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Maternal Hurricane Exposure and Fetal Distress Risk

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Logistic regression and spatial analytic techniques are used to model fetal distress risk as a function of maternal exposure to Hurricane Andrew. First, monthly time series compare the proportion of infants born distressed in hurricane affected and unaffected areas. Second, resident births are analyzed in Miami-Dade and Broward counties, before, during, and after Hurricane Andrew. Third, resident births are analyzed in all Florida locales with 100,000 or more persons, comparing exposed and unexposed gravid females. Fourth, resident births are analyzed along Hurricane Andrew's path from southern Florida to northeast Mississippi. Results show that fetal distress risk increases significantly with maternal exposure to Hurricane Andrew in second and third trimesters, adjusting for known risk factors. Distress risk also correlates with the destructive path of Hurricane Andrew, with higher incidences of fetal distress found in areas of highest exposure intensity. Hurricane exposed African-American mothers were more likely to birth distressed infants. The policy implications of *in utero* costs of natural disaster exposure are discussed.

KEY WORDS: Fetal distress; Hurricane Andrew; maternal exposure; natural experiment

1. INTRODUCTION

Hurricane Andrew, the second most destructive hurricane in U.S. history, caused an estimated \$26.5 billion in property loss (\$40.2 billion in 2008 U.S. dollars), left 1.4 million residents in southern Florida without electricity, and rendered more than 180,000 persons homeless.(1) At least 31 people died as a consequence of the storm and hundreds more suffered serious injuries.(2) Hurricane Andrew resulted in food shortages, the closure of schools and businesses, racial discord at relief centers and temporary shelters, sharp spikes in regional unemployment and domestic violence crime, and strain on hospital facilities, policing, and emergency response agencies.(3)

Although the mental, and to a lesser extent physical, health consequences of Hurricane Andrew have been well documented, there is no research available on the relationship between maternal exposure to Hurricane Andrew and fetal distress risk. This represents an important gap in the literature, especially in light of recent research that suggests that prenatal exposure to natural disasters may negatively impact general intellectual and language development among toddlers(4) and could result in increased risk of schizophrenia,(5) among other deleterious outcomes.

In this article, we investigate the statistical relationship between maternal exposure to Hurricane Andrew and fetal distress risk. Our article is

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organized into four sections. First, we review literature detailing the relationship between natural disasters and human stress response, particularly for socially vulnerable populations. Second, we describe and integrate scientific literature showing links between maternal stress and negative fetal outcomes. Third, we present descriptive, GIS, and regression results. Fourth, we discuss the findings and consider the risk management policy implications of our research.

2. DISASTERS, VULNERABILITY, AND STRESS

Large-scale disasters affect many persons simultaneously and engender an array of stressors, including threat to one's own life and physical integrity, exposure to the dead and dying, bereavement, financial loss, property damage, displacement, and social and community hardship.(6) Disasters are associated with increased incidence of hypertension, diabetes, lymphoma, and leukemia; spontaneous abortion; arthritic flares; gastrointestinal disturbance; and other issues such as fatigue, confusion, memory lapses, and general sluggishness.(7) Persons harmed by a disaster event often show symptoms of anger, depression, and generalized distress,(8–10) and many exposed individuals evidence short-lived posttraumatic stress disorder (PTSD) or subclinical stress reactions that abate over the course of several months or longer.(11) In the most catastrophic events, which cause extraordinary destruction and disruption, populations may exhibit strikingly high levels of depression and posttraumatic stress for years after the disaster.(12)

In their review of the disaster mental health literature, Norris and colleagues(6) classify Hurricane Andrew as a "high-impact event" based on the "atypically high levels of impairment" that were observed among affected youth and adult populations. Between one and four months after Hurricane Andrew, Ironson and colleagues(13) gathered data from 180 subjects living in neighborhoods in Miami that were directly impacted by the disaster. One-third (33%) of the sample met criteria for PTSD, 44% fell in the high-impact range on the Impact of Events (IES) scale, and the sample scored significantly lower on several immune measures when compared to laboratory controls. In a follow-up study conducted nine to 12 months after Andrew, researchers found that the intensity of disruption related to the rebuilding process was significantly associated with elevated PTSD scores and other measures of chronic stress.(14) David and colleagues(15) recruited adults from neighborhoods most severely affected by Hurricane Andrew. They discovered that over half of the sample met criteria for a new-onset disorder, including PTSD (36%), major depression (30%), and other anxiety disorders (20%). A study of residents of southern Dade County, Florida, found that six months post-Andrew, 20–30% of adults in the area met criteria for PTSD and 33–45% were meaningfully depressed,(12) indicating that Hurricane Andrew had substantial and long-lasting effects on the population.

Hurricane Andrew had severe consequences for all affected communities. However, racial minorities and women were disproportionately impacted.(15–18) The differential vulnerability of these groups is explained by fewer available resources to anticipate, prepare for, cope with, respond to, and recover from disaster.(19–21)

Studies conducted after Hurricane Andrew demonstrated that African Americans were less likely to evacuate prior to landfall.(22) African Americans were also more likely to seek refuge in government-constructed "tent cities,"(23) to struggle with postdisaster unemployment, to report troubles with accessing public and private aid,(24) and to experience inequities in insurance settlements, which resulted in members of this population group residing in severely damaged homes for longer periods of time.(25,26) Six months after Hurricane Andrew, Perilla, Norris, and Lavizzo(18) interviewed 404 residents of heavily impacted neighborhoods in southern Florida. They found that ethnic groups differed strongly in the prevalence of PTSD, with Caucasians exhibiting the lowest rates of trauma (15%).

Ironson and colleagues(13) found that gender was a significant correlate of both PTSD and IES symptoms, with women reporting more traumatic stress symptoms than men. Riad and Norris(27) discovered that women who were relocated after Hurricane Andrew experienced elevated rates of depression in comparison to other groups. In Miami's Federal Emergency Management Agency trailer camps, Morrow and Enarson(28) observed that women were often isolated, were fearful for their personal safety, lacked mental and reproductive health services and reliable transportation, and were unable to access needed community services. Moreover, there was no consistent child care, elder care, or family respite care offered to support women with dependents in their efforts to repair homes, search for new housing, or access relief services.[29] The lack of social support, combined with ongoing disruption within the home, left many women vulnerable to chronic stress reactions.[27,30]

3. MATERNAL STRESS AND FETAL DISTRESS

Much of the direct evidence pointing to detrimental effects of maternal stress on birth outcomes comes from experiments in rodents and nonhuman primates.[31–45] Increasingly, retrospective and prospective studies with human subjects demonstrate similar links between mothers' stress and their newborns' health.[37–39,41,46,47] This includes physiological stresses such as maternal undernutrition or malnutrition,[48–50] as well as psychological and emotional strains linked to, for example, mothers' depression, anxiety, or trauma.[51–53] In both cases, a range of studies link increased frequency of preterm births, low birth weight, and other negative birth outcomes with mothers' levels of perceived stress during gestation,[51,52,54] while maternal social support, particularly from spouses or partners, is associated with reduced levels of circulating stress hormones and negative birth outcomes.[55–57]

