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Regular Article

Stress in pregnancy: Clinical and adaptive behavior of offspring following Superstorm Sandy

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Abstract

The current study investigated 304 children from a longitudinal project (the Stress in Pregnancy (SIP) Study) who were exposed and unexposed to Superstorm Sandy ("Sandy") *in utero*. They were prospectively followed from 2 to 6 years of age and their clinical and adaptive behaviors were assessed annually. Using a hierarchical linear model, the study found that *in utero* Sandy exposure was associated with greater clinical (anxiety, depression, and somatization) and lower adaptive behaviors (social skills and functional communication) at age 2 years. However, the trajectories were notably different between the two groups. Anxiety increased more rapidly among the exposed than unexposed group at ages 2–4, and depression increased only among the exposed. In contrast, social skills and functional communication were lower in exposed compared to unexposed children at age 2, but quickly increased and exceeded the capacities of unexposed children by age 3. The findings confirm that prenatal Sandy exposure is not only associated with an increase in anxiety, depression, and somatization in offspring, but also with greater adaptive skills as the children got older. Our study demonstrates that while children who have experienced stress *in utero* demonstrate elevated suboptimal clinical behaviors related to affective disorders, they nevertheless have the potential to learn adaptive skills.

Keywords: clinical and adaptive behaviors, natural disaster, objective stress exposure, prenatal maternal stress

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Many aspects of an individual's behaviors and health outcomes across the life span can be traced back to the *in utero* period, when rapid changes occur in the structure and function of the fetus's brain and body (Barker, 2002; Glover, 2011; Zhang et al., 2018). There is growing evidence of critical periods during pregnancy when a fetus is particularly vulnerable to adverse maternal experiences such as stress (DiPietro, Hodgson, Costigan, & Johnson, 1996; Wadhwa, Sandman, & Garite, 2001). Prenatal maternal stress induces changes in the fetal immune and inflammatory responses and alters the hypothalamic–pituitary–adrenal (HPA) axis functioning. The biological impact of prenatal stress can include: dysregulation of the HPA axis; broad alterations in brain growth, size, or density; alterations within specific brain regions or white matter abnormalities; and hypomyelination in multiple brain regions (Buss, Davis, Muftuler, Head, & Sandman, 2010; Li et al., 2012; Lou et al., 1994; Lupien, McEwen, Gunnar, & Heim, 2009; Qiu et al., 2013). These alterations can also be associated with long-term neurodevelopmental, health, and behavioral consequences (Buitelaar, Huizink, Mulder, de Medina, & Visser, 2003; Talge, Neal, & Glover, 2007; van den Bergh, van Calster, Smits, van Huffel, & Lagae, 2008).

Prior preclinical research has demonstrated that the impact of prenatal stress on neuromotor, behavioral, and hormonal development (Hamada & Matthews, 2019; Jafari, Mehla, Kolb, & Mohajerani, 2017) is a function of the intensity, duration, and frequency of the exposure (Fujioka et al., 2001). Human studies have replicated some of the associations between prenatal stress and cognitive neurobehavioral functioning deficits observed in animal models (Gartstein, Putnam, & Rothbart, 2012; Lin et al., 2017; Neumann et al., 2019). However, notable limitations of these human studies, as compared to more intensive events such as exposure to natural disasters, are that the measures of prenatal stress are typically broad, lack negative valence, and draw on general stressful events from everyday life (Li et al., 2012). Consistent with prior research from the 2008 Ice storm (Cao, Laplante, Brunet, Ciampi, & King, 2012), we observed that prenatal exposure to a natural disaster results in protracted stress related to infant temperament indicative of internalizing problems (Nomura et al., 2019; Zhang et al., 2018). Thus, *in utero* perturbations can persist to affect the phenotype of the offspring, resulting in a long-lasting risk for dysregulation and illness across the life cycle (Barker, 2002). To date, several studies, including our own, have documented significant associations between maternal prenatal stress and temperament characteristics, including irritability, crying, fearfulness, distractibility, and poor adaptation in

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early childhood (Bergman et al., 2007; Brand, Engel, Canfield, & Yehuda, 2006; Davis, Snidman, Wadhwa, Glynn, Schetter, et al., 2004; Milgrom, Westley, & McCloud, 1995; Nolvi et al., 2016; Nomura et al., 2019; van der Wal, van Eijsden, & Bonsel, 2007; Zhang et al., 2018).

