester exposure and incorrectly suggest that first trimester exposure has a larger impact on birth outcomes.

[3] One caveat to the advantage of using gestational age to assign exposure compared with birth month is that individuals conceived nine months prior to the event may not all remain *in utero* to experience exposure to the shock. Because the mechanism for the early timing of these births would be unrelated to the event, given that it has not occurred yet, the composition of these births will be mirrored by similar births in the control group and thus does not present an issue of endogenous selection. These births do, though, lead to measurement error for the cohort considered exposed in the last months of gestation because they are assigned to a treated group, but in truth are not exposed to the focal event. If this issue is present it would lead to estimates for the earliest conception month cohorts in the treatment group to be biased toward no effect. To assess the extent of this bias, all the main analyses in this article are also provided using the alternative method of assigning exposure based on birth month. These results, presented in the tables of section B in the online appendix, provide no evidence that the use of gestational age to assign exposure is leading to substantial underestimation or incorrect inference regarding the impact of late gestation exposure to maternal psychological distress on birth outcomes.

[4] Researchers have argued that some elements of the birth certificate data, especially parental characteristics and gestational age, are incomplete and imprecise (Reichman and Hade 2001). In terms of measurement error resulting from imprecise gestational age information, because there is no reason to think the inaccuracy would have a specific pattern or relationship to the timing of 9/11, the only concern would be less precision in the estimated coefficients. The power gains from the large sample size do to an extent, though, help to offset this concern. With regard to missing information, the primary dependent variable—birth weight—is missing for only .1% of the sample, and there is no evidence that lack of birth weight information is related to 9/11 exposure. In addition, only 2.8% of the sample is missing any information used in the primary specification. In the main analysis, when a control variable has missing information, it is assigned the mean value from the sample and for each variable an indicator that identifies observations with missing information is added to the regression. Results are comparable when alternatively any individual with a missing value for an independent variable is dropped.

[5] Robustness checks that additionally exclude individuals from the entire New York City metropolitan area are also conducted and included in section A of the online appendix.

[6] One potential concern is that because gestational age is predominately calculated based on women's self-reports, if error in this measure is systematically related to 9/11 it could bias the results. The most plausible way this type of nonrandom misreporting could occur is if pregnant women were less likely to obtain or delayed prenatal care following the terrorist attack, given that knowledge and accuracy of gestational age is partly based on health care usage. As discussed in the section Parental Composition and Maternal Behaviors, this is not the case.

birth month information, a rough approximation for conception week can be estimated.[7] In this study, *conception week* is calculated in the following way. First, to get an estimate of the number of weeks before birth that conception occurred, I take the gestational age (measured in weeks since the last menstrual period) and subtract 2 (conception usually occurs two weeks after the last normal menstrual period). Second, I divide that estimate by four to convert weeks since conception into months. Third, I subtract this estimate of months since conception from the birth month. Last, if the resulting estimated conception week is less than 1, I increase it by 12.[8] Conception year is then calculated as either the birth year or the birth year less 1 if the conception month is larger than the birth month.[9] Because weekly data must be subtracted from monthly data to generate conception week, each estimated conception week covers a range of potential conception weeks. For example, if an infant is born in the first week of a month, the conception week generated in the data is correct. If an infant is born in the last week of a month, though, the conception week generated in the data is early by three weeks. As such, to make sure to exclude all births conceived after the event, I include only infants with a calculated conception date of August 14, 2001, or earlier.[10]

The model for this approach is estimated as follows:

$$b_{imjt} = \alpha_0 + \mathbf{Treat}_i^{'}\beta + \mathbf{X}_{im}^{'}\delta + \gamma_{yrproxy} + \gamma_{week} + \gamma_j + \gamma_{yrproxy,j} + \varepsilon_{imjt}. \quad (1)$$

In Eq. (1), b_{imjt} is the birth information of interest for individual i conceived at date t to mother m, who resides in state j. $\mathbf{Treat}_i^{'}$ is a matrix of eight indicators for each month of conception from January 1, 2001, to August 14, 2001, representing the exposure period. Additionally, the matrix $\mathbf{X}_{im}^{'}$ contains controls suggested by the medical literature, including mother characteristics (education, race, marital status, age, plurality, and an indicator for diabetes) and birth information (plurality and sex of infant).[11] Because the VSNB is a large data set, many of these variables can be controlled for nonparametrically, rather than linearly or quadratically, which is the general practice in the literature. Thus, indicator variables are used for mother's education (18 levels), mother's age (36 levels, including a level for less than 16 years of age, and a level for

[7] Birth month is the finest level of birth date information available for each child.