Adding further evidence to these associations, a body of literature now documents connections between the stresses and inequalities associated with racial disparities and higher levels of circulating stress hormones and greater risks for negative birth outcomes.[50,58–63] The pathways linking maternal stress with fetal development and birth outcomes are not fully understood, and a constellation of interrelated mechanisms may be implicated.[37,41] Nevertheless, multiple lines of research support the involvement of alterations in neuroendocrine systems and levels of circulating hormones in pregnant mothers and their fetuses.[46,64] In addition, the perception of stress leads to physiologically aroused "fight or flight" states associated with the activation of both the sympathetic nervous system and the hypothalamus-pituitary-adrenal axis. These lead to the release of stress hormones such as epinephrine as well as glucocorticoids like cortisol, which themselves activate a number of physiological systems that prepare the body for action and allow it to respond to immediate environmental challenges. In the case of a mother, the activation of the stress response draws blood to peripheral systems like muscles and limbs and away from processes such as reproduction judged nonessential to immediate action and survival.[65–68] In fact, maternal stress has been shown to influence blood flow to the endometrial lining or placenta, thus drawing vital nutrients and oxygen away from developing fetuses.[69] Maternal vascular changes associated with the stress response could therefore explain low birth weights and also, of particular relevance to this article, other negative birth outcomes like fetal hypoxia and distress.

Similarly, recent research shows that maternal stress hormones like epinephrines and cortisol can pass across the placenta to the developing fetus.[37,41,46] Fetuses are normally shielded from maternal hormones by producing enzymes such as 11B-Hydroxysteroid Dehydrogenase (11-BHSD) that convert the active form of stress hormones (in this case, cortisol) to their inactive forms (cortisone), thus protecting themselves in normal circumstances from excessive maternal sympathetic and adrenal stress arousal.[41,50] However, in cases of excessive stress and resultant high levels of maternal cortisol, this buffering capacity may be exceeded, with infants unable to convert hormones such as cortisol to their inactive forms. [41,50] In these cases, high levels of circulating cortisol in the fetus itself can lead directly to a *fetal* stress response, drawing off fetal resources from less necessary processes like growth or setting off a cascade of reactions shortening gestation and triggering an early parturition.[50,66,70,71] Logically, such a situation might lead to excess fetal movement, oxygen consumption, and fatigue characteristic of fetal distress. Excessive excitation of the stress responses would lead eventually to a drop in fetal cortisol levels, which would compound fatigue and exhaustion.[72] Or, such chronic excitation of the fetal stress response could compromise the unborn child's immune system, leading to higher risks of infection, further fetal distress and exhaustion, and negative birth outcomes.[65,73–75][5]

[5] Of note, the activation of this fetal, as opposed to merely maternal, stress response might be precipitated by the movement of other noncortisol stress hormones from the mother into the intrauterine environment and fetus.[51,52,84–86] For example, maternal corticotrophin-releasing hormone or adrenocorticotropic hormone, precursors to the release of cortisol, might pass into the fetus and precipitate such a response. The neuroendocrinological pathways would be slightly different as compared to cases where maternal cortisol itself directly crossed into the intrauterine environment. Nevertheless, these hormones would activate the fetus's adrenal gland to increase circulating cortisol, thus leading to a fetal distress response through physiological changes, such as alterations in the circulation or consumption of nutrients and oxygen, similar to those described above.

4. RESEARCH DESIGN

Maternal exposure to Hurricane Andrew is derived from information on the timing and geography of the hurricane event, the estimated gestation length of the pregnancy, the estimated date of live birth, and maternal county residence as indicated in National Center for Health Statistics (NCHS) Vital Statistics Natality Birth Data, 1991–1997.(76) To illustrate, assume a child was born in the third week of September 1992 in Miami-Dade. Hurricane Andrew's landfall date was August 24, 1992. Because this child was born shortly after the event, we can assume that he/she was exposed in womb. The child's recorded gestational age at birth allows us to estimate the trimester period of exposure. Assume that the child's gestational age at birth was 30 weeks. Trimester period is then determined by subtracting the difference of landfall date and birth date from gestational age. For this child, we estimate that he/she was exposed to the hurricane event at approximately 26 weeks, or the third trimester of pregnancy. The same logic is used to code all birth events.

Our estimates of maternal exposure assume that the county of maternal residence is where a mother lived through the gestation period. To strengthen this assumption, we limit analysis to mothers with matched birth occurrence and residential FIPS codes. Although data do not permit the identification of gravid females who suffered personal property loss, displacement, or trauma, Hurricane Andrew was so destructive of routine life in Miami-Dade and Broward counties that one can reasonably assume generalized stress in the resident population with measurable psychosomatic effects.(13)

To investigate the relationship between maternal exposure to Hurricane Andrew and fetal distress we conduct four tests. First, we render monthly time-series showing the proportion of infants born distressed in Miami-Dade and Broward counties versus unaffected counties. Insofar as maternal exposure is related to fetal distress, we should observe a spike in the proportion of infants born distressed corresponding to the timing of Hurricane Andrew in affected but not unaffected areas.

Second, a binary logistic regression procedure is used to model fetal distress risk in Miami-Dade and Broward counties as a function of maternal exposure to Hurricane Andrew, adjusting for known risk factors of fetal distress. All birth events before, during, and after Hurricane Andrew are analyzed. We censor the analysis at 1997 to limit confounding effects of Hurricane Mitch, which made landfall in 1998 and impacted our study area.

Third, we recapitulate logistic regression models in step two with the addition of all birth events[6] in Florida over the same time period in counties with large populations (>100,000). Inclusion of birth events in unaffected areas imposes spatial control on exposure estimates in step two of the analysis.

Fourth, we analyze birth events along Hurricane Andrew's path from southern Florida through Louisiana and Mississippi. We estimate the destructive force of the hurricane along this path with property damage data from the Spatial Hazard Losses and Events Database for the United States (SHELDUS).(77) Insofar as fetal distress risk is statistically related to maternal exposure to Hurricane Andrew, the risk of fetal hypoxia should correlate with hurricane intensity, dissipating predictably as Hurricane Andrew shifts from a Category 5 hurricane in Florida to a tropical depression in Mississippi.[7]

4.1. Variable Operations

Fetal distress is measured as a binary outcome, with 1 = condition present, and 0 = condition not present. Fetal distress refers to a measurable deficiency in the amount of oxygen reaching fetal tissues. This condition of fetal hypoxia is present in approximately 2.6% of 1,097,409 observed births in Florida from 1991 to 1997. Variable operations are summarized in Table I, and descriptive statistics are presented in Table II. Demographic, health, and birth information are from the NCHS, Vital Statistics Natality Birth Data.(76)

We divide maternal hurricane exposure into trimesters (first = 0 to 12 weeks; second = 13–25 weeks; and third = 26+ weeks). Trimester exposure variables are measured as binary outcomes, with 1 = exposed, and 0 = not exposed. Exposure intensity is

[6] Birth events in select counties of the western Florida panhandle are excluded from the analysis to limit the contaminating effects of Hurricane Opal in 1995.

