

Abnormal labor outcomes as a function of maternal exposure to a catastrophic hurricane event during pregnancy

Sammy Zahran · Lori Peek · Jeffrey G. Snodgrass ·
Stephan Weiler · Lynn Hempel

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Abstract Hurricane Andrew, which made landfall on August 24, 1992, was one of the most destructive hurricanes in American history, causing atypically high levels of psychological and physical health impairment among the resident population and especially among vulnerable groups. This article investigates whether maternal exposure to Hurricane Andrew during pregnancy increased the risk of dystocia (or dysfunctional labor) and infant delivery by cesarean section, the standard medical response to abnormal labor progression. We analyze 297,996 birth events in Miami-Dade and Broward counties in Florida from 1992 to 1993 using propensity score methodology with stratification and nearest-neighbor matching algorithms. Results show that hurricane-exposed pregnant women were significantly more likely to experience stress-induced abnormal labor and cesarean delivery outcomes as compared to statistically matched comparison groups. The conclusion details the policy implications of our results, with particular attention to the importance of maternal prenatal care in the aftermath of disasters.

Keywords Dysfunctional labor · Cesarean section · Maternal exposure · Hurricane Andrew · Disaster · Gender · Vulnerability · Propensity scoring

S. Zahran (✉) · S. Weiler
Economics, Colorado State University, Fort Collins, CO, USA
e-mail: szahran@colostate.edu

S. Weiler
e-mail: Stephan.Weiler@colostate.edu

L. Peek · L. Hempel
Sociology, Colorado State University, Fort Collins, CO, USA
e-mail: Lori.Peek@colostate.edu

L. Hempel
e-mail: Lynn.Hempel@colostate.edu

J. G. Snodgrass
Anthropology, Colorado State University, Fort Collins, CO, USA
e-mail: Jeffrey.Snodgrass@colostate.edu

1 Introduction

Hurricane Andrew, which made landfall along the southern tip of Florida on August 24, 1992, remains the second costliest natural disaster in American history, causing an estimated \$26.5 billion in property loss (\$40.6 billion in 2011 U.S. dollars). The Category 5 winds left a band of destruction 300 blocks wide (Girard and Peacock 1997) and affected about 130,000 households in South Dade (Morrow 1997). More than 180,000 persons left their homes for some period of time, while 1.4 million had no electricity for days or weeks (Lewis 1993). Hurricane Andrew produced considerable familial and social disruption, resulting in a 20% increase in parents relinquishing custody of their children; a 30% higher divorce rate in Dade county; permanent closure of numerous child care centers, schools, and businesses; sharp increases in regional unemployment; and unequal recovery outcomes by race, ethnicity, gender, and social class (Peacock et al. 1997).

Norris et al. classify Hurricane Andrew as a “high-impact disaster event” based on the “atypically high levels” of physical and emotional impairment observed among affected youth and adult populations (Norris et al. 2002). For example, blood samples from impacted populations in Miami differed significantly from control groups on measures of depressed immune function, including natural killer cell cytotoxicity, white blood cell, and CD4 and CD8 counts (Ironson et al. 1997). College students who experienced Hurricane Andrew showed more symptoms of physical stress and did more poorly on cognitive tasks than classmates who were not directly affected by the hurricane (Rotton et al. 1996). A study of residents in southern Dade county found that 6 months post-Andrew, 20–30% of adults in the area met criteria for PTSD and 33–45% were found to be meaningfully depressed (Norris et al. 1999).

The widespread negative physical and psychological health effects of Hurricane Andrew are generally well documented. However, less research is available on the gender-specific health-related consequences of this event. In this article, we examine the relationship between pregnant women’s exposure to Hurricane Andrew and abnormal pregnancy outcomes. A limited body of prior research shows that maternal stress caused by disaster during pregnancy increases the risk of spontaneous pre-term birth, dystocia or abnormal labor, and cesarean section delivery and may compromise the health of an unborn fetus and future development of an infant (Zahran et al. 2010; Paarlberg et al. 1995; Laplante et al. 2004; King et al. 2009). Insofar as Hurricane Andrew caused generalized stress among the resident population, and maternal stress during pregnancy increases the risk of negative birth outcomes, we anticipate a higher rate of abnormal labor outcomes among pregnant women exposed to Hurricane Andrew.

This investigation of maternal exposure to Hurricane Andrew and dysfunctional labor is organized into four sections. First, we review the literature on women’s disproportionate vulnerability to disaster and integrate scientific literature on links between maternal stress during pregnancy and negative birth outcomes. Second, we detail the aspects of our research design, including statistical procedures and variable operations. Third, we present descriptive, regression, and propensity score matching results, testing whether maternal exposure to Hurricane Andrew (in developmentally meaningful periods of gestation) increased the risk of both dystocia and primary cesarean section outcomes among pregnant women. We conclude with a discussion of the policy implications of our results.

2 Literature review

2.1 Women and disaster vulnerability

Several studies since the 1990s have found that women are disproportionately vulnerable to negative mental and physical health outcomes following disasters (Fothergill 1996, 2004). Discrimination, power differentials, a lack of mobility, and gendered social roles all heighten the risks women face in disasters, with low-income and racial-ethnic minority women (Enarson et al. 2006) and single mothers (Tobin-Gurley et al. 2010) often experiencing the most severe vulnerability due to their caregiving responsibilities and structural locations in society.

Following Hurricane Andrew, Ironson et al. (1997) found that gender was a significant correlate of post-traumatic stress disorder (PTSD) symptoms, with affected women reporting more traumatic stress than men. Women who relocated from their homes for more than 1 week following Andrew experienced elevated rates of depression when compared with their male counterparts (Riad and Norris 1996), and those women who sought temporary shelter in temporary trailer camps reported that they felt isolated and fearful (Morrow and Enarson 1996). Low-income African-American women in Miami were among those most likely to return to heavily damaged substandard housing units after the hurricane (Grenier and Morrow 1997). Single mothers received the least assistance preparing for and recovering from Andrew (Enarson and Morrow 1997) and subsequently reported elevated stress in their relationship with significant others, children, relatives, and friends (Morrow 1997).

Zahran et al. (2010) found that fetal distress risk increased significantly with maternal exposure to Hurricane Andrew in second and third trimesters, adjusting for known risk factors. Moreover, higher incidences of fetal distress risk occurred in areas most impacted by the hurricane and among African-American mothers. Studies following Hurricane Katrina also revealed that women were significantly more likely to give birth to low-weight infants after the storm (Callaghan et al. 2007). Negative effects on women's and infants' health were likely due to tenuous access to health care before the hurricane (Bennett 2005), and to further deterioration to maternal and child health programs after the disaster (Jones-DeWeever 2008).