[8] For example, for a gestational age of 36 weeks and birth month of 12, the conception week would be calculated in the following way. The gestational age minus 2 and divided by 4 is 8.5, suggesting that conception occurred 8.5 months before birth. Subtracting 8.5 from the birth month, 12, suggests the estimated conception week was the third week of March. Alternatively, for a birth month of 3, subtracting 8.5 from the birth month would give −5.5. Because this value is less than 1, 12 would be added back to give 6.5, or the third week of the previous June.

[9] In the previous example in which gestational age was 36 weeks and the birth month was 12, conception year would equal birth year. Alternatively, in the example in which the birth month was instead 3, conception year would equal birth year minus 1.

[10] As I discuss in the upcoming main analysis section, cohorts conceived after the event are from endogenously and positively selected families, and thus their inclusion would jeopardize the randomness of the treatment/control designation.

[11] In section B of the online appendix, the results are checked for robustness to additionally including controls for county-level economic conditions.

50 and over), mother's race (five levels: White, African American, Hispanic, Asian, and other), and parity (eight levels, including a level for live birth order of eight and above).[12]

Because the method of identification is temporal, controlling for time trends nonparametrically is imperative to proper analysis of this event's impact on birth outcomes. This is made a bit more complicated by the fact that the coefficients of interest include month-by-month indicators for all conceptions in 2001, and thus a conception year fixed effect for 2001 would be perfectly collinear with these treatment variables. To include time fixed effects without damaging interpretation of the treatment point estimates, I place the data into six equal 16-month groups based on conception date. Thus, although true conception year fixed effects are not included, these six 16–month interval fixed effects, $\gamma_{yrproxy}$, will serve as controls for time trends.[13] In addition, it is critical in this type of study to control for seasonality in birth outcomes, and thus indicators for week of conception, γ_{week}, are included. Fixed effects for mother's state of residence, γ_j, are also used in the model to account for unobserved heterogeneity that is time-invariant within the mother's residence state. Finally, heterogeneity specific to the state-conception year level is controlled by including fixed effects for the 16-month conception interval–mother's state of residence pair, $\gamma_{yrproxy,j}$.[14]

All tables provided that use the individual-level VSNB data report robust standard errors, with results that are statistically significant using the Schwarz criteria shown in bold. The Schwarz criteria is a Bayesian approach to hypothesis testing and is included because it provides a stricter interpretation of statistical significance. In particular, it

[12] When a characteristic of the mother has a missing value, it is replaced with the mean value from the sample, and an indicator variable is created and included for each characteristic that equals 1 if the information for that factor is missing. Results do not qualitatively or quantitatively differ if all observations missing a value for any independent variable are instead dropped. Results available upon request.

[13] Given the nonstandard form that must be used for the cohort fixed effects, alternative controls for temporal heterogeneity have also been assessed. In Table A1 of the online appendix, the six 16-month interval fixed effects are replaced by linear splines using 6 periods, linear splines using 10 periods, quadratic trends, or 10-month fixed effects. In each case, the magnitudes of the coefficients are qualitatively and quantitatively equivalent, or larger, and the pattern is similar.

[14] Alternatively, an approach that estimates conception date as nine months prior to birth date—mirroring what is typically found in the literature when using only birth timing information—is provided in Table B1 of the online appendix. This approach uses all infants delivered before June 1, 2002, in an effort to limit the sample as much as possible, to children conceived prior to the event. Similarly, for births in September 2001, it cannot be determined whether they were exposed or not. Thus, as an attempt to err on the side of a nonresult, I considered them to be part of the control. Specifically, I estimate the following equation:

$$b_{imjt} = \alpha_0 + \mathbf{Treat}_i'\beta + \mathbf{X}_{im}'\delta + \gamma_{yrproxy} + \gamma_{month} + \gamma_j + \gamma_{yrproxy,j} + \varepsilon_{imjt},$$

In this equation, the matrix **Treat** is eight indicators of being born in one of the eight months from October 2001 to May 2002, representing the exposure period. Although true birth year fixed effects are not included, six 16-month interval fixed effects, $\gamma_{yrproxy}$, will serve as controls for time trends, and seasonality is controlled by birth fixed effects, γ_{month}. To account for unobserved heterogeneity that is time-invariant within the mother's residence state, I add dummy variables for mother's state of residence to the model, γ_j. Finally, the interaction of an observation's 16-month birth interval and mother's state of residence, $\gamma_{yrproxy,j}$, are incorporated into the specification.

requires the significance level to be inversely related to sample size: critical t is calculated as the square root of the natural log of n (Schwarz 1978).