[7] SHELDUS data(77) are collected at the county scale. In SHELDUS, a county is defined as exposed if it incurred measureable dollar losses in property damage and crop loss. Variation in damage corresponds with the intensity of the hurricane track. For example, the amount of damage incurred in Miami-Dade, FL is greater than the amount of damage experienced in Jefferson Parish, LA, which experienced greater damage than Sumter, AL. We use SHELDUS damage figures along the hurricane track.

Table I. Operations for Predictors and Response Variables

Variable Label	Variable Definition
Maternal hypertension	An increase in blood pressure of at least 30 mmHg systolic or 15 mmHg diastolic on two measurements taken 6 hours apart after the 20th week of gestation. 1 = condition present, 0 = condition not present.
Maternal lung disease	Disease of the lungs during pregnancy. 1 = condition present, 0 = condition not present.
Maternal Rh sensitization	The process or state of becoming sensitized to the Rh factor as when an Rh-negative woman is pregnant with an Rh-positive fetus. 1 = condition present, 0 = condition not present.
Maternal diabetes	Disordered metabolism resulting in high blood sugar levels. 1 = condition present, 0 = condition not present.
Oligohydramnios	Any noticeable excess or lack of amniotic fluid. 1 = condition present, 0 = condition not present.
Abruptio placenta	Premature separation of a normally implanted placenta from the uterus. 1 = condition present, 0 = condition not present.
Infant birth weight	Infant weight in grams
Infant cord prolapsed	Premature expulsion of the umbilical cord in labor before the fetus is delivered. 1 = condition present, 0 = condition not present.
Maternal cigarette use	Average number of cigarettes smoked per day, 0–97 = number stated.
Maternal alcohol use	Average number of drinks per week, 0–97 = number stated.
African American	1 = African American, 0 = non African American.
Maternal age	Age of mother in single years, 11–54.
First trimester hurricane exposure	Maternal exposure to catastrophic hurricane event 0–12 weeks into gestation. 1 = condition present, 0 = condition not present.
Second trimester hurricane exposure	Maternal exposure to catastrophic hurricane event 13–25 weeks into gestation. 1 = condition present, 0 = condition not present.
Third trimester hurricane exposure	Maternal exposure to catastrophic hurricane event 26+ weeks into gestation. 1 = condition present, 0 = condition not present.
Exposure intensity	Total crop loss and property damage in US$ (2000), divided by 10 million.
Fetal distress	Signs indicating fetal hypoxia (deficiency in amount of oxygen reaching fetal tissues). 1 = condition present, 0 = condition not present.

Table II. Descriptive Statistics for Predictors and Response Variable

Variable Label	Mean	Standard Deviation	Min	Max
Maternal hypertension	0.031	0.172	0	1
Maternal lung disease	0.003	0.051	0	1
Maternal Rh sensitization	0.007	0.085	0	1
Maternal diabetes	0.028	0.165	0	1
Oligohydramnios	0.008	0.092	0	1
Abruptio placenta	0.004	0.066	0	1
Infant birth weight	3314.43	601.95	227	7,886
Infant cord prolapsed	0.002	0.045	0	1
Maternal cigarette use	1.49	4.73	0	97
Maternal alcohol use	0.029	0.567	0	97
African American	0.231	0.421	0	1
Maternal age	26.90	6.09	11	54
First trimester hurricane exposure	0.0110	0.104	0	1
Second trimester hurricane exposure	0.0119	0.109	0	1
Third trimester hurricane exposure	0.0144	0.119	0	1
Exposure intensity	36.68	24.47	0.00006	61.03
Fetal distress	0.0255	0.157	0	1

measured as the total crop loss and property damage (in year 2000 $US) suffered by a county, divided by $10 million. Dollar damage estimates are from SHELDUS.[77]

To adequately estimate the relationship of maternal hurricane exposure and fetal distress risk, we control for a series of known risk factors of fetal distress. From the NCHS, Vital Statistics Natality Birth Data,[76] we obtained information on maternal hypertension, maternal lung disease, maternal Rh sensitization, maternal diabetes, oligohydramnios, abruptio placenta, and infant cord prolapse. These variables are all measured as binary outcomes, with 1 = condition present, and 0 = condition not present or not reported. Infant birth weight is measured in grams, ranging from 227g to 7,886g among observed infants. We also control for maternal behavioral risks. Maternal cigarette use is measured continuously as the number of cigarettes smoked per day. Maternal alcohol use is estimated as the average number of drinks consumed per week. Finally, we measure two demographic characteristics. Maternal race is measured as 1 = African American