2.2 Maternal stress in pregnancy

Experiments on rodents and non-human primates provide considerable evidence that maternal stress during pregnancy increases negative birth outcomes (Anderson et al. 1985; Maccari et al. 2003; Neumann et al. 1998; Morishima et al. 1979; Pinto and Shetty 1995; Rhee and Fleming 1981; Rondó 2007; Weinstock 1997, 2001, 2002, 2005; Weinstock et al. 1992, 1988; Williams et al. 1995, 1999). Retrospective and prospective studies on human subjects demonstrate similar links between maternal stress and fetal health (Rondó 2007; Weinstock 1997, 2001, 2005; Diego et al. 2006; Mulder et al. 2002). Maternal stressors may be physiological or psychological, resulting from malnutrition, depression, anxiety, insufficient social support, and/or acute trauma (Barker and Osmond 1986; Gluckman et al. 2008; Kuzawa and Sweet 2009; Wadhwa et al. 1996, 1997; Yehuda and Bierer 2008). Additional studies relate a mothers' perceived stress during gestation to increased frequency of pre-term births, low birth weight, and dysfunctional labor outcomes (Wadhwa et al. 1996, 1997; Dole et al. 2003).

The pathways linking maternal stress with negative birth outcomes are not fully understood, and a constellation of mechanisms are presumed involved (Rondó 2007; Weinstock 2005). Research supports the involvement of neuroendocrine systems and the circulation of

hormones in pregnant women and their unborn infants (Diego et al. 2006; McEwen 2001), as well as the activation of the sympathetic nervous system and the hypothalamus–pituitary–adrenal (HPA) axis. In expectant mothers, a stress response involving the release of hormones like epinephrine and/or cortisol draws blood to peripheral systems like muscles and limbs and away from processes such as reproduction (Chrousos 1997; Omer 1986; Sjostrom et al. 1997; Teixeira et al. 1999). Maternal stress is also shown to influence blood flow to the endometrial lining or placenta, drawing vital nutrients and oxygen away from developing fetuses (Gluckman 2004). Such maternal changes associated with a stress response could explain negative birth outcomes like dysfunctional labor and associated medical interventions to dystocia like delivery by cesarean section.

An estimated two-thirds of non-elective cesarean section procedures result from dystocia or ‘abnormal’ labor (Gifford et al. 2000). Based on a review of extant research, the multifactor causes of dystocia and c-sections are modeled by Lowe and include women’s physical and psychological characteristics, fetal factors, intrapartum care and interventions, assessments and clinical decision-making of health care providers, and the social and physical environment of childbirth (Lowe 2007). Important among the causes identified by Lowe is stress. Environmental stressors can lead to the release of excess amounts of the catecholamines epinephrine (EPI) and norepinephrine (NEP). Such a release can interfere with uterine contractions in straightforward mechanisms involving beta-adrenergic receptors on smooth muscles such as are commonly found in the uterus of humans and non-human primates (Lowe 2007; Monga and Sanborn 2004). Lowe observes: “The clinical hypothesis is that exaggerated production of catecholamines, particularly epinephrine, in response to pain and stress, disrupts coordinated uterine contractions by binding with beta-adrenergic receptors on the myometrial smooth muscle cells” [55: 220].¹

Summarizing the logic and evidence, Hurricane Andrew is observed to have caused generalized stress in resident population, with women experiencing high levels of measured depression, anxiety, and psychological trauma. Among pregnant women, stress is known to complicate pregnancy outcomes. The precise reasons why the experience of heightened stress during pregnancy complicates birth outcomes are not fully understood, but researchers believe that stress-related hormones are involved. Stress-related hormones are directly implicated in fetal outcomes like hypoxia and appear to partially inform abnormal labor outcomes by compromising uterine contractility (Zahran et al. 2010). In terms of a testable causal logic, then, we anticipate higher risk of dysfunctional labor among pregnant women exposed to Hurricane Andrew, as well as higher risk of delivery by cesarean section. In the next section, we describe variable operations and our empirical strategy to assess this intuition.

3 Research design

3.1 Dependent variable operations

In this article, we draw on birth outcome information from the National Center for Health Statistics, Vital Statistics Natality Birth files. Two dependent outcomes are examined:

¹ Although an emerging body of research shows links between psychosocial stress and negative pregnancy outcomes like preterm births, studies on stress-related problems related to labor at term are rare (Lowe 2007). Existing studies reveal associations between psychological measures of stress experienced by women, elevated plasma EPI, and dystocia (Lowe 2007; Crammond 1954; Zuspan et al. 1962; Kapp et al. 1963; Lederman et al. 1977; Lederman et al. 1978, 1979; Austin and Leader 2000; Alehagen et al. 2001).

dysfunctional labor and *primary c-section*. Dysfunctional labor is measured as a binary outcome, with 1 = condition present and 0 = condition not present. This condition was present in approximately 2.059% of 297,996 fully observed births in the state of Florida from 1992 to 1993. Normal vaginal deliveries are characterized by known milestones in that normal labor involves uterine contractions that progressively dilate and efface the cervix (Friedman 1955). Failure to meet these milestones defines dysfunctional labor, indicating increased risk of an unfavorable birth outcome. Dysfunctional labor can result from inadequate size and contour of the pelvis, malpresentation of the infant, and/or insufficient power or uterine contractility. Protracted or arrested cervical dilatation alerts the obstetrician to consider delivery by cesarean section to minimize health risks to the mother and infant. Blood samples taken from women having undergone cesarean section indicate that myometrial lactic acidosis and decreased oxygen saturation may influence uterine contractility and dysfunctional labor outcomes (Quenby et al. 2004). If maternal stress response is associated with compromised contractility, it follows that mother's exposed to a stressful event like a catastrophic hurricane may be at greater risk of abnormal delivery.

Primary cesarean section is also measured as a binary variable, with 1 = condition present and 0 = condition not present. Primary c-sections were performed on 15.2% of observed births in the state of Florida over our study period. Analytically, insofar as maternal exposure to Hurricane Andrew induced a stress response, increasing the likelihood that a pregnant woman will experience a dysfunctional labor, then we ought to observe a similar increase in the probability of cesarean section delivery (which is a common medical response to abnormal labor indication).

3.2 Empirical strategy

Maternal exposure to Hurricane Andrew (e) is determined by the date and geography of the hurricane event (h), the estimated clinical gestational age of the child at birth (g), the estimated date of live birth (b), and maternal county residence as indicated in National Center for Health Statistics (NCHS) Vital Statistics Natality Birth Data, 1992–1993. Insofar as the live birth date follows the landfall date of the hurricane, and that the birth event occurs in a hurricane-affected area, maternal exposure is calculated as gestational age minus the difference between the birth and hurricane landfall dates, $e = g - (b - h)$. The derived value indicates the estimated week of gestation a mother was exposed to the hurricane event. Although data do not allow the identification of pregnant women who suffered directly from the event in terms of property loss or other forms of harm, Hurricane Andrew was sufficiently destructive of routine life in Miami-Dade and Broward counties that one can reasonably assume generalized stress in resident population and particularly for the exposed female population (Ironson et al. 1997). Our coding of maternal exposure assumes that the reported county of residence is where a mother lived through the gestation period.² To strengthen this assumption, we limit our analysis to mothers with matched birth occurrence and residential county codes. To investigate the relationship between maternal exposure to Hurricane Andrew and dysfunctional labor and primary c-section outcomes,

² Unlike Hurricane Katrina, which led to the temporary evacuation of over 1.5 million people and the permanent displacement of hundreds of thousands, Hurricane Andrew did not cause such large-scale displacement. The majority of persons rendered homeless by Hurricane Andrew relocated to the homes of family or friends, "tent cities," hotels/motels, or other temporary shelters within Miami-Dade and Broward counties (Yelvington 1997).

we use a semi-parametric propensity score matching methodology, the logic of which is described below.