To evaluate the impact of maternal psychological stress on early-life health, the birth outcomes tested include overall birth weight as well as indicators for LBW and VLBW births. Although these outcomes are the standard in the literature, they may obfuscate the pathway driving the poor birth outcome because birth weight can be caused by both restricted intrauterine growth and shortened gestation. To more finely focus the analysis on the biological process driving the birth outcome, I include two additional dependent variables. First, to strip the birth weight measure of the impact of gestational length in order to assess the impact solely on intrauterine growth, I create a birth weight–for–gestational age z score and use it as an outcome variable.[15] Second, to look at the other part of the birth outcome equation, I examine gestational age. In addition, because a medical literature suggests that maternal stress may impact the sex ratio by reducing male births (for a review, see Catalano et al. 2006), I evaluate an indicator for being a male infant.

After providing estimates for the overall impact of maternal exposure to 9/11 on the birth outcomes, Eq. (1) is further utilized to investigate any change in the composition of mothers during this period by substituting maternal characteristics such as race and education, as well as delivery characteristics, such as the likelihood of producing a live birth, as the dependent variable.[16] In addition, behavioral responses related to prenatal care, maternal weight gain, smoking, and alcohol use are also examined by using information on these actions as the dependent variable in Eq. (1). Last, to explore the heterogeneity of the overall effect, I stratify the sample by the child's gender, the mother's education, and the size of the city of mother's residence.

The other critical analysis I conduct is an analysis of potential selective fertility that may occur after the events of 9/11. To take a closer look at this issue, I compare maternal characteristics of non–NYC and DC infants conceived in the first seven months following the terrorist attack with the composition of maternal attributes in the rest of the sample period. The regression used in this analysis is as follows:

$$b_{imjt} = \alpha_0 + \beta \cdot POST + \gamma_{1994} + \ldots + \gamma_{2000} + \gamma_{week} + \gamma_j + \varepsilon_{imjt}, \qquad (2)$$

where $POST$ is an indicator for being conceived in the first seven months after 9/11, $\gamma_{1994} + \ldots + \gamma_{2000}$ are seven indicators for being conceived in the years from 1994 to 2000, and γ_{week} and γ_j are the same as in Eq. (1). For this test, b_{imjt} will be three maternal characteristics: an indicator for whether the mother is African American, an indicator of whether the mother attended any college, and a measure of the number of years of school the mother completed. As such, β is the coefficient of interest and will indicate whether the mothers of children conceived post-event are significantly different from mothers of children conceived in the first eight months of 2001.

[15] Birth weight–for–gestational age z score is calculated as an infant's birth weight minus the mean birth weight from 1995 to 2000 for that infant's gestational age, all divided by the standard deviation of birth weight from 1995 to 2000 for that infant's gestational age.

[16] Maternal characteristic controls are excluded from these regressions.

Results

Main Analysis

To provide context to the data, Table 1 displays the descriptive statistics for the variables used in the main analyses.[17] The 1995–2002 VSNB data contain 27,568,056 records of live births calculated to have been conceived before August 14, 2001. The mean birth weight, a characteristic available for 99.9% of the records, is 3,322.7 grams; 7.4% and 1.3% of the children were born LBW (<2,500 grams) or VLBW (<1,500 grams), respectively. Gestational age, another key indicator of the birth outcome available in the data, indicates that 11.3% of the children were born preterm (gestational age < 37 weeks).[18]

Moving to the primary analysis, each row of Table 2 represents a separate regression and provides the estimates of the β coefficients from Eq. (1). The estimates indicate that almost the entire cohort of children *in utero* during the attacks had significantly reduced birth weight, by as much as 15 grams. Furthermore, there is a large increase in the risk of having a child of LBW or VLBW. Children exposed mid-gestation to increased psychological stress were at a 4% to 5% and 6% to 14% increased risk of being born LBW and VLBW, respectively.[19]

The estimates in Table 2 also show that once gestational age is controlled for using the z score, intrauterine growth is significantly restricted by stress exposure only in early gestation. The timing of exposure also seems to matter for the relationship of maternal psychological stress and preterm birth: it is apparent that parturition timing is most sensitive to this exposure in the middle of pregnancy given that those cohorts were significantly more likely to be born preterm (with as much as a 9% increased risk factor). One counterintuitive result is the finding that those exposed in the first month of gestation were *less* likely to be born preterm. Results reported in the upcoming Parental Composition and Maternal Behaviors section provide some evidence that this finding may be driven by positive behavioral changes of the mothers in this cohort. These results also make it clear that a child's risk of being born LBW or VLBW is much more closely related to maternal stress's impact on gestational age than through intrauterine

[17] Equivalent summary statistics are provided by conception year in Tables C1–C7 in the online appendix.