While the adverse effect of prenatal stress on programming for the postnatal environment is clear, a more nuanced concept – the stress inoculation theory (Edge et al., 2009) – has proposed that exposure to manageable and moderate stress can enhance the offspring's ability to manage subsequent stress and increase adaptive skills. Studies, including our own, on whether temperament alterations occur in response to prenatal stress have suggested adaptability of certain temperament characteristics in infancy (Zhang et al., 2018). As such, to understand the role of prenatal stress on child neurobehavioral development, it is important to examine both clinical and adaptative aspects in relation to the exposure.

Utilizing a quasi-experiment such a natural disaster provides the possibility for a longitudinal examination of the extent to which a specific prenatal stress influences subsequent child behavioral and developmental trajectories that can help to enhance understanding of the long-term impact of prenatal stress (Blake, Kimberlain, Berg, Cangialosi, & Beven, 2013). To do that, we leveraged Superstorm Sandy ("Sandy"), a horrendous natural disaster in the northeast United States, which resulted in severe financial and personal damage to the affected area, including 117 deaths (CDCP, 2013). We examined the longitudinal connections between Sandy exposure *in utero* and child behaviors (clinical and adaptive) during early childhood. We hypothesized that children who were exposed to Sandy *in utero*, as compared to unexposed children, would have higher levels of clinical behaviors (especially anxiety). We also hypothesized that prenatally-exposed children would have greater levels of adaptive behaviors (daily living functioning, functional communication, and social skills) than unexposed children.

Method

Participants

The current study included mothers and their offspring from a longitudinal project (the Stress in Pregnancy (SIP) study) investigating the effects of Sandy exposure during pregnancy (Finik & Nomura, 2017). Pregnant women were recruited from the prenatal obstetrics and gynecological clinics at Mount Sinai Medical Center and NewYork-Presbyterian/Queens in New York City during their second trimester. They were followed throughout their pregnancy in the first phase of the study (SIP Study Phase I). During the second phase of the study (SIP Study Phase II), they completed questionnaires about their child's development annually, at 2, 3, 4, 5, and 6 years of age. Children rejoined the second phase of the study at different times ($N = 358$). Children who had at least two assessments of behavior outcomes were included, giving a subsample of 322 mother–child dyads. Of those 322, an additional 18 were excluded from the analysis due to their prenatal exposure to cocaine, leaving a final sample of 304 children. Exclusion criteria for participation included HIV infection, maternal psychosis, maternal age <15 years, life-threatening maternal medical complications, and congenital or chromosomal abnormalities in the fetus. Further details of the study can be found elsewhere (Finik & Nomura, 2017). All participants provided written consent, according to the protocol approved by the Institutional Review Boards at the City University of New York, NewYork-Presbyterian/Queens, and the Icahn School of Medicine at Mount Sinai.

Among the 304 participants, child behavioral (clinical and adaptive scores) data were collected for 184 children at 2 years of age, 164 at 3 years, 195 at 4 years, 172 at 5 years, and 170 at 6 years. Participants had three assessments on average. Major demographic characteristics between those who were included ($N = 304$) and those who were excluded ($N = 54$) in this study did not differ significantly; that is, child sex ($p = .68$), race ($p = .52$), ethnicity ($p = .09$), marital status ($p = .52$), and maternal age ($p = .99$). Demographic information, including maternal education, marital status, race, and age reported by participants during the second trimester is presented in Table 1. There were no major demographic differences between those who were exposed and unexposed to Sandy *in utero*, including sex of child ($p = .17$), race of the child ($p = .08$), ethnicity of the child ($p = .07$), maternal age ($p = .51$), marital status ($p = .11$), and maternal education ($p = .18$). In addition, maternal psychological states (depression and anxiety) were measured by self-report questionnaires and substance use was ascertained through a face-to-face interview by clinical social workers during the second trimester. There were no differences in reported maternal anxiety ($p = .71$), depression ($p = .32$), cannabis use ($p = .74$) or tobacco use ($p = .74$) during pregnancy between those who were exposed and unexposed to Sandy *in utero*. Mothers who experienced Sandy during pregnancy, as compared to mothers who did not, consumed alcoholic beverages more often ($p = .04$, 12.0% vs. 5.2%).