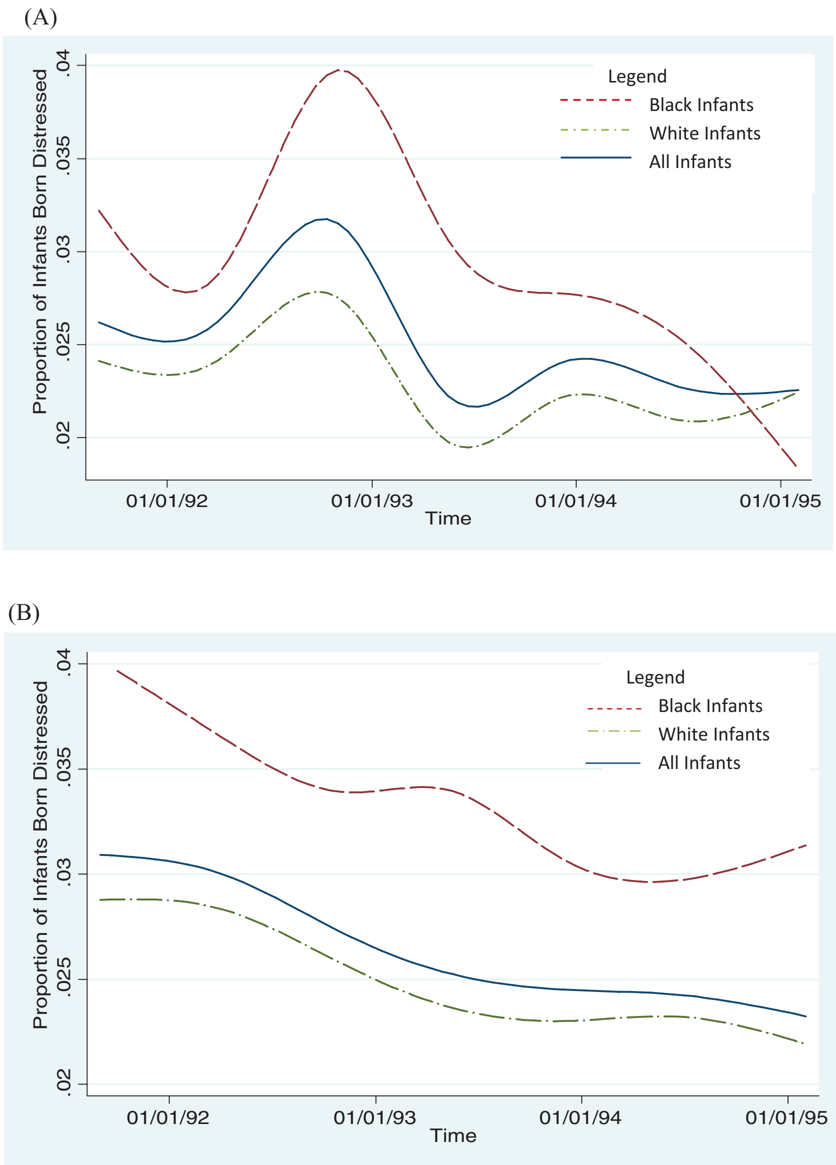

Fig. 1. (A) Monthly proportion of distressed infants in Miami-Dade and Broward counties. (B) Monthly proportion of distressed infants in unaffected Florida counties.

and 0 = non African American.[8] Maternal age is measured in single years, ranging from 11 to 54. We exclude birth events having incomplete information on study covariates.

[8] Two things motivated our inclusion of an African-American race code. The first, and more important theoretical motive, had to do with the known demographic epidemiology of fetal distress. Previous studies note differences between whites and blacks on fetal distress risk. The second, less important motive (but perhaps more interesting from a social science standpoint), had to do with the known facts of disproportionate loss experienced by African Americans during and following the hurricane event. Peacock *et al.*'s[(3)] text on Hurricane Andrew, the gold standard study in the field, provides ample evidence that African-American populations were less likely to evacuate, more likely to be injured, less likely to have adequate home insurance to cover losses, and more likely to be rendered homeless by the event, etc. Hispanic populations (mostly of Cuban origin), we learn from Peacock *et al.*,[(3)] were less likely to experience such traumas. In fact, the negative outcomes visited on Hispanic populations in Miami-Dade (with the exception of Hispanic migrant labor populations in the area) were statistically similar to negative outcomes experienced by white populations. We therefore focused our analytic energies on the African-American population.

Table III. Logistic Regression Odds Ratios Predicting Fetal Distress

	Fetal Distress Miami-Dade and Broward, 1991–1997 OR (95% CI)	Fetal Distress Florida Counties, 1991–1997 OR (95% CI)	Fetal Distress Hurricane Andrew Track, 1992–1993 OR (95% CI)
Maternal hypertension	1.80 (1.62 to 2.01)*** (0.100)	1.73 (1.65 to 1.83)*** (0.047)	1.83 (1.44 to 2.33)*** (0.225)
Maternal lung disease	3.15 (2.29 to 4.33)*** (0.511)	1.57 (1.30 to 1.89)*** (0.148)	1.60 (0.573 to 4.45) (0.839)
Maternal Rh sensitization	1.64 (1.17 to 2.30)*** (0.283)	1.37 (1.21 to 1.55)*** (0.087)	1.06 (0.432 to 2.60) (0.486)
Maternal diabetes	1.62 (1.44 to 1.82)*** (0.096)	1.43 (1.35 to 1.52)*** (0.045)	1.86 (1.45 to 2.37)*** (0.234)
Oligohydramnios	3.25 (2.70 to 3.89)*** (0.303)	2.85 (2.64 to 3.08)*** (0.112)	3.47 (2.16 to 5.57)*** (0.839)
Abruptio placenta	4.92 (4.17 to 5.80)*** (0.414)	4.67 (4.28 to 5.10)*** (0.207)	1.44 (1.29 to 1.61)*** (0.083)
Infant birth weight	0.99 (0.99 to 0.99)*** (0.00002)	0.99 (0.99 to 0.99)*** (9.56e-06)	0.99 (0.99 to 0.99)*** (0.00004)
Infant cord prolapsed	5.61 (4.45 to 7.07)*** (0.662)	6.52 (5.78 to 7.36)*** (0.403)	3.61 (2.17 to 6.00)*** (0.937)
Maternal cigarette use	1.001 (0.995 to 1.01) (0.003)	1.005 (1.003 to 1.008)*** (0.001)	1.009 (0.998 to 1.02) (0.006)
Maternal alcohol use	1.018 (0.994 to 1.007) (0.012)	1.014 (1.002 to 1.03)** (0.006)	1.006 (0.950 to 1.07) (0.029)
African American	1.15 (1.10 to 1.20)*** (0.027)	1.22 (1.19 to 1.26)*** (0.018)	1.40 (1.27 to 1.55)*** (0.071)
Maternal age	1.014 (1.01 to 1.02)*** (0.002)	1.014 (1.012 to 1.016)*** (0.001)	1.007 (0.999 to 1.015)* (0.004)
First trimester hurricane exposure	0.965 (0.858 to 1.09) (0.058)	0.977 (0.869 to 1.10) (0.063)	
Second trimester hurricane exposure	1.20 (1.08 to 1.33)*** (0.063)	1.21 (1.10 to 1.34)*** (0.062)	
Third trimester hurricane exposure	1.26 (1.15 to 1.38)*** (0.059)	1.29 (1.18 to 1.41)*** (0.060)	
Exposure intensity			1.011 (1.01 to 1.02)*** (0.02)
Log likelihood	−41,889.01	−127,516.70	−8,184.58
Wald X^2	1,548.46	6,655.34	330.04
N	352,462	1,097,409	59,056

Note: Robust standard errors in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

5. RESULTS

Fig. 1(A) is a time series of the proportion of infants born distressed in Miami-Dade and Broward counties. We observe noticeable spikes in the proportion of infants born distressed for all racial groups that correspond with the onset of Hurricane Andrew. The risk of fetal distress (for all infants) is higher during the hurricane exposure period of late August 1992 to May 1993 ($\mu = 0.029$, CI 95% = 0.026 to 0.032) as compared to nonexposure periods ($\mu = 0.024$, CI 95% = 0.023 to 0.025). Higher fetal distress risk in the hurricane exposure period is statistically significant ($t = -3.903$, $p \leq 0.001$). Fig. 1(B) is a time series of the proportion of infants born distressed in Florida counties outside the high-impact areas of Miami-Dade and Broward counties. In unaffected areas[9] of Florida, fetal distress risk is downward sloping through the hurricane event.

Next, we model fetal distress as a function of maternal exposure to Hurricane Andrew, adjusting for known risk factors. Table III shows odds ratios (OR) and 95% CIs for predictors of fetal distress.

[9] Birth events in Florida counties with populations (>100,000) included in NCHS, Vital Statistics Natality Birth Data[82] are: Alachua, Bay, Brevard, Charlotte, Clay, Collier, Duval, Hernando, Hillsborough, Lake, Lee, Leon, Manatee, Marion, Martin, Orange, Osceola, Palm Beach, Pasco, Pinellas, Polk, Saint Lucie, Sarasota, Seminole, and Volusia.