[18] Table 1 indicates that at times, maternal information is missing in the birth records. In terms of control variables, the issue of missing information is minimal. Specifically, live birth order is missing for 0.5% of records, gestational diabetes status is missing for 1.2% of records, and mother's years of education is missing for 1.3% of records. When a characteristic of the mother has a missing value, it is replaced with the mean, and an indicator variable is created and included for each characteristic that equals 1 if the information for that factor is missing. Results do not qualitatively or quantitatively differ if all observations missing a value for any independent variable are instead dropped. Missing values for maternal pregnancy behaviors are more prevalent: 1.9% of records are missing prenatal care information, 20.1% of records are missing maternal weight gain information, 17.7% of records are missing gestational smoking behavior, and 14.8% of records are missing gestational alcohol use. With regard to the impact of potential measurement error or sample selection bias in these variables, because there is no reason to think the missingness or inaccuracy in these variables would have a specific pattern or relationship to the timing of 9/11, the only concern would be loss of external validity and less precision in the estimated coefficients for regressions that use those behaviors as the dependent variable.

[19] The risk factor estimates are calculated as the increased incidence divided by the mean incidence in the population.

Table 1 Summary statistics using natality data on births conceived before August 14, 2001, excluding residents of New York City and the Washington, DC, metropolitan area[a]

	Number of Observations	Mean	SD
Birth Outcomes			
Birth weight	27,552,002	3,322.7	602.7
Birth weight for gestational age z score	27,552,002	0.00	1.00
LBW (<2,500 grams) (%)	27,552,002	7.4	26.1
VLBW (<1,500 grams) (%)	27,552,002	1.3	11.5
Preterm (<37 weeks) (%)	27,568,056	11.3	31.7
Birth Characteristics			
Male (%)	27,568,056	51.2	50.0
Plural (%)	27,568,056	2.9	16.8
Live birth order	27,443,511	2.0	1.2
Maternal Characteristics			
Mother is non-Hispanic White (%)	27,568,056	61.0	48.8
Mother is African American (%)	27,568,056	14.2	34.9
Mother is Hispanic (%)	27,568,056	19.7	39.8
Mother had gestational diabetes (%)	27,229,795	2.7	16.3
Mother is married (%)	27,568,056	67.7	46.8
Mother's age	27,568,056	27.0	6.2
Mother's years of education	27,196,339	12.8	2.8
Mother's Pregnancy Behavior			
Late/no prenatal care[b] (%)	27,033,176	3.7	19.0
Maternal weight gain	22,013,433	30.8	13.4
Smoking while pregnant (%)	22,691,092	13.3	34.0
Alcohol use while pregnant (%)	23,483,669	1.1	10.7

Source: Data obtained from National Center of Health Statistics 1995–2002 birth certificates.

[a] The *Washington, DC, metropolitan area* is defined as the Washington, DC-MD-VA-WV primary metropolitan statistical area (PMSA).

[b] *Late/no prenatal care* is defined as either starting prenatal care in the third trimester or never receiving prenatal care.

growth restriction. The sex ratio appears to be unaffected by acute maternal psychological stress.

The analyses in Table 2 use data restricted by two nontrivial sample selection choices. First, the decision to exclude NYC and DC residents from the analysis was influenced by previous studies indicating that along with being exposed to the stress of 9/11, these individuals also have a higher likelihood of having been exposed to a pollution and/or resource shock, either of which would be negatively related to birth outcomes and thus confound the estimation of the effect of psychological stress (Bram et al. 2002; Currie and Schwandt 2016; Landrigan et al. 2004; Perera et al. 2005).

Second, the decision to exclude individuals conceived after the events of 9/11 was made given the concern that family planning choices may have been significantly altered in the months following the tragic events of 9/11. Estimating Eq. (2) provides

Table 2 Departure of birth outcomes for children *in utero* during 9/11 attack using natality data on births conceived before August 14, 2001, excluding residents of New York City and the Washington, DC, metropolitan area[a]

			Month of Conception							
Birth Outcome	Number of Observations	Mean	Jan. 2001	Feb. 2001	March 2001	April 2001	May 2001	June 2001	July 2001	Aug. 2001[b]
Birth Weight	27,552,002	3,323	–1.48	**–5.60****	**–9.70****	**–7.66****	**–10.07****	**–6.61****	**–8.40****	**–15.29****
			(1.21)	(1.21)	(1.21)	(1.23)	(1.21)	(1.19)	(1.19)	(1.72)
Birth Weight for Gestational Age *z* Score	27,552,002	0.00	0.004*	0.001	0.003	0.000	0.008**	**–0.009****	**–0.011****	**–0.015****
			(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)
LBW (<2,500 grams) (%)	27,552,002	7.4	0.09	0.13*	**0.26****	**0.38****	**0.27****	0.16**	0.19**	0.22**
			(0.05)	(0.05)	(0.05)	(0.06)	(0.05)	(0.05)	(0.05)	(0.08)
VLBW (<1,500 grams) (%)	27,552,002	1.3	0.04	0.00	0.08**	**0.16****	**0.19****	0.09**	0.07**	–0.02
			(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.02)	(0.02)	(0.04)
Preterm (<37 weeks) (%)	27,568,056	11.3	–0.08	**0.51****	**0.98****	**0.67****	**0.35****	0.03	0.15*	**–0.49****
			(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.08)
Male (%)	27,568,056	51.2	0.09	–0.10	0.16	0.05	0.03	–0.05	–0.04	0.08
			(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.15)