Measures

Prenatal Sandy exposure

Sandy exposure status was defined as whether mothers were pregnant ($N = 92$, or 30.3%) or not pregnant ($N = 212$, or 69.7%) during Sandy. Children in this study, categorized in two groups (Figure 1), were born between January 2011 and October 2014. Mothers of exposed children were pregnant at the time when Sandy made landfall, whereas unexposed children were either born prior to or after the storm (Finik & Nomura, 2017).

Child behaviors

The Behavior Assessment System for Children – Second Edition, Parent Rating (BASC2-P) (Reynolds & Kamphaus, 2004), a well-standardized, multidimensional evaluation of the behavior of young children, was used to measure clinical and adaptive dimensions of behaviors. Clinical scales used included measures of hyperactivity, aggression, anxiety, depression, somatization, atypical behaviors, withdrawn behaviors, and attention problems. Adaptive scales included adaptive skills, social skills, activity of daily living, and functional communication. In this study, we used standardized *t*-scores, with 50 being the mean and 10 being the standard deviation (*SD*) that the BASC2-P system produces, which was normalized for sex and age for the category. A score of 60 or above on clinical scales and a score of 40 or less on adaptative scales are considered "at-risk" (Reynolds & Kamphaus, 2004).

Covariates

Severity of objective stress exposures to Sandy

Objective stress exposures specific to Sandy were assessed using Storm32 (Yong et al., 2015). Storm32 comprises 20 questions that encompass salient aspects of the disaster exposure objectively with four scales (scope of trauma, loss, threat, and change). The

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Table 1. Characteristics of participants by status of prenatal exposure to Superstorm Sandy ($N = 304$)

		Prenatal Sandy exposure		
Demographic characteristics		Not exposed ($n = 212$)	Exposed ($n = 92$)	Statistics, p value
Child's sex	Female, *N* (%)	97(45.8)	53 (57.6)	χ^2 (1) = 3.60, $p = .06$
Maternal age	Mean (*SD*)	28.06 (6.12)	27.55 (5.78)	$F = .44$, $p = .51$
Maternal prenatal anxiety trait	Mean (*SD*)	39.04 (10.98)	39.57 (9.56)	$F = .15$, $p = .71$
Maternal depression symptoms	Mean (*SD*)	9.68 (5.04)	9.05 (4.64)	$F = 1.01$, $p = .32$
Mother's education	Primary school, *N* (%)	7 (3.3)	2 (2.2)	
	Some high school, *N* (%)	28 (13.3)	5 (5.4)	
	High school/GED, *N* (%)	42 (19.9)	17 (18.9)	
	Some college, *N* (%)	50 (23.7)	21 (22.8)	
	Associate degree, *N* (%)	22 (10.4)	8 (8.7)	
	Bachelor's degree, *N* (%)	36 (17.1)	18 (19.6)	
	Graduate degree, *N* (%)	26 (12.3)	21 (22.8)	χ^2 (6) = 8.96, $p = .18$
Marital status	Married, *N* (%)	88 (41.5)	50 (54.4)	
	Common law, *N* (%)	9 (4.2)	6 (6.5)	
	Single, *N* (%)	111 (52.4)	35 (38.0)	
	Divorced/separated, *N* (%)	4 (1.9)	1 (1.1)	χ^2 (3) = 5.99, $p = .11$
Child's race	White, *N* (%)	73 (34.4)	46 (50.0)	
	Black, *N* (%)	105 (49.5)	33 (35.9)	
	Asian, *N* (%)	21 (9.9)	8 (8.7)	
	Other, *N* (%)	13 (6.1)	5 (5.4)	χ^2 (3) = 6.76, $p = .08$
Child's ethnicity	Hispanic, *N* (%)	43 (25.6)	49 (36.0)	χ^2 (1) = 3.40, $p = .07$
Socioeconomic status (SES)[a]	High, *N* (%)	41 (19.3)	26 (28.3)	χ^2 (2) = 3.31, $p = .19$
	Medium, *N* (%)	72 (34.0)	25 (27.2)	
	Low, *N* (%)	99 (46.7)	41 (30.3)	
Prenatal tobacco use	Yes, *N* (%)	23 (11.1)	9 (9.8)	χ^2 (1) = 0.11, $p = .74$
Prenatal cannabis use	Yes, *N* (%)	16 (7.6)	6 (6.5)	χ^2 (1) = 0.11, $p = .74$
Prenatal alcohol use	Yes, *N* (%)	11 (5.2)	11 (12.0)	χ^2 (1) = 4.38, $p = .04$