Fig. 2. Hurricane Andrew track with Saffir-Simpson scale of intensity.

Multivariate analyses were performed in Stata 9.2. Model 1 analyzes 352,462 fully observed birth events (84.5% of all births) in Miami-Dade and Broward counties from 1991 to 1997. Residents of Miami-Dade and Broward counties were hardest hit by Hurricane Andrew, as the bulk of property damage recorded for the event occurred in these counties.[78] All things held equal, first trimester exposure is statistically insignificant. Results show that maternal exposure to Hurricane Andrew in the second trimester increases the odds of fetal distress by a multiplicative factor of 1.20 (95% CI, 1.08–1.33). The risk of fetal distress increases by 26% (95% CI, 1.15–1.38) for third trimester exposed mothers. Combining coefficients, we find that third trimester exposed African-American mothers are 1.45 times more likely to birth a distressed infant than white unexposed mothers. Higher risk of birthing a distressed infant among hurricane exposed African-American mothers is corroborated by supplementary regression analyses appearing in the Appendix, Table A1.

Model 2 reports logistic regression results for 1,097,409 birth events in Florida counties (with 100,000+ residents) from 1991 to 1997. Whereas Model 1 exercises temporal control by analyzing birth events before, during, and after Hurricane Andrew, Model 2 adds geographic control by analyzing all births in unaffected Florida locales of 100,000+ persons over the same time period. As with Model 1, we find that second trimester maternal exposure to Hurricane Andrew increases the odds of fetal distress by a multiplicative factor of 1.21 (95% CI, 1.10 to 1.34). Similarly, the risk of fetal distress increases 0.29-fold for mothers exposed to this catastrophic event in the third trimester. Across Models 1 and 2, exposure coefficients are stable with results suggesting that the risk of fetal distress is higher among later-term exposed mothers.

Fig. 2 tracks the intensity of Hurricane Andrew as it intersects southern Florida as a Category 4/5 storm, bending northwest and striking coastal Louisiana as a Category 2/3, and ending as a tropical depression in northeast Mississippi. Model 3 analyzes resident birth events along this hurricane path of varying intensity and destruction. Results show that a \$10 million dollar increase in property damage increases the odds of fetal distress among hurricane exposed expectant mothers by a multiplicative factor of 1.011 (95% CI, 1.01 to 1.02). Similarly, a unit change of \$1 billion dollars of ruin produces nearly a two-fold increase in the risk of fetal distress (95% CI, 1.91 to 4.31). The effect size of \$1 billion dollars of hurricane damage rivals the effect size of known risk factors like maternal hypertension, maternal diabetes, and abruptio placenta.

6. DISCUSSION AND CONCLUSION

Economic assessments of natural disasters focus overwhelmingly on the explicit market-based costs of the events, particularly on business activity and property loss.[79] These evaluations often leverage analytical tools from regional economics, where traditional multiplier approaches to positive impacts of economic expansions are reversed to consider the cascading effects on local economies in the path of disasters such as Hurricane Andrew. Although one would generally expect such devastation to cause harm to local economic prospects, analysts have pointed to the fact that disasters may be superficially seen as increasing local output and income in the longer run, as short-run shutdowns of firms are quickly replaced by waves of incoming emergency response and recovery resources.[80]

In effect, commitments to rebuilding affected areas often lead to apparent longer-term economic growth, as insurance payments and recovery funding not only improve local economic indicators such as income and jobs but also can revive a region's entire economic base.[81] Yet this perspective is myopic in the sense that such studies are often limited in their analytical scope by failing to incorporate postdisaster liabilities, the improper modeling of systemic losses, and the failure to account for nonmarket losses.[82]

As the final point of this critique emphasizes, more traditional market-oriented considerations neglect some very real, yet relatively hidden, nonmarket costs to society, such as those suggested by the above analyses on the relationship between hurricane incidence and fetal distress. As indicated by the statistical results, fetuses from mothers in areas affected by Hurricane Andrew are considerably more likely to suffer distress, which in turn can lead to physical disadvantages that could require additional long-term monitoring and care. The psychological costs of the former can affect the mother's well-being (as well as earning potential, caregiving capabilities, familial relationships, etc.) for years. While such psychic costs can be difficult to quantify, they are nevertheless very real human costs, with none of the positive fringe benefits of rebuilding noted above. While some human costs may indeed be difficult to measure, others can be estimated to at least a rough order of magnitude by leveraging the key statistical results on health outcomes detailed above.

Additional health care costs to affected mothers and their children are particularly important examples. Incremental care costs for both mother and a newborn's first year can be estimated by examining the parallel implications of other afflictions that cause similar increases in hospital stays for mother and newborn. In particular, fetal distress can significantly raise the risk of abruptio placentia, where the placenta prematurely and abruptly separates from the uteran wall. While such occurrences are rare, they are also serious. Fetal distress raises the probability of such an event, adding hospitalization costs for both mother and infant.[83]

Fetal distress also has longer-term health care implications for affected children. Longer-term care of children disadvantaged from the outset by fetal distress and related hypoxia may require yet more resources that could have been dedicated to other needs. Such babies are also at greater long-term risk for various chronic health problems, such as asthma.

Potential policy implications can be drawn from the results. First, when designing disaster planning initiatives, emergency managers should include representatives from groups and agencies that focus on women's reproductive health. Women who are considering motherhood should be made aware of the increased risk of remaining in hurricane-prone areas during their pregnancies, in the same way they are cautioned about smoking, alcohol, and other risk factors. Evacuations of areas under imminent threat of hurricanes could specifically prioritize expectant mothers to minimize additional risks to fetuses. Relief centers should be organized to ensure that they meet women's needs for prenatal care. Also, those mothers who remain in affected areas during the recovery phase of disaster should be offered health monitoring for signs of fetal distress. Pregnant women should be encouraged and empowered to take remedial action when possible. Finally, as the results clearly show, disasters do not impact all women uniformly.[29] Because minority women are at heightened risk, the data suggest that education, evacuation, and fetal monitoring efforts can be targeted toward such vulnerable groups.