Notes: Robust standard errors are shown in parentheses. Values in bold are statistically significant using the Schwarz criteria. Each regression controls for 16-month conception cohort fixed effects, conception week fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8 levels), fixed effects for mother's age (36 levels), fixed effects for mother's education level (18 levels), plurality, mother's diabetes, mother's race (White, African American, Hispanic, and other), child's gender, and an interaction of the conception cohort and mother's state of residence fixed effects.

Source: Data obtained from National Center of Health Statistics 1995–2002 birth certificates.

[a] The *Washington, DC, metropolitan area* is defined as the Washington, DC-MD-VA-WV PMSA.

[b] Conceived between August 1, 2001, and August 14, 2001.

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

Table 3 Change in maternal characteristics for infants conceived after 9/11 attack, excluding residents of New York City and the Washington, DC, metropolitan area[a]

Maternal Characteristic	Number of Observations	Mean	Post-Event Cohort[b]
Mother Is African American (%)	29,821,033	14.2	**–0.19****
			(0.03)
Mother's Years of Education	29,417,747	12.8	**0.019****
			(0.003)
Mother, Some College (%)	29,417,747	45.6	**0.37****
			(0.05)

Notes: Includes all births from January 1, 1995, to December 31, 2003, conceived before March 14, 2002. Robust standard errors are shown in parentheses. Values in bold are significant using the Schwarz criteria. Each regression controls for conception year fixed effects, conception week fixed effects, and mother's state of residence fixed effects.

Source: Data obtained from National Center of Health Statistics 1995–2002 birth certificates.

[a] The *Washington, DC, metropolitan area* is defined as the Washington, DC-MD-VA-WV PMSA.

[b] Considered conceived after event if conception week is after August 14, 2001.

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

insight into whether there was endogenous nonrandom fertility behavior after 9/11 as well as how including those cohorts would potentially have biased the main estimates. The results of this analysis, shown in Table 3, make a strong statement that the mothers who conceived children after the event are significantly different than the mothers from the previous cohort. Specifically, the mothers have a statistically significantly different racial composition (less likely to be African American) and are statistically significantly more educated (both in overall years of school as well as the likelihood of having attended college). Given the bias that would be caused by including infants from mothers that are endogenously self-selected in a way that is positively correlated with birth weight, the choice to cut the sample at those conceived before the event helps preserve the randomness needed for identification.

Alternative Specifications

To assess the sensitivity of the main results from Table 2, I examine several alternative specifications.[20] For example, the estimates in Table A2 of the online appendix exclude residents of not only NYC and DC but also the NYC primary metropolitan statistical area. These estimates are statistically and economically indistinguishable from the baseline results.

In the next two sensitivity tests, many additional control variables are added to the original specification. Given the large number of independent variables and massive sample size being used, the computational burden for these alternative specifications can be quite substantial. To reduce the computational burden for these two tests, I transform the data from individual-level data to combined cell data. Specifically, I

[20] I also conduct similar alternative specifications using only birth data information. The results from these regressions mirror those presented in this section, providing evidence of the robustness of Table 2's findings. These estimates are shown in Tables B2–B5 in the online appendix.

collapse the data such that each cell contains all the individuals from the same county of residence, week of gestation, year of conception, and sex. Each of the variables of interest are calculated as the mean value for each cell group, and the regressions are weighted by the number of individuals that make up each cell. Table A3 in the online appendix is a replication of Table 2 using these new cell data. The coefficient estimates, standard errors, and inference generated in Table A3 are not meaningfully different from those in Table 2.

Although studies have shown a loss in job hours and earnings in NYC after 9/11, it is also quite possible that resource shocks from 9/11 may have differentially and significantly impacted areas all over the country. To address this concern, I add 15 variables to Eq. (1); these variables, calculated from the U.S. Bureau of Labor Statistics, Local Area Unemployment Statistics, indicate the unemployment level in the child's county of residence during the 15 months following the estimated conception date. These added controls can proxy for possible economic fluctuations faced by each child's parents during and following the gestation period.[21] Results from this analysis can be found in Table A4 of the online appendix. The estimates of this sensitivity test are qualitatively equivalent to those found in the main results; if anything, the point estimates from this analysis are generally larger in magnitude than those in Table 2, suggesting that differential economic fluctuations related to the 9/11 tragedy are not driving the results.