Let y_{1i} denote the pregnancy outcome for mother i if exposed to Hurricane Andrew (given by $T_i = 1$), and let y_{0i} denote the pregnancy outcome of mother i if not exposed to Andrew ($T_i = 0$). The average exposure effect, τ , equals, where the first term represents the mean effect observed for exposed mothers and the second term indicates the average pregnancy outcome for unexposed mothers. Absent random assignment to exposed or unexposed groups, generally $\tau \neq \hat{y}_1 - \hat{y}_0$. Studies on socially vulnerable populations show that natural disasters impact demographic groups non-randomly (Cutter et al. 2003; Zahran et al. 2008). Therefore, hurricane-exposed pregnant women are likely to differ substantially from non-exposed pregnant women in the rest of Florida on relevant demographic characteristics, making exposed and unexposed pregnant women statistically *non-exchangeable*.

Regression technologies are commonly used to resolve the problem of non-identical or non-exchangeable exposure groups. However, regression solutions can suffer from *omitted variable bias*. If relevant covariates go unmeasured, effect estimates are confounded (Oakes 2006). A more subtle problem, called *off-support inference*, involves the distribution of regression covariates for each exposure group. Group comparison requires that unexposed persons have some probability of receiving the treatment; otherwise, effect estimates are pure extrapolation or off-support of the data (Rosenbaum 2002). Treatment effect estimation is only reasonable when exposure groups are exchangeable and have some non-zero probability of being exposed to the treatment (Oakes 2006). The problem of *off-support inference* in regression analysis can obscure fundamental differences between exposure groups, undermining the identification of treatment effects by observation.

Another approach to resolving observed differences between exposed and unexposed groups is sub-classification. With large datasets, covariate overlap or distributional balance between exposed and unexposed groups on relevant characteristics can be assessed. However, with only six binary demographic variables on which to sub-classify exposure groups, for example, one needs 64 strata to achieve exchangeability. If the analytic problem involves relevant continuous covariates, sub-classification is near computationally impossible.

Propensity score matching addresses this covariate *dimensionality problem* as well as the problem of *off-support inference*. The propensity score matching solution advanced by Rosenbaum and Rubin is to find a vector of covariates, Z , such that

$$y_1, y_0 \perp T | Z, \quad \text{pr}(T = 1 | Z) \in (0, 1), \quad (1)$$

where \perp denotes independence (Rosenbaum and Rubin 1983). To estimate average treatment effects, in our case maternal exposure to Hurricane Andrew, the following weaker condition is required:

$$E[y_0 | T = 1 | Z] = E[y_0 | T = 0 | Z] = E[y_0 | Z], \quad \text{pr}(T = 1 | Z) \in (0, 1), \quad (1')$$

To obtain condition (1'), the conditioning set of Z should be multidimensional. Finding observations with identical values for covariates in Z is computationally untenable. However, Rosenbaum and Rubin prove that conditioning on $p(Z)$ is equivalent to conditioning on Z , where $p(Z) = \text{pr}(T = 1 | Z)$ is the propensity score (Rosenbaum and Rubin 1983). This resolves the dimensionality problem in sub-classification by matching exposure groups on a single dimension or propensity score. The propensity score $p(Z)$ is

estimated for hurricane-exposed and unexposed mothers via probit. With the propensity score estimated, a matching algorithm is used.

The most common algorithms are *stratification* and *nearest-neighbor* matching. The *stratification method* involves dividing the range of propensity scores into intervals or blocks such that within each block exposed and unexposed mothers have, on average, equal propensity scores (Rosenbaum 2002). In other words, hurricane-exposed mothers are matched with hurricane-unexposed mothers on shared demographic characteristics that condition one's likelihood of receiving a treatment effect (in our case being exposed to Hurricane Andrew during pregnancy). In nearest-neighbor matching, each exposed mother is paired with an unexposed mother whose propensity score is closest in absolute value (Dehejia and Wahba 2002).³ Unmatched unexposed mothers are dropped from the dataset. Both matching methods identify unexposed mothers that approximate/match exposed mothers on relevant demographic characteristics that enable exchangeability. The pregnancy effects of maternal exposure to Hurricane Andrew on exposed mothers (IT) are therefore given by

$$\begin{aligned}\tau_{IT} &= E[y_1 | T = 1, p(Z)] - E[y_0 | T = 0, p(Z)] \\ &= E[y_1 - y_0 | p(Z)].\end{aligned}\quad (2)$$

Balancing and specification tests are conducted to assure that after conditioning on the propensity score, the distribution of the conditioning covariates Z does not differ across exposure groups in the matched sub-sample. In analyses that follow, the density distribution of propensity scores of hurricane-exposed and unexposed pregnant women is similar.

The probit model of the probability of each pregnant mother being exposed to Hurricane Andrew involves the analysis of a suite of demographic variables that condition the probability of pregnant female exposure to the hurricane event. Both empirical and statistical reasons inform selection of demographic variables to derive propensity score-matched comparison groups. Empirically, Hurricane Andrew-exposed counties of Miami-Dade and Broward are demographically distinct. Miami-Dade is home to a disproportionately high number of Hispanics, and Broward County is home to a disproportionately high number of African-Americans. Marital status and educational attainment, as indicators of socioeconomic status, influence residential choices as do historical and contemporary patterns of housing discrimination. The confluence of these and many other factors results in racial and ethnic minorities and persons of lower socioeconomic status disproportionately residing in zones of higher hurricane risk in Miami-Dade and Broward counties (Norris et al. 2002). Conditioning variables to derive propensity scores therefore include maternal marital status (1 = married, 0 = non-married), maternal educational attainment (1 = mother with at least 1 year of post-secondary education; 0 = high school education or less), maternal Hispanic identity (1 = mother Hispanic, 0 = mother non-Hispanic), and maternal African-American identity (1 = mother African-American, 0 = mother non-African-American). These demographic variables were also obtained from the National Center for Health Statistics, Vital Statistics Natality Birth files. Descriptive statistics and variable operations for variables examined for the 297,996 fully observed births in Florida from 1992 to 1993 are summarized in Table 1.