APPENDIX

Table A.1. Logistic Regression Odds Ratios Predicting Fetal Distress for African-American Population

	Fetal Distress Miami-Dade and Broward, 1991–1997 OR (95% CI)	Fetal Distress Florida Counties, 1991–1997 OR (95% CI)	Fetal Distress Hurricane Andrew Track, 1992–1993 OR (95% CI)
Maternal hypertension	2.16 (1.82 to 2.57)***	1.74 (1.57 to 1.92)***	1.79 (1.26 to 2.56)***
	(0.189)	(0.088)	(0.325)
Maternal lung disease	3.08 (1.65 to 5.75)***	1.39 (0.977 to 1.97)*	1.63 (0.355 to 7.48)
	(0.982)	(0.248)	(1.27)

(*Continued*)

Table A.1. (Continued)

	Fetal Distress Miami-Dade and Broward, 1991–1997 OR (95% CI)	Fetal Distress Florida Counties, 1991–1997 OR (95% CI)	Fetal Distress Hurricane Andrew Track, 1992–1993 OR (95% CI)
Maternal Rh sensitization	2.01 (0.941 to 4.29)* (0.777)	1.81 (1.29 to 2.54)*** (0.31)	0.982 (0.129 to 7.49) (1.01)
Maternal diabetes	1.93 (1.58 to 2.36)*** (0.198)	1.62 (1.44 to 1.82)*** (0.099)	2.13 (1.44 to 3.16)*** (0.428)
Oligohydramnios	4.04 (2.91 to 5.61)*** (0.676)	3.26 (2.83 to 3.74)*** (0.227)	2.55 (1.44 to 3.16)*** (0.428)
Abruptio placenta	5.00 (3.76 to 6.64)*** (0.726)	5.07 (4.31 to 5.95)*** (0.416)	1.48 (1.24 to 1.78)*** (0.138)
Infant birth weight	0.99 (0.99 to 0.99)*** (0.00003)	0.99 (0.99 to 0.99)*** (0.00002)	0.99 (0.99 to 0.99)*** (0.00006)
Infant cord prolapsed	6.33 (4.08 to9.84)*** (1.42)	5.50 (4.25 to 7.11)*** (0.723)	5.29 (2.43 to 11.54)*** (2.10)
Maternal cigarette use	0.999 (0.983 to 1.02) (0.009)	0.993 (0.984 to 1.001)* (0.004)	0.989 (0.966 to 1.10) (0.012)
Maternal alcohol use	1.01 (0.982 to 1.04) (0.015)	1.02 (0.996 to 1.037) (0.011)	1.03 (0.967 to 1.10) (0.033)
Maternal age	1.01 (1.01 to 1.02)*** (0.003)	1.016 (1.01 to 1.02)*** (0.002)	1.014 (1.00 to 1.025)** (0.006)
First trimester hurricane exposure	1.27 (1.05 to 1.53)*** (0.121)	1.19 (0.989 to 1.43)* (0.112)	
Second trimester hurricane exposure	1.35 (1.14 to 1.60)*** (0.117)	1.26 (1.07 to 1.49)*** (0.107)	
Third trimester hurricane exposure	1.46 (1.26 to 1.70)*** (0.114)	1.38 (1.19 to 1.60)*** (0.105)	
Exposure intensity			1.01 (1.01 to 1.02)*** (0.004)
Log likelihood	−13,477.95	−33,760.77	−3,334.54
Wald X^2	552.35	1,481.32	93.67
N	105,414	253,014	19,867

Note: Robust standard errors in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

REFERENCES

1. Final Report. Tallahassee, FL: Disaster Planning and Response Review Committee, 1993 Jan.
2. Bourque LB, Siegel JM, Kano M, Wood MM. Weathering the storm: The impact of hurricanes on physical and mental health. Annals of the American Academy of Political and Social Science, 2006; 604:129–151.
3. Peacock WG, Morrow BH, Gladwin H (eds). Hurricane Andrew: Ethnicity, Gender, and the Sociology of Disaster. New York: Routledge, 1997.
4. Laplante DP, Barr RG, Brunet A, Galbaud Du Fort G, Meaney ML, Saucier J, Zelazo PR, King S. Stress during pregnancy affects general intellectual and language functioning in human toddlers. Pediatric Research, 2004; 56:400–410.
5. King S, Mancini-Marie A, Brunet A, Walker E, Meaney MJ, Laplante DP. Prenatal maternal stress from a natural disaster predicts dermatoglyphic asymmetry in humans. Development and Psychopathology, 2009; 21:343–353.
6. Norris FH, Friedman MJ, Watson PJ, Byrne CM, Diaz E, Kaniasty K. 60,000 disaster victims speak: Part I. An empirical review of the empirical literature, 1981–2001. Psychiatry, 2002; 65:207–239.
7. Lutgendorf SK, Antoni MH, Ironson G, Fletcher MA, Penedo F, Baum A, Schneiderman N, Klimas N. Physical symptoms of chronic fatigue syndrome are exacerbated by the stress of Hurricane Andrew. Psychosomatic Medicine, 1995; 57:310–323.
8. Green BL, Solomon SD. The mental health impact of natural and technological disasters. Pp. 163–180 in Freedy JR, Hobfoll SE (eds). Traumatic Stress: From Theory to Practice. New York: Plenum Press, 1995.
9. Phifer JF, Norris FH. Psychological symptoms in older adults following natural disaster: Nature, timing, duration, and course. Journal of Gerontology, 1989; 44:207–212.
10. Smith EM, North CS. Posttraumatic stress disorder in natural disasters and technological accidents. Pp. 405–419 in Wilson JP, Raphael B (eds). International Handbook of Traumatic Stress Syndromes. New York: Plenum Press, 1993.
11. Bonanno GA. Loss, trauma, and human resilience: Have we underestimated the human capacity to thrive after extremely aversive events? American Psychologist, 2004; 59:20–28.
12. Norris FH, Perilla JL, Riad JK, Kaniasty K, Lavizzo EA. Stability and change in stress, resources, and psychological distress following natural disaster: Findings from Hurricane Andrew. Anxiety, Stress, and Coping, 1999; 12:363–396.
13. Ironson G, Wynings C, Schneiderman N, Baum A, Rodriguez M, Greenwood D, Benight C, Antoni M, LaPerriere A, Huang H, Kilmas N, Fletcher MA. Posttraumatic stress symptoms, intrusive thoughts, loss, and immune function after Hurricane Andrew. Psychosomatic Medicine, 1997; 59:128–141.