Finally, because the computational burden is reduced when the cell-level data are used, an analysis can be conducted in which the state-level (γ_j) and state-time ($\gamma_{yrproxy,j}$) fixed effects are replaced with county-level and county-time fixed effects. By using this finer level of geographic information, any unobserved heterogeneity at the county or county-year level can be swept out of the coefficient estimates of interest. The results from this specification are displayed in Table A5 in the online appendix; as in the rest of this section, the results are not statistically or economically different from those in Table 2. In all, the alternative specifications provide additional support for the main results in terms of magnitudes and the temporal variation of the effect.

Parental Composition and Maternal Behaviors

An important assumption in the models that needs verification is that the composition of the treatment and control groups are not leading to incorrect conclusions. To test for this issue, I conduct an analysis of the maternal characteristics of the treatment group using Eq. (1) but replacing the dependent variable with maternal characteristics.[22] These results are provided in Table 4.

As shown in Table 4, there appears to be no racial composition difference between treatment mothers and control mothers. Additionally, in terms of college attendance and years of education, despite a few significant differences, the positive direction of the coefficients makes it clear that this change is not driving the adverse relationship found in Table 2 between exposure and birth outcomes. The presence of more highly

[21] The economic activity from approximately six months after birth is included in case the parents are able to reasonably predict coming economic hardship/prosperity and made earlier adjustments to their consumption that would affect the relevant pregnancy.

[22] Maternal characteristic controls are excluded from these regressions.

Table 4 Departure of maternal attributes of children *in utero* during 9/11 attack using natality data on births conceived before August 14, 2001, excluding residents of New York City and the Washington, DC, metropolitan area[a]

			Month of Conception							
Maternal Characteristics	Number of Observations	Mean	Jan. 2001	Feb. 2001	March 2001	April 2001	May 2001	June 2001	July 2001	Aug. 2001[b]
Mother Is African American (%)	27,568,056	14.2	0.02	–0.04	0.12	–0.04	0.04	–0.16*	–0.06	–0.04
			(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.10)
Mother's Years of Education	27,196,339	12.8	0.01	0.00	0.01	0.01	0.01	**0.04****	**0.03****	**0.04****
			(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Mother Has Some College (%)	27,196,339	45.5	0.04	0.05	0.12	0.02	0.16	**0.62****	**0.46****	0.61**
			(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.15)
Number of Live Births[c]	781,490	35.3	0.21	1.39	1.14	0.08	0.34	1.01	1.16	1.37
			(1.14)	(1.14)	(1.14)	(1.12)	(1.14)	(1.15)	(1.17)	(1.43)

Notes: Robust standard errors are shown in parentheses. Values in bold are statistically significant using the Schwarz criteria. Each regression controls for 16-month conception cohort fixed effects, conception week fixed effects, mother's state of residence fixed effects, and an interaction of the conception cohort and mother's state of residence fixed effects.

Source: Data obtained from National Center of Health Statistics 1995–2002 birth certificates.

[a] The *Washington, DC, metropolitan area* is defined as the Washington, DC-MD-VA-WV PMSA.

[b] Conceived between August 1, 2001, and August 14, 2001.

[c] This analysis uses the cell-level data described in the Alternative Specifications section.

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

educated mothers in the early gestation exposure groups may also contribute to the estimated negative relationship between exposure in the first month of gestation and preterm births.

One potential cause of this positive selection in mother's conceiving in June, July, and August of 2001 is selective miscarriage or abortion for lower-educated women following the 9/11 attacks. To test this, I use the cell-level data to conduct a regression similar to Eq. (1), with the total number of live births in each cell as the dependent variable. This analysis explores whether the cohorts exposed *in utero* to the 9/11 attacks are smaller, the same, or larger than surrounding cohorts. If they are smaller, this would indicate the presence of increased miscarriage or abortion as a result of elevated maternal psychological distress. The results of this analysis, shown in row 4 of Table 4, suggest that there is no statistically significant relationship between exposure to the 9/11 attacks during pregnancy and the likelihood of having a live birth.