³ Denote by $U(i)$ the set of unexposed mothers matched to an exposed mother i with an estimated propensity score of p_i . Nearest-neighbor matching sets $U(i) = \min \|p_i - p_j\|$, allowing for multiple nearest neighbors.

Table 1 Variable definitions and descriptive statistics

Variable label	Variable definition	Mean	Standard deviation	Min	Max
Balancing variables					
Marital status	1 = mother married; 0 = mother not married.	0.6576	0.4745	0	1
Post-secondary education	1 = mother with at least 1 year of post-secondary education; 0 = high school education or less.	0.4006	0.49001	0	1
Hispanic	1 = mother Hispanic, 0 = mother non-Hispanic.	0.1798	0.3840	0	1
African-American	1 = mother African-American, 0 = mother non-African-American.	0.2391	0.4265	0	1
Treatment variables					
Gestation exposure	Maternal exposure to catastrophic hurricane event during gestation. 1 = condition present, 0 = condition not present.	0.1404	0.3475	0	1
First-trimester exposure	Maternal exposure to catastrophic hurricane event 0–12 weeks into gestation. 1 = condition present, 0 = condition not present.	0.0403	0.1966	0	1
Second-trimester exposure	Maternal exposure to catastrophic hurricane event 13–25 weeks into gestation. 1 = condition present, 0 = condition not present.	0.0440	0.2051	0	1
Third-trimester exposure	Maternal exposure to catastrophic hurricane event 26+ weeks into gestation. 1 = condition present, 0 = condition not present.	0.0562	0.2303	0	1
Response variables					
Dysfunctional labor	Maternal failure to progress in a normal pattern of labor. 1 = condition present, 0 = condition not present.	0.0206	0.1420	0	1
Primary C-section	Primary cesarean section delivery. 1 = procedure used, 0 = procedure not used.	0.1516	0.3586	0	1

Statistically, in our study, the main aim of propensity score matching is to balance hurricane-exposed and unexposed pregnant mothers on relevant characteristics that may influence the non-random experience of being exposed to a destructive hurricane. So long as hurricane-exposed and unexposed pregnant mothers are similar on the propensity of hurricane exposure, observed differences in pregnancy outcomes like dysfunctional labor and c-section delivery can be attributed to the hurricane effect and not differences in demographic characteristics that may influence these pregnancy outcomes. Satisfying the balancing property of group matching does not eliminate the problem of omitted variable bias, but over sufficiently large samples and Gaussian model residuals, this bias is minimized.

4 Results

Table 2 reports regression coefficients used to calculate propensities of maternal hurricane exposure during pregnancy, as well as during the first (0–12 weeks), second (13–25 weeks), and third (26+ weeks) trimesters. Statistically, selected covariates satisfy a balancing property of the matching methodology, where mean propensity scores of

Table 2 Probit regression coefficients predicting treatment exposure

	Gestation exposure	First-trimester exposure	Second-trimester exposure	Third-trimester exposure
Marital status	0.0457*** (0.0071)	0.0321*** (0.0102)	0.0230** (0.0100)	0.0423*** (0.0092)
Hispanic	0.8180*** (0.0072)	0.5692*** (0.0100)	0.5994*** (0.0098)	0.6363*** (0.0091)
African-American	0.4767*** (0.0076)	0.3282*** (0.0111)	0.3822*** (0.0107)	0.3743*** (0.0099)
Post-secondary	0.1105*** (0.0065)	0.0991*** (0.0093)	0.0729*** (0.0091)	0.0769*** (0.0084)
Constant	-1.4723*** (0.0071)	-2.0366*** (0.0103)	-2.0037*** (0.0100)	-1.9054*** (0.0093)
Log likelihood	-113,377.5	-48,460.79	-51,523.55	-61,533.45
LR	13,934.89	3,320.65	3,982.04	5,094.55
Count R^2	0.860	0.960	0.956	0.944

Standard errors in parentheses *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Table 3 Number of exposed and unexposed mothers by the inferior bound of propensity score blocks

	Inferior bound of propensity score blocks	Unexposed pregnant women	Exposed pregnant women	Total pregnant women
	0	919	221	1,140
	0.05	28,519	1,899	30,418
	0.075	56,323	4,386	60,709
	0.08125	6,712	626	7,338
	0.0875	68,400	7,447	75,847
	0.15	33,213	6,761	39,974
	0.1625	10,059	2,264	12,323
	0.175	6,035	1,305	7,340
	0.2	7,819	1,712	9,531
	0.25	25,832	9,707	35,539
	0.3	11,096	5,071	16,167
	0.4	1,217	453	1,670
	Total	256,144	41,852	297,996

hurricane-exposed and unexposed women are statistically similar within an optimal number of propensity score blocks. All variables in the probit model perform as expected. Table 3 reports the number of exposed and unexposed mothers by the inferior bound of derived propensity score blocks. With twelve balanced blocks derived, we compare hurricane-exposed versus unexposed pregnant women. Again, only pregnant women with equal demographically determined propensity to be exposed to hurricane-related stress are compared on the birth complication of dysfunctional labor and consequent intervention of primary c-section.

Table 4 reports independent samples t test results by propensity score blocks. In 7 of the 12 blocks, we observe significant mean differences ($p \leq 0.05$) between hurricane-exposed and unexposed pregnant women on the proportion of live births with dysfunctional labor

Table 4 Independent samples *t* test of exposed versus unexposed pregnant women on dysfunctional labor by block

Block (propensity score)	Control (<i>C</i>) group: hurricane unexposed		Treatment (<i>T</i>) group: hurricane exposed		$T_{\mu}-C_{\mu}$	<i>t</i> test	Pr ($T < C$)
	<i>N</i>	Mean (SE)	<i>N</i>	Mean (SE)			
Block 1	919	0.0163 (0.0042)	221	0.0136 (0.0078)	-0.0027	-0.2939	0.6156
Block 2	28,519	0.0147 (0.0007)	1,899	0.0205 (0.0033)	0.0058	2.0111	0.0222
Block 3	56,323	0.0162 (0.0005)	4,386	0.0276 (0.0025)	0.0114	5.5992	0.0000
Block 4	6,712	0.0222 (0.0018)	626	0.0383 (0.0077)	0.0161	2.5462	0.0055
Block 5	68,400	0.0213 (0.0006)	7,447	0.0436 (0.0024)	0.0224	12.1324	0.0000
Block 6	33,213	0.0116 (0.0006)	6,761	0.0124 (0.0013)	0.0009	0.6009	0.2739
Block 7	10,059	0.0106 (0.001)	2,264	0.0128 (0.0024)	0.0024	0.8937	0.1858
Block 8	6,035	0.0154 (0.0016)	1,305	0.0238 (0.0042)	0.0084	2.1214	0.0170
Block 9	7,819	0.0215 (0.0016)	1,712	0.0263 (0.0039)	0.0048	1.2167	0.1119
Block 10	25,832	0.0208 (0.0009)	9,707	0.0275 (0.0017)	0.0068	3.8196	0.0001
Block 11	11,096	0.0506 (0.0690)	5,071	0.0690 (0.0036)	0.0184	4.7005	0.0000
Block 12	1,217	0.0074 (0.0025)	453	0.0133 (0.0054)	0.0059	1.1263	0.1301