14. Burnett K, Ironson G, Benight C, Wynings C, Greenwood D, Carver CS, Cruess D, Baum A, Schneiderman N. Measurement of perceived disruption during rebuilding following Hurricane Andrew. Journal of Traumatic Stress, 1997; 10:673–680.
15. David D, Mellman TA, Mendoza LM, Kulick-Bell R, Ironson G, Schneiderman N. Psychiatric morbidity following Hurricane Andrew. International Journal for Traumatic Stress Studies, 1996; 9:607–612.
16. Breslau N, Davis GC, Andreski P, Peterson EL, Schultz LR. Sex differences in posttraumatic stress disorder. Archives of General Psychiatry, 1997; 54:1044–1048.
17. Breslau N, Davis GC, Peterson EL, Schultz LR. Psychiatric sequelae of posttraumatic stress disorder in women. Archives of General Psychiatry, 1997; 54:81–87.
18. Perilla JL, Norris FH, Lavizzo EA. Ethnicity, culture, and disaster response: Identifying and explaining ethnic differences in PTSD six months after Hurricane Andrew. Journal of Social and Clinical Psychology, 2002; 21(1):20–45.
19. Bolin B. Race, class, ethnicity, and disaster vulnerability. Pp. 113–129 in Rodriguez H, Quarantelli EL, Dynes RR, (eds). Handbook of Disaster Research. New York: Springer, 2006.
20. Enarson E, Fothergill A, Peek L. Gender and disaster: Foundations and directions. Pp. 130–146 in Rodriguez H, Quarantelli EL, Dynes RR, (eds). Handbook of Disaster Research. New York: Springer; 2006.
21. Wisner B, Blaikie P, Cannon T, Davis I. At Risk: Natural Hazards, People's Vulnerability, and Disaster, 2nd ed. London: Routledge, 2004.
22. Gladwin H, Peacock WG. Warning and evacuation: A night for hard houses. Pp. 52–74 in Peacock WG, Morrow BH, Gladwin H (eds). Hurricane Andrew: Ethnicity, Gender, and the Sociology of Disaster. New York: Routledge, 1997.
23. Yelvington KA. Coping in a temporary way: The tent cities. Pp. 92–115 in Peacock WG, Morrow BH, Gladwin H (eds). Hurricane Andrew: Ethnicity, Gender, and the Sociology of Disaster. New York: Routledge, 1997.
24. Morrow BH. Stretching the bonds: The families of Andrew. Pp. 141–170 in Peacock WG, Morrow BH, Gladwin H (eds). Hurricane Andrew: Ethnicity, Gender, and the Sociology of Disaster. New York: Routledge, 1997.
25. Girard C, Peacock WG. Ethnicity and segregation: Post-hurricane relocation. Pp. 191–205 in Peacock WG, Morrow BH, Gladwin H (eds). Hurricane Andrew: Ethnicity, Gender, and the Sociology of Disaster. New York: Routledge, 1997.
26. Peacock WG, Girard C. Ethnic and racial inequalities in hurricane damage and insurance settlements. Pp. 171–190 in Peacock WG, Morrow BH, Gladwin H (eds). Hurricane Andrew: Ethnicity, Gender, and the Sociology of Disaster. New York: Routledge, 1997.
27. Riad JK, Norris FH. The influence of relocation on the environmental, social, and psychological stress experienced by disaster victims. Environment and Behavior, 1996; 28:163–182.
28. Morrow BH, Enarson E. Hurricane Andrew through women's eyes: Issues and recommendations. International Journal of Mass Emergencies and Disaster, 1996; 14(1):1–22.
29. Enarson E, Morrow BH. A gendered perspective: The voices of women. Pp. 116–140 in Peacock WG, Morrow BH, Gladwin H (eds). Hurricane Andrew: Ethnicity, Gender, and the Sociology of Disaster. New York: Routledge, 1997.
30. Enarson E. Gender. Pp. 123–154 in Phillips BD, Thomas BDK, Fothergill A, Blinn-Pike L (eds). Social Vulnerability to Disasters. Boca Raton, FL: CRC Press, 2009.
31. Anderson DK, Rhees RW, Fleming DE. Effects of prenatal stress on differentiation of the sexually dimorphic nucleus of the preoptic area (SDN-POA) of the rat brain. Brain Research, 1985; 332:113–118.
32. Maccari S, Darnaudery M, Morley-Fletcher S, Zuena AR, Cinque C, Van-Reeth O. Prenatal stress and long-term consequences: Implications of glucocorticoid hormones. Neuroscience and Biobehavioral Reviews, 2003; Rev. 27:119–127.
33. Neumann ID, Johnstone HA, Hatzinger M, Liebsch G, Shipston M, Russell JA, Landgraf R, Douglas AJ. Attenuated neuroendocrine responses to emotional and physical stressors in pregnant rats involve adenohypophyseal changes. Journal of Physiology, 1998; 508:289–300.
34. Morishima HO, Yeh MN, James LS. Reduced uterine blood flow and fetal hypoxemia with acute maternal stress: Experimental observation in the pregnant baboon. American Journal of Obstetrics and Gynecology, 1979; 134(3):270–275.
35. Pinto ML, Shetty PS. Influence of exercise-induced maternal stress on fetal outcome in Wistar rats: Inter-generational effects. British Journal of Nutrition, 1995; 73:645–653.
36. Rhees RW, Fleming DE. Effects of malnutrition, maternal stress, or ACTH injections during pregnancy on sexual behavior of male offspring. Physiology and Behavior, 1981; 27:879–992.
37. Rondó PHC. Maternal stress/distress and low birth weight, preterm birth and intrauterine growth restriction—A review. Current Women's Health Reviews, 2007; 3:13–29.
38. Weinstock M. Does prenatal stress impair coping and regulation of the hypothalamic–pituitary–adrenal axis? Neuroscience and Biobehavioral Reviews, 1997; Rev. 21:1–10.
39. Weinstock M. Alterations induced by gestational stress in brain morphology and behaviour of the offspring. Progress in Neurobiology, 2001; 65:427–451.
40. Weinstock M. Can the behavioral abnormalities induced by gestational stress in rats be prevented or reversed? Stress, 2002; 5:167–176.
41. Weinstock M. The potential influence of maternal stress hormones on developmental and mental health of the offspring. Brain, Behavior, and Immunity, 2005; 19:296–308.
42. Weinstock M, Matlina E, Maor GI, Rosen H, McEwen BS. Prenatal stress selectively alters the reactivity of the hypothalamic–pituitary adrenal system in the female rat. Brain Research, 1992; 595:195–200.
43. Weinstock M, Fride E, Hertzberg R. Prenatal stress effects on functional development of the offspring. Progress in Brain Research, 1988; 73:319–331.
44. Williams MT, Hennessy MB, Davis HN. CRF administered to pregnant rats alters offspring behavior and morphology. Pharmacology Biochemistry and Behavior, 1995; 52:161–167.
45. Williams MT, Davis HN, McCrea AE, Long SJ, Hennessy MB. Changes in the hormonal concentrations of pregnant rats and their fetuses following multiple exposures to a stressor during the third trimester. Neurotoxicology and Teratology, 1999; 21:403–414.
46. Diego MA, Jones NA, Field T, Hernandez-Reif M, Schanberg S, Kuhn C, Gonzalez-Garcia A. Maternal psychological distress, prenatal cortisol, and fetal weight. Psychosomatic Medicine, 2006; 68:747–753.
47. Mulder EJH, Robles de Medina PG, Huizink AC, Van Den Bergh BRH, Buitelaar JK, Visser GHA. Prenatal maternal stress: Effects on pregnancy and the (unborn) child. Early Human Development, 2002; 70:3–14.
48. Barker DJ, Osmond C. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. Lancet, 1986; 1:1077–1081.
49. Gluckman PD, Hanson MA, Cooper C, Thornburg KL. Effect of in utero and early-life conditions on adult health and disease. New England Journal of Medicine, 2008; 359:61–73.
50. Kuzawa C, Sweet E. Epigenetics and the embodiment of race: Developmental origins of US racial disparities in cardiovascular health. American Journal of Human Biology, 2009; 21:2–15.
51. Wadhwa PD, Dunkel-Schetter C, Chicz-De MA, Porto M, Sandman CA. Prenatal psychosocial factors and the