In addition, to explore whether the poor birth outcomes found in Table 2 are related to the biological mechanisms connecting stress to restricted intrauterine growth and gestational age or stress-related behavioral responses of the mothers, it is important to examine whether the events of 9/11 adversely impacted mothers' health behaviors. To conduct this analysis, I calculate Eq. (1) with maternal behaviors (maternal weight gain, as well as, indicators for whether prenatal care started late or never was used, smoking during pregnancy, and alcohol use during pregnancy) as the dependent variables.[23] As shown in Table 5, there does not seem to be any systematic negative behavioral reaction by mothers to being exposed to the 9/11 events. In fact, it appears that mothers who conceived just before the 9/11 attacks went on to more actively and more quickly utilize prenatal care, which may help to explain the counterintuitive negative relationship between exposure in the first month of gestation and preterm births.

Heterogenous Effects

The main results suggest that increased psychological stress among pregnant women leads to statistically significantly poorer birth outcomes for the children exposed to 9/11 *in utero*, but this finding may be hiding larger impacts for important subpopulations. For example, a recent study by Torche and Kleinhaus (2012) found that maternal exposure to psychological stress *in utero* has a much stronger negative impact on female children, particularly early in gestation. To explore this issue, I calculate estimates splitting the sample by gender. Table 6, rows 1 and 2, contain the results on the impact of acute maternal psychological stress on birth weight for the male and female infant population, respectively. These findings do not show any clear pattern of one gender being discernibly more sensitive to insults of maternal psychological stress.[24]

One possible concern with the approach taken in Eq. (1) is that by excluding individuals living in the attacked cities, the analysis may lose some of the most

[23] As shown in Table 1, there are a nontrivial number of observations missing alcohol (14.8%) and smoking (17.7%) behavior. In addition, these variables have strong potential for being measured with error. However, because it is unlikely that the missingness or possible inaccuracy is caused by or related to the 9/11 attacks, these issues, at worst, lead to a decrease in external validity and precision, but they do not generate bias.

[24] Similarly, there is no evidence of gender heterogeneity in the impact of maternal psychological stress *in utero* on gestational age. Results are available upon request.

Table 5 Departure of maternal behaviors of children *in utero* during 9/11 attack using natality data on births conceived before August 14, 2001, excluding residents of New York City and the Washington, DC, metropolitan area[a]

			Month of Conception							
Maternal Behavior	Number of Observations	Mean	Jan. 2001	Feb. 2001	March 2001	April 2001	May 2001	June 2001	July 2001	Aug. 2001[b]
Late/No Prenatal Care[c] (%)	27,033,176	3.7	–0.02	0.02	–0.06	–0.03	–0.04	–0.07	–0.10*	–0.15**
			(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.05)
Maternal Weight Gain	22,013,433	30.8	0.02	0.03	0.06	0.08*	**0.15****	**0.17****	0.10**	0.01
			(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.05)
Smoking While Pregnant (%)	22,691,092	13.3	–0.06	–0.01	0.05	–0.03	–0.12	–0.19**	–0.16*	–0.18
			(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.10)
Alcohol Use While Pregnant (%)	23,483,669	1.1	0.00	0.02	0.01	0.02	0.03	–0.02	0.01	0.02
			(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.03)

Notes: Robust standard errors are shown in parentheses. Values in bold are statistically significant using the Schwarz criteria. Each regression controls for 16-month conception cohort fixed effects, conception week fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8 levels), fixed effects for mother's age (36 levels), fixed effects for mother's education level (18 levels), plurality, mother's diabetes, mother's race (White, African American, Hispanic, and other), child's gender, and an interaction of the conception cohort and mother's state of residence fixed effects.

Source: Data obtained from National Center of Health Statistics 1995–2002 birth certificates.

[a] The *Washington, DC, metropolitan area* is defined as the Washington, DC-MD-VA-WV PMSA.

[b] Conceived between August 1, 2001, and August 14, 2001.

[c] *Late/no prenatal care* is defined as either starting prenatal care in the third trimester or never receiving prenatal care.

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

Table 6 Departure of birth weight for children *in utero* during 9/11 attack using natality data on births conceived before August 14, 2001, excluding residents of New York City and the Washington, DC, metropolitan area[a]

			Month of Conception							
Subgroup	Number of Observations	Mean	Jan. 2001	Feb. 2001	March 2001	April 2001	May 2001	June 2001	July 2001	Aug. 2001[b]
Males	14,093,082	3,378	–0.63	–4.69**	**–9.89****	**–7.94****	**–10.09****	–6.03**	**–8.69****	**–12.66****
			(1.74)	(1.74)	(1.73)	(1.76)	(1.73)	(1.71)	(1.71)	(2.45)
Females	13,458,920	3,265	–2.36	–6.50**	**–9.51****	**–7.40****	**–10.02****	**–7.16****	**–8.04****	**–18.09****
			(1.68)	(1.68)	(1.68)	(1.71)	(1.68)	(1.66)	(1.65)	(2.42)
Large Cities	1,747,147	3,269	–2.55	–3.93	–16.51**	–9.52	**–19.47****	–11.05*	–13.02**	–3.98
			(4.98)	(4.94)	(4.97)	(4.99)	(5.00)	(5.00)	(5.00)	(7.11)
High School Graduate	21,597,004	3,346	–2.38	**–5.78****	**–9.60****	**–7.74****	**–10.17****	**–6.78****	**–7.92****	**–14.47****
			(1.37)	(1.37)	(1.36)	(1.38)	(1.36)	(1.33)	(1.33)	(1.93)
Not High School Graduate	5,954,998	3,240	2.05	–5.24*	–9.97**	–6.96**	–9.19**	–4.96	–9.22**	**–17.58****
			(2.61)	(2.58)	(2.61)	(2.65)	(2.64)	(2.65)	(2.65)	(3.84)