outcome. For example, in Block 5, comparing 68,400 unexposed and 7,447 hurricane-exposed pregnant women, we find that exposure to Hurricane Andrew more than doubled the risk of dysfunctional labor outcome ($\mu = 0.0213$ vs. 0.0436 , $t = 12.13$, $p \leq 0.01$). For this block of demographically matched pregnant women, Hurricane Andrew-related stress effects caused an estimated 166 additional cases of dystocia. Overall, we find that 4,813 of 256,144 of demographically matched unexposed pregnant women suffered a dysfunctional labor outcome, constituting 18.79 per 1,000 live births. For hurricane-exposed women, we find that 1,323 of 41,852 expectant mothers experienced an abnormal delivery, translating to a rate of 31.62 per 1,000 live births.⁴ If not for Hurricane Andrew, we estimate that 537 pregnant women would have been spared the experience of dystocia.

Table 5 shows measured differences between hurricane-exposed and unexposed pregnant women by stratification and nearest-neighbor matching algorithms. Both matching procedures generate near identical results. Our results show that hurricane-exposed pregnant mothers are significantly more likely to experience a dysfunctional pregnancy. The average treatment effect for treated population (ATT) or derived mean difference between *treatment* and *control* groups in the proportion of women experiencing dystocia is 0.01, constituting more than a 50% increase in dysfunctional labor risk for hurricane-exposed mothers. Interestingly, results also indicate that the mean difference between hurricane-exposed and unexposed expectant mothers amplifies in the third trimester of gestation, suggesting that the stress-inducing effects (or socially disrupting effects) of Hurricane Andrew may be most harmful as pregnancies approach full term.

⁴ Dysfunctional labor rates per 1,000 women for hurricane exposed and unexposed females are derived by: $((n_1 \times \mu_1 + n_2 \times \mu_2 + \dots + n_n \times \mu_n) / (n_1 + n_2 + \dots + n_n)) \times 1,000$, where n_n is the number of observations per block and μ_n is the proportion of females within each block experiencing dystocia. To derive the added number of dysfunctional labor cases attributable to maternal exposure to Hurricane Andrew we: $((n_1 \times \mu_{c1} + n_2 \times \mu_{c2} + \dots + n_n \times \mu_{cn}) / (n_1 + n_2 + \dots + n_n)) - ((n_1 \times \mu_{t1} + n_2 \times \mu_{t2} + \dots + n_n \times \mu_{tn}) / (n_1 + n_2 + \dots + n_n))$, where n_n is the number of hurricane exposed pregnant mothers per block and μ_{cn} is the proportion of unexposed (or control) females and μ_{tn} is the proportion of exposed (or treatment) females experiencing dystocia.

Table 5 ATT estimation of exposure on dysfunctional labor risk by stratification and nearest-neighbor matching

Period exposed	Exposed <i>N</i>	Unexposed <i>N</i>	ATT stratification*	<i>T</i>	Exposed <i>N</i>	Unexposed <i>N</i>	ATT nearest neighbor*	<i>T</i>
Exposed	41,631	255,225	0.010 (0.001)	11.208	41,852	256,144	0.010 (0.001)	11.365
First trimester	11,958	284,898	0.007 (0.002)	4.173	12,001	285,995	0.007 (0.002)	4.260
Second trimester	13,044	283,812	0.006 (0.002)	4.290	13,107	284,889	0.007 (0.002)	4.399
Third trimester	16,629	280,227	0.013 (0.001)	8.732	16,744	281,252	0.013 (0.001)	8.909

* Analytic standard errors in parentheses

Table 6 Independent samples *t* test of exposed versus unexposed pregnant women on primary C-section by block

Block (propensity score)	Control (C) group: hurricane unexposed		Treatment (T) group: hurricane exposed		$T_{\mu}-C_{\mu}$	<i>t</i> test	Pr ($T < C$)
	<i>N</i>	Mean (SE)	<i>N</i>	Mean (SE)			
Block 1	917	0.1592 (0.1091)	220	0.1091 (0.0211)	-0.0501	-1.8736	0.0306
Block 2	28,437	0.1332 (0.002)	1,897	0.1639 (0.0085)	0.0307	3.7923	0.0001
Block 3	56,017	0.1366 (0.0015)	4,381	0.1689 (0.0057)	0.0324	5.9632	0.0000
Block 4	6,685	0.1868 (0.0048)	626	0.2268 (0.0167)	0.0400	2.4389	0.0074
Block 5	67,946	0.1608 (0.0014)	7,433	0.1905 (0.0046)	0.0297	6.5746	0.0000
Block 6	33,137	0.1200 (0.0018)	6,759	0.1454 (0.0042)	0.0254	5.7671	0.0000
Block 7	10,002	0.1307 (0.0034)	2,264	0.1492 (0.0075)	0.0186	2.3480	0.0094
Block 8	6,003	0.1697 (0.0049)	1,302	0.2005 (0.0111)	0.03071	2.6435	0.0041
Block 9	7,741	0.1736 (0.0043)	1,711	0.1806 (0.0093)	0.0070	0.6873	0.2460
Block 10	25,793	0.1473 (0.0022)	9,699	0.1786 (0.0039)	0.0313	7.2377	0.0000
Block 11	11,053	0.1984 (0.0038)	5,063	0.2123 (0.0058)	0.0139	2.0398	0.0207
Block 12	1,216	0.1497 (0.0102)	452	0.1704 (0.0177)	0.0206	1.0364	0.1501

Given that dysfunctional labor is the major reason for non-elective cesarean section delivery, and that our results suggest that pregnant women experience higher dysfunctional labor risk with hurricane exposure, then we ought to observe a similar increase in the probability of cesarean section delivery. Table 6 reports independent samples *t* test results comparing hurricane-exposed and unexposed pregnant women on primary c-section outcome by propensity score blocks. In 9 of 12 blocks, we find statistically significant differences between control (unexposed) and treatment (exposed) groups (where, $p \leq 0.05$). For example, in Block 5, we observe that 10,926 of 67,946 ($\mu = 0.1608$) unexposed pregnant women had a c-section delivery, whereas 1,195 of 7,433 ($\mu = 0.1905$) hurricane-exposed pregnant mothers had a c-section delivery. For this block of propensity score-matched pregnant mothers, Hurricane Andrew added 221 c-sections that may not have otherwise happened. Overall, if Hurricane Andrew had not occurred, we estimate that approximately 1,083 c-sections may have been averted.