NB: Demographics information was ascertained based on self-reports and prenatal substance use status was ascertained based on face-to-face interview with a clinical social worker during pregnancy.
[a]SES was extracted based on education, work status (full-time, part-time, and not working), work prestige index for the pre-pregnancy job, and welfare status, using a latent class analysis.

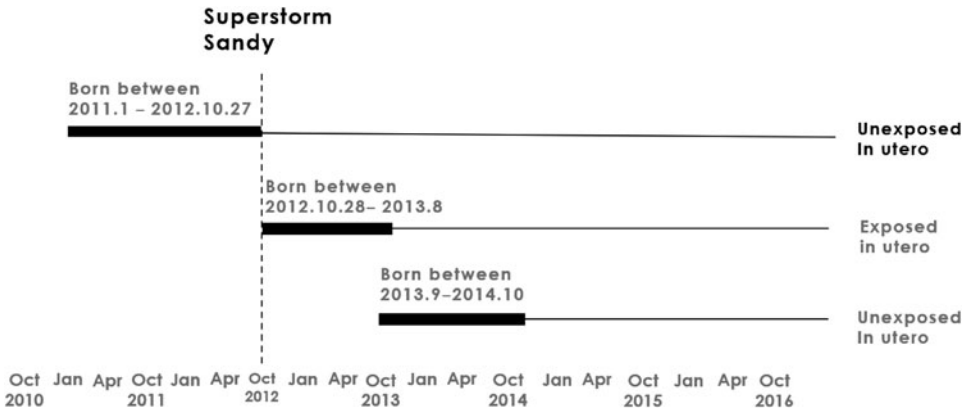

Figure 1. SIP study – exposure to Superstorm Sandy *in utero.*

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total score was used as a severity scale. The mean (*SD*) was 2.90 (2.98) with a range of 17. Cronbach's alpha was .899.

Prenatal maternal anxiety and depression symptomatology

Maternal anxiety and depression symptomatology were ascertained using the State–Trait Anxiety Inventory (Spielberger, 1989) and the Edinburgh Postnatal Depression Scale (Murray & Cox, 1990) during pregnancy based on mothers' self-reports. Trait anxiety, a long-standing quality of anxiety level, was used as a measure of prenatal maternal anxiety. Internal consistency, as measured by Cronbach's alpha, for anxiety traits and depression symptomatology in this study was .89 and .84, respectively. The mean (*SD*) and range for anxiety traits were 39.34 (10.50) and 50, while those for depression symptomatology were 9.25 (4.87) and 24, respectively.

Mother's drug abuse

The absence or presence of tobacco, cannabis, and alcohol use were ascertained during pregnancy via face-to-face clinical interview.

Child's and mother's demographic variables

An a priori confounder selection included child sex and race, as well as maternal age and education. Maternal age at the birth of the child was calculated based on the mother's and child's dates of birth. The socioeconomic status (SES) indicator was extracted using four factors – maternal education, pre-pregnancy occupation prestige (Evans & Mills, 1998), work status (full-time, part-time, and not working), and welfare status. Using latent class analysis, the optimal class solution (two-, three-, and four-class solutions) was tested for the SES index. The Lo–Mendell–Rubin (LMR) test for the three-class solution demonstrated significant model improvement (LMR = 84.58, $p < .0001$) with an entropy score of .914 showing it was a great model. Consequently, a three-category solution was chosen to be the best solution and used as an SES index (for details, see Supplementary Table 1).

Statistical analyses

Hierarchical linear modeling (HLM) was used to estimate both within-person longitudinal effects and between