neuroendocrine axis in human pregnancy. Psychosomatic Medicine, 1996; 58(5):432–446.
52. Wadhwa PD, Sandman CA, Chicz-De MA, Porto M. Placental CRH modulates maternal pituitary adrenal function in human pregnancy. Annals of New York Academy of Sciences, 1997; 814:276–281.
53. Yehuda R, Bierer LM. Transgenerational transmission of cortisol and PTSD risk. Progress in Brain Research, 2008; 167:121–135.
54. Dole N, Savitz DA, Hertz-Picciotto I, Siega-Riz AM, McMahon MJ, Buekens P. Maternal stress and preterm birth. American Journal of Epidemiology, 2003; 157:14–24.
55. Feldman PJ, Dunkel-Schetter C, Sandman CA, Wadhwa PD. Maternal social support predicts birth weight and fetal growth in human pregnancy. Psychosomatic Medicine, 2000; 62:715–725.
56. Hodnett ED, Fredericks S. Support during pregnancy for women at increased risk of low birthweight babies. Cochrane Database of Systematic Reviews, 2003; 3.
57. Oakley A, Rajan L, Grant A. Social support and pregnancy outcome. British Journal of Obstetrics and Gynecology, 1990; 97:155–162.
58. Borders AE, Grobman WA, Amsden LB, Holl JL. Chronic stress and low birth weight neonates in a low-income population of women. Obstetrics and Gynecology, 2007; 109(2, Part 1):331–338.
59. Dole N, Savitz DA, Siega-Riz AM, Hertz-Picciotto I, McMahon MJ, Buekens P. Psychosocial factors and preterm birth among African American and white women in central North Carolina. American Journal of Public Health, 2004; 94:1358–1365.
60. Dominguez TP, Schetter CD, Mancuso R, Rini CM, Hobel C. Stress in African American pregnancies: Testing the roles of various stress concepts in prediction of birth outcomes. Annals of Behavioral Medicine, 2005; 29:12–21.
61. Giscombe CL, Lobel M. Explaining disproportionately high rates of adverse birth outcomes among African Americans: The impact of stress, racism, and related factors in pregnancy. Psychology Bulletin, 2005; 131:662–683.
62. Mustillo S, Krieger N, Gunderson EP, Sidney S, McCreath H, Kiefe CI. Self-reported experiences of racial discrimination and black-white differences in preterm and low-birthweight deliveries: The CARDIA Study. American Journal of Public Health, 2004; 94:2125–2131.
63. Pike IL. Maternal stress and fetal responses: Evolutionary perspectives on preterm delivery. American Journal of Human Biology, 2005; 17:55–65.
64. McEwen BS. From molecules to mind. Stress, individual differences, and the social environment. Annals of New York Academy of Sciences, 2001; 935:42–49.
65. Chrousos GP. Stressors, stress and the neuroendocrine integration of the adaptive response: The 1997 Hans Selye memorial lecture. Annals of New York Academy of Sciences, 1998; 851:311–335.
66. Omer H. Possible psychophysiologic mechanisms in premature labor. Psychosomatics, 1986; 27:580–584.
67. Sjostrom K, Valentin L, Thelin T, Marsal K. Maternal anxiety in late pregnancy and fetal hemodynamics. European Journal of Obstetrics and Gynecology and Reproductive Biology, 1997; 74:149–155.
68. Teixeira JM, Fisk NM, Glover V. Association between maternal anxiety in pregnancy and increased uterine artery resistance index: Cohort based study. British Medical Journal, 1999; 318:153–157.
69. Gluckman PD, Hanson MA. Maternal constraint of fetal growth and its consequences. Seminars in Fetal and Neonatal Medicine, 2004; 9:419–425.
70. Challis JR, Bloomfield FH, Bocking AD, Casciani V, Chisaka H, Connor K, Dong X, Gluckman P, Harding JE, Johnstone J, Li W, Lye S, Okamura K, Premyslova M. Fetal signals and parturition. Journal of Obstetrics and Gynaecology Research, 2005; 31:492–499.
71. Sandman CA, Wadhwa PD, Chicz-DeMet A, Dunkel-Schetter C, Porto M. Maternal stress, HPA activity, and fetal/infant outcome. Annals of New York Academy of Sciences, 1997; 24:266–275.
72. Adam EK, Klimes-Dougan B, Gunnar M. Social regulation of stress physiology in infancy, childhood and adulthood: Implications for psychopathology and education. In Coch D, Dawson G, Fischer K (eds). Human Behavior, Learning, and the Developing Brain: Atypical Development. New York: Guilford Press, 2007.
73. De Souza EB. Corticotropin-releasing factor and interleukin-1 receptors in the brain-endocrine-immune axis. Role in stress response and infection. Annals of the New York Academy of Sciences, 1993; 697:9–27.
74. Giles WB, McLean M, Davies JJ, Smith R. Abnormal umbilical artery Doppler waveforms and cord blood corticotrophin-releasing hormone. Obstetrical and Gynecological Survey, 1996; 87:107–111.
75. McEwen BS, Biron CA, Brunson KW. The role of adrenocorticoids as modulators of immune function in health and disease: Neural, endocrine and immune interactions. Brain Research, 1997; Rev 23:79–133.
76. National Center for Health Statistics. Vital Statistics Birth Data, 1991–1997.
77. Spatial Hazard Losses and Events Database for the United States, 2009. Available at: http://webra.cas.sc.edu/hvri/products/sheldus.aspx, Accessed June 11, 2010.
78. Peacock WG, Morrow BH, Gladwin H (eds). Hurricane Andrew: Ethnicity, Gender, and the Sociology of Disaster. New York: Routledge, 1997.
79. Rose A, Benavides J, Chang SE, Szczesniak P, Lim D. The regional economic impact of an earthquake: Direct and indirect effects of electricity lifeline disruptions. Journal of Regional Science, 1997; 37(3):437–458.
80. West C, Lenze D. Modeling the regional impact of natural disasters and recovery: A general framework and an application to Hurricane Andrew. International Regional Science Review, 1994; 17(2):121–150.
81. Bennett D. Do natural disasters stimulate economic growth? New York Times, July 8, 2008.
82. Cochrane H. Economic loss: Myth and measurement. Disaster Prevention and Management, 2004; 13(4):290–296.
83. Adams EK, Melvin C. Costs of maternal conditions attributable to smoking during pregnancy. American Journal of Preventive Medicine, 1998; 15(3):212–219.
84. Challis JRG. Mechanism of parturition and preterm labor. Obstetrical and Gynecological Survey, 2000; 10:650–660.
85. McLean M, Bisits A, Davies J, Woods R, Lowry P, Smith R. A placental clock controlling the length of human pregnancy. Nat Med, 1995; 1:460–463.
86. Quartero HW, Noort WA, Fry CH, Keirse MJ. Role of prostaglandins and leukotrienes in the synergistic effect of oxytocin and corticotrophin-releasing hormone (CRH) on the contraction force in human gestational myometrium. Prostaglandins, 1991; 42:137–150.