Table 7 ATT estimation of exposure and primary C-section risk by stratification and nearest-neighbor matching

Period exposed	Exposed <i>N</i>	Unexposed <i>N</i>	ATT stratification*	<i>T</i>	Exposed <i>N</i>	Unexposed <i>N</i>	ATT nearest neighbor*	<i>T</i>
Exposed	41,631	255,225	0.026 (0.002)	12.753	41,852	254,947	0.026 (0.002)	12.683
First trimester	11,958	284,898	0.017 (0.004)	4.682	12,001	284,765	0.017 (0.004)	4.679
Second trimester	13,044	283,812	0.027 (0.003)	7.889	13,107	283,660	0.027 (0.003)	7.850
Third trimester	16,629	280,227	0.024 (0.003)	7.937	16,744	280,030	0.024 (0.003)	7.873

* Analytic standard errors in parentheses

Table 7 shows c-section risk differences between hurricane-exposed (treatment) and unexposed (control) pregnant women by stratification and nearest-neighbor matching algorithms. Again, both matching procedures generate identical results. The mean difference between *treatment* and *control* groups on the proportion of pregnant women that undergo cesarean section delivery is 0.026, representing about a 20% increase in c-section risk for hurricane-exposed mothers. Again, we find that mean differences between exposed and unexposed pregnant mothers are higher for among second- and third-trimester exposed mothers.

5 Discussion and conclusion

Given that Hurricane Andrew caused atypically high levels of traumatic stress in the resident female population, we expected an increase in dystocia because of the known relationship between maternal stress and abnormal labor outcomes. We also reasoned that an increase in cesarean section delivery would follow from an observed spike in stress-induced dysfunctional labor outcomes, as c-section delivery is a common medical response to weak uterine contractility. Our results show that Hurricane Andrew did indeed increase the incidences of dysfunctional labor and c-section deliveries with approximately 530 cases of dystocia (about a 50% increase over normal risk) and about 1,000 cases of c-section delivery (about a 20% increase over normal risk).

Because maternal exposure to a catastrophic event during pregnancy appears to increase the risks of dysfunctional labor and higher-cost c-section deliveries, the value of adequate prenatal care is especially important during post-disaster situations. In supplemental analyses of birth data, we find a substantial increase in the number of hurricane-exposed pregnant mothers with *no* prenatal visits.

Data in Table 8 clearly show that women in hurricane-damaged areas were less likely to have access to adequate prenatal care after Andrew. Independent samples *t* test results comparing hurricane-exposed and unexposed pregnant women on zero prenatal visits outcome by propensity score blocks show significant differences in all but 1 block (where $p < 0.01$). Hurricane Andrew added an estimated 2,954 pregnant women to the ranks of women with zero prenatal visitation/care. While our initial intuition emphasized the biological effects of catastrophe-caused stress, the apparent rise in the number of pregnant women with no prenatal care before delivery suggests an important socio-contextual component for the observed increase in c-section deliveries. Because of the profound social

Table 8 Independent samples *t* test of exposed versus unexposed pregnant women on no prenatal visitation by block

Block (propensity score)	Control (<i>C</i>) group: hurricane unexposed		Treatment (<i>T</i>) group: hurricane exposed		$T_{\mu-C_{\mu}}$	<i>t</i> test	Pr (<i>T</i> < <i>C</i>)
	<i>N</i>	Mean (SE)	<i>N</i>	Mean (SE)			
Block 1	919	0.2459 (0.0142)	221	0.3258 (0.0316)	0.0799	2.4304	0.0076
Block 2	28,519	0.1467 (0.0021)	1,899	0.2391 (0.0098)	0.0923	10.8571	0.0000
Block 3	56,323	0.2507 (0.0018)	4,386	0.3821 (0.0073)	0.1315	19.1722	0.0000
Block 4	6,712	0.1992 (0.0049)	626	0.3275 (0.0188)	0.1283	7.5628	0.0000
Block 5	68,400	0.3716 (0.0019)	7,447	0.4827 (0.0058)	0.1111	18.7784	0.0000
Block 6	33,213	0.1605 (0.002)	6,761	0.2120 (0.005)	0.0594	11.8560	0.0000
Block 7	10,059	0.2135 (0.0041)	2,264	0.2708 (0.0093)	0.0572	5.9073	0.0000
Block 8	6,035	0.2085 (0.0052)	1,305	0.2452 (0.0119)	0.0368	2.9324	0.0017
Block 9	7,819	0.3086 (0.0052)	1,712	0.3505 (0.0115)	0.0419	3.3756	0.0004
Block 10	25,832	0.2263 (0.0026)	9,707	0.2649 (0.0045)	0.0386	7.6309	0.0000
Block 11	11,096	0.4198 (0.0047)	5,071	0.4784 (0.0070)	0.0585	6.9687	0.0000
Block 12	1,217	0.2284 (0.0120)	453	0.2450 (0.0103)	0.0166	0.7133	0.2379

disruption caused by Hurricane Andrew, higher numbers of pregnant women were likely separated from their usual source of care. With medical care professionals displaced and hospitals overextended and understaffed, obstetricians may be more likely to perform a c-section than go through a more lengthy standard delivery, particularly if the obstetrician is unfamiliar with the patient’s medical history. How women’s prenatal care histories and post-disaster health care conditions affect provider decisions regarding performing c-sections represents an important area worthy of additional research.

In addition, this study suggests the need for crucial practical interventions. Pre- and post-disaster public health initiatives should target pregnant women and their health care providers. These groups should be educated and informed regarding the attendant risks for pregnant women of living in a highly stressful post-disaster environment. Moreover, disaster preparedness and response efforts should aim to ensure access to prenatal care for mothers in highly disrupted hurricane-affected areas. This may mean providing free or reduced cost prenatal care to uninsured women and also assisting with continuity of services for those pregnant women who were in the care of a medical professional prior to the disaster. While limited resources often suggest zero-sum tradeoffs between similar policy investments in low hurricane risk locations, the unique context of hurricane zones indicates that policy concentrations in such areas in fact can provide higher returns on such social investments due to the associated higher risks of complex deliveries in these settings. Indeed, investments in prenatal care in these zones are likely to yield greater benefits to maternal and child health and lower eventual social costs by minimizing potential multiplicative risks.

Furthermore, prenatal investments in hurricane zones may yield high returns if targeted at struggling socioeconomic groups, who themselves may be clustered in the lowest cost, most hazardous, and highest risk areas of such zones. These mothers are likely to have the least access to prenatal care a priori, yet are just as likely to have stress-induced dystocia as others in hurricane-vulnerable regions. Doctors in disaster situations may be particularly likely to lean toward c-sections when faced with a pregnant woman with an unclear patient

history given that uncertainty and time constraints in such scenarios make the certainty of c-sections an understandable defensive-medicine posture. In that sense, the targeted pre-natal care of underprivileged mothers not only may mitigate more general pregnancy and infancy difficulties, but also may effectively reduce critical uncertainties for medical decisions in times of crisis.

References

- Alehagen S, Wijma K, Lundberg U, Melin B, Wijma B (2001) Catecholamine and cortisol reaction to childbirth. *Int J Behav Med* 8:50–65
- Anderson DK, Rhees RW, Fleming DE (1985) Effects of prenatal stress on differentiation of the sexually dimorphic nucleus of the preoptic area (SDN-POA) of the rat brain. *Brain Res* 332:113–118
- Austin MP, Leader L (2000) Maternal stress and obstetric and infant outcomes: epidemiological findings and neuroendocrine mechanisms. *Aust N Z J Obstet Gynaecol* 40:331–337
- Barker DJ, Osmond C (1986) Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet* 1:1077–1081
- Bennett T (2005) Women's health: after the fall. *Women's Health Issues* 15:237–239
- Callaghan W, Rasmussen S, Jamieson D, Ventura S, Farr SL, Sutton PD, Mathews TJ, Hamilton BE, Shealy KR, Brantley D, Posner S (2007) Health concerns of women and infants in times of natural disasters: lessons learned from Hurricane Katrina. *Matern Child Health* 11:307–311
- Chrousos GP (1997) Stressors, stress and the neuroendocrine integration of the adaptive response: the 1997 Hans Selye Memorial Lecture. *Ann N Y Acad Sci* 1998(851):311–335
- Crammond WA (1954) Psychological aspects of uterine dysfunction. *Lancet* 2:1241–1245
- Cutter SL, Boruff BJ, Shirley WL (2003) Social vulnerability to environmental hazards. *Soc Sci Q* 84:242–261
- Dehejia RH, Wahba S (2002) Propensity score-matching methods for nonexperimental causal studies. *Rev Econ Stat* 84:151–161
- Diego MA, Jones NA, Field T, Hernandez-Reif M, Schanberg S, Kuhn C, Gonzalez-Garcia A (2006) Maternal psychological distress, prenatal cortisol, and fetal weight. *Psychosom Med* 68:747–753
- Dole N, Savitz DA, Hertz-Picciotto I, Siega-Riz AM, McMahon MJ, Buekens P (2003) Maternal stress and preterm birth. *Am J Epidemiol* 157:14–24
- Enarson E, Morrow BH (1997) A gendered perspective: the voices of women. In: Peacock WG, Morrow BH, Gladwin H (eds) *Hurricane Andrew: ethnicity, gender, and the sociology of disaster*. Routledge, New York, pp 116–140
- Enarson E, Fothergill A, Peek L (2006) Gender and disaster: foundations and directions. In: Rodriguez H, Quarantelli EL, Dynes RR (eds) *Handbook of disaster research*. Springer, New York, pp 130–146
- Fothergill A (1996) Gender, risk, and disaster. *Int J Mass Emerg Disasters* 14:33–56
- Fothergill A (2004) Heads above water: gender, class, and family in the grand forks flood. State University of New York Press, Albany
- Friedman EA (1955) Primipregnant labor; a graphicostatistical analysis. *Obstet Gynecol* 6:567–589
- Gifford DS, Morton SC, Fiske M, Keeseey J, Keeler E, Kahn KL (2000) Lack of progress in labor as a reason for cesarean. *Obstet Gynecol* 95:589–595
- Girard C, Peacock WG (1997) Ethnicity and segregation: post-hurricane relocation. In: Peacock WG, Morrow BH, Gladwin H (eds) *Hurricane Andrew: ethnicity, Gender, and the Sociology of Disaster*. Routledge, New York, pp 191–205
- Gluckman, PD, Hanson MA (2004) Maternal constraint of fetal growth and its consequences. *Sem Fetal Neonatal Med* 9:419–425
- Gluckman PD, Hanson MA, Cooper C, Thornburg KL (2008) Effect of in utero and early-life conditions on adult health and disease. *New Engl J Med* 359:61–73
- Grenier GJ, Morrow BH (1997) Before the storm: the socio-political ecology of Miami. In: Peacock WG, Morrow BH, Gladwin H (eds) *Hurricane Andrew: ethnicity, gender, and the sociology of disaster*. Routledge, New York, pp 36–51
- Ironson G, Wynings C, Schneiderman N, Baum A, Rodriguez M, Greenwood D, Benight C, Antoni M, LaPerriere A, Huang H, Kimas N, Fletcher M (1997) Posttraumatic stress symptoms, intrusive thoughts, loss, and immune function after Hurricane Andrew. *Psychosom Med* 59:128–141

- Jones-DeWeever A (2008) Women in the wake of the storm: examining the post-Katrina realities of the women of New Orleans and the Gulf Coast. Institute for Women's Policy Research (IWPR) report briefing, part three. IWPR, Washington, DC
- Kapp TT, Hornstein S, Graham VT (1963) Some psychologic factors in prolonged labor due to inefficient uterine action. *Compr Psychiatry* 4:9–18
- King S, Mancini-Marie A, Brunet A, Walker E, Meaney MJ, Laplante DP (2009) Prenatal maternal stress from a natural disaster predicts dermatoglyphic asymmetry in humans. *Dev Psychopathol* 21:343–353
- Kuzawa C, Sweet E (2009) Epigenetics and the embodiment of race: developmental origins of US racial disparities in cardiovascular health. *Am J H Bio* 21:2–15
- Laplante DP, Barr RG, Brunet A, Galbaud Du Fort G, Meaney ML, Saucier J, Zelazo PR, King S (2004) Stress during pregnancy affects general intellectual and language functioning in human toddlers. *Pediatric Res* 56:400–410
- Lederman RP, McCann DS, Work B, Huber MJ (1977) Endogenous plasma epinephrine and norepinephrine in last-trimester pregnancy and labor. *Am J Obstet Gynecol* 129:5–8
- Lederman RP, Lederman E, Work BA, McCann DS (1978) The relationship of maternal anxiety, plasma catecholamines, and plasma cortisol to progress in labor. *Am J Obstet Gynecol* 132:495–500
- Lederman RP, Lederman E, Work BA, McCann DS (1979) Relationship of psychological factors in pregnancy to progress in labor. *Nurs Res* 28:94–97
- Lewis PD (1993) Governor's disaster planning and response review committee: final report, Florida. Diane Publishing Company, Darby
- Lowe NK (2007) A review of factors associated with dystocia and cesarean section in nulliparous women. *J Midwifery Women's Health* 52:216–228
- Maccari S, Darnaudery M, Morley-Fletcher S, Zuena AR, Cinque C, Van-Reeth O (2003) Prenatal stress and long-term consequences: implications of glucocorticoid hormones. *Neurosci Biobehav* 27:119–127
- McEwen BS (2001) From molecules to mind. Stress, individual differences, and the social environment. *Ann NY Acad Sci* 935:42–49
- Monga M, Sanborn BM (2004) Biology and physiology of the reproductive tract and control of myometrial contraction. In: Creasy RK, Resnik R (eds) *Maternal-fetal medicine*, 5th ed. Saunders, Philadelphia, pp 69–78
- Morishima HO, Yeh MN, James LS (1979) Reduced uterine blood flow and fetal hypoxemia with acute maternal stress: experimental observation in the pregnant baboon. *Am J Obstet Gynecol* 134(3):270–275
- Morrow BH (1997) Stretching the bonds: the families of Andrew. In: Peacock WG, Morrow BH, Gladwin H (eds) *Hurricane Andrew: ethnicity, gender, and the sociology of disaster*. Routledge, New York, pp 141–170
- Morrow BH, Enarson E (1996) Hurricane Andrew through Women's eyes: issues and recommendations. *Int J Mass Emerg Disasters* 14(1):1–22
- Mulder EJJ, Robles de Medina PG, Huizink AC, Van den Bergh BRH, Buitelaar JK, Visser GHA (2002) Prenatal maternal stress: effects on pregnancy and the (unborn) child. *Early Human Dev* 70:3–14
- Neumann ID, Johnstone HA, Hatzinger M, Liebsch G, Shipston M, Russell JA, Landgraf R, Douglas AJ (1998) Attenuated neuroendocrine responses to emotional and physical stressors in pregnant rats involve adenohipophysial changes. *J Physiol* 508:289–300
- Norris FH, Perilla JL, Riad JK, Kaniasty K, Lavizzo EA (1999) Stability and change in stress, resources, and psychological distress following natural disaster: findings from Hurricane Andrew. *Anxiety Stress Coping* 12:363–396
- Norris FH, Friedman MJ, Watson PJ, Byrne CM, Diaz E, Kaniasty K (2002) 60,000 disaster victims speak: part I. An empirical review of the empirical literature, 1981–2001. *Psychiatry* 65:207–239
- Oakes JM (2006) Commentary: advancing neighbourhood-effects research—selection, inferential support, and structural confounding. *Int J Epidemiol* 35:643–647
- Omer H (1986) Possible psychophysiological mechanisms in premature labor. *Psychosomatics* 27:580–584
- Paarberg KM, Vingerhoets AJ, Passchier J, Dekker GA, Van Geijn HP (1995) Psychosocial factors and pregnancy outcome: a review with emphasis on methodological issues. *J Psychosom Res* 39(5):563–595
- Peacock WG, Morrow BH, Gladwin H (eds) (1997) *Hurricane Andrew: ethnicity, gender, and the sociology of disaster*. Routledge, New York
- Pinto ML, Shetty PS (1995) Influence of exercise-induced maternal stress on fetal outcome in Wistar rats: inter-generational effects. *Br J Nutr* 73:645–653
- Quenby S, Pierce SJ, Brigham S, Wray S (2004) Dysfunctional labor and myometrial lactic acidosis. *Obstet Gynecol* 103:718–723

- Rhees RW, Fleming DE (1981) Effects of malnutrition, maternal stress, or ACTH injections during pregnancy on sexual behavior of male offspring. *Physiol Behav* 27:879–992
- Riad JK, Norris FH (1996) The influence of relocation on the environmental, social, and psychological stress experienced by disaster victims. *Environ Behav* 28(2):163–182
- Rondó PHC (2007) Maternal stress/distress and low birth weight, preterm birth and intrauterine growth restriction—A review. *Curr Women's Health Rev* 3:13–29
- Rosenbaum PR (2002) *Observational studies*, 2nd edn. Springer, New York
- Rosenbaum PR, Rubin DB (1983) The central role of the propensity score in observational studies for causal effects. *Biometrika* 41–55
- Rotton J, Dubitsky SS, Milov A, White SM, Clark MC (1996) Distress, elevated cortisol, cognitive deficits, and illness following a natural disaster. *Environ Psychol* 17(2):85–98
- Sjostrom K, Valentin L, Thelin T, Marsal K (1997) Maternal anxiety in late pregnancy and fetal hemodynamics. *Eur J Obstet Gynecol Reprod Biol* 74:149–155
- Teixeira JM, Fisk NM, Glover V (1999) Association between maternal anxiety in pregnancy and increased uterine artery resistance index: cohort based study. *Br Med J* 318:153–157
- Tobin-Gurley J, Peek L, Loomis J (2010) Displaced single mothers in the aftermath of Hurricane Katrina: resource needs and resource acquisition. *Int J Mass Emerg Disasters* 28(2):170–206
- Wadhwa PD, Dunkel-Schetter C, Chicz-De MA, Porto M, Sandman CA (1996) Prenatal psychosocial factors and the neuroendocrine axis in human pregnancy. *Psychosom Med* 58:432–446
- Wadhwa PD, Sandman CA, Chicz-De MA, Porto M (1997) Placental CRH modulates maternal pituitary adrenal function in human pregnancy. *Ann NY Acad Sci* 814:276–281
- Weinstock M (1997) Does prenatal stress impair coping and regulation of the hypothalamic–pituitary–adrenal axis? *Neurosci Biobehav* 21:1–10
- Weinstock M (2001) Alterations induced by gestational stress in brain morphology and behaviour of the offspring. *Prog Neurobiol* 65:427–451
- Weinstock M (2002) Can the behavioral abnormalities induced by gestational stress in rats be prevented or reversed? *Stress* 5:167–176
- Weinstock M (2005) The potential influence of maternal stress hormones on developmental and mental health of the offspring. *Brain Behav Immun* 19:296–308
- Weinstock M, Fride E, Hertzberg R (1988) Prenatal stress effects on functional development of the offspring. *Progr Brain Res* 73:319–331
- Weinstock M, Matlina E, Maor GI, Rosen H, McEwen BS (1992) Prenatal stress selectively alters the reactivity of the hypothalamic–pituitary adrenal system in the female rat. *Brain Res* 595:195–200
- Williams MT, Hennessy MB, Davis HN (1995) CRF administered to pregnant rats alters offspring behavior and morphology. *Pharmacol Biochem Behav* 52:161–167
- Williams MT, Davis HN, McCrear AE, Long SJ, Hennessy MB (1999) Changes in the hormonal concentrations of pregnant rats and their fetuses following multiple exposures to a stressor during the third trimester. *Neurotoxicol Teratol* 21:403–414
- Yehuda R, Bierer LM (2008) Transgenerational transmission of cortisol and PTSD risk. *Prog Brain Res* 167:121–135
- Yelvington KA (1997) Coping in a temporary way: the tent cities. In: Peacock WG, Morrow BH, Gladwin H (eds) *Hurricane Andrew: ethnicity, gender, and the sociology of disaster*. Routledge, New York, pp 171–190
- Zahran S, Brody SD, Peacock WG, Vedlitz A, Grover H (2008) Social vulnerability and the natural and built environment: a model of flood casualties in Texas. *Disasters* 32:537–560
- Zahran S, Snodgrass JG, Peek L, Weiler S (2010) Maternal hurricane exposure and fetal distress risk. *Risk Anal* 30:1590–1601
- Zuspan FP, Cibils LA, Pose A (1962) Myometrial and cardiovascular responses to alterations in plasma epinephrine and norepinephrine. *Am J Obstet Gynecol* 84:841–851