Notes: Robust standard errors are shown in parentheses. Values in bold are statistically significant using the Schwarz criteria. Each regression controls for 16-month conception cohort fixed effects, conception week fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8 levels), fixed effects for mother's age (36 levels), fixed effects for mother's education level (18 levels), plurality, mother's diabetes, mother's race (White, African American, Hispanic, and other), child's gender, and an interaction of the conception cohort and mother's state of residence fixed effects.

Source: Data obtained from National Center of Health Statistics 1995–2002 birth certificates.

[a] The *Washington, DC, metropolitan area* is defined as the Washington, DC-MD-VA-WV PMSA.

[b] Conceived between August 1, 2001, and August 14, 2001.

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

intensely distressed mothers. Although qualitative research suggests that a nontrivial number of individuals throughout the country felt increased psychological stress from the 9/11 attacks, the next few estimates look into a few important subgroups in an attempt to find an upper bound on the impact of psychological stress exposure.

A subpopulation that may have experienced higher levels of psychological stress after the 9/11terrorist attacks comprises those living in large cities. Individuals living in large cities other than NYC and DC may have experienced a higher exposure to psychological stress because they may have internalized the fact that the areas they lived in would be the most likely targets for any potential future attacks. Thus, in an attempt to examine whether the country-wide sample is obscuring some larger effect of psychological stress on birth outcomes, I run the analysis on only those mothers living in cities (other than NYC and DC) with a population more than 1,000,000 residents. Although an analysis focused solely on residents of big cities loses some of the identification clarity of the baseline analysis because it faces potential issues of selective migration, it should provide some evidence of whether the country-wide analysis grossly underestimates the impact of psychological stress exposure. The third row of Table 6 displays the estimates of the impact of the psychological stress of 9/11 on the birth weight of children *in utero* during the attacks among women living in large cities. Most of the point estimates from this analysis are larger than when the entire sample is used, but these results do not suggest that the findings in Table 2 substantially underestimate the effects of individuals living in more intensely exposed areas.

Another set of hypotheses would suggest that education may be related to the level of psychological burden a mother experiences as a result of the attacks or that the education of the mother may influence her ability to insulate her pregnancy from the trauma of the event. Table 6, rows 4 and 5, provide estimates when the sample is limited to mothers with a high school diploma and those without, respectively. Neither of these two subgroups produce results that are qualitatively different from those in Table 2; moreover, there is no clear pattern to suggest that the mother's education had any impact on the relationship between exposure and birth outcomes.

Conclusion

Using an unfortunate and unanticipated national tragedy and a robust source of data, this study estimates the impact that elevated maternal stress has on birth outcomes. The strength of the analysis as compared with the previous literature is the ability to develop a clean identification strategy by excluding residents of the attacked areas, who were exposed to other important health and resource shocks in addition to psychological stress, and limiting the sample to those who had made their fertility decision before the event. The findings of this study suggest that infants exposed *in utero* to increased maternal stress were born significantly smaller and earlier than previous cohorts. In addition, the adverse relationship between maternal stress exposure and important birth weight cutoffs used by clinicians, such as LBW and VLBW, are driven by reductions in the length of gestation rather than fetal development. These relationships are found despite the fact that the estimates are limited to leveraging the possible maternal psychological distress of the 9/11 attacks rather than anthropometric measures of maternal stress levels. Last, a novel feature of this study is that it provides a month-

by-month analysis indicating that the timing of the stress insult does lead to important differences in the health outcome of the child: intrauterine growth is most sensitive to stress shocks in the first trimester, and gestational age is most susceptible mid-pregnancy.

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Data Availability All data used for this article are publicly and freely available. The Vital Statistics Natality Birth Data is available at https://www.cdc.gov/nchs/nvss/births.htm. U.S. Bureau of Labor Statistics is available at https://www.bls.gov/.

Compliance With Ethical Standards

Conflict of Interest The author declares that there are no conflicts of interest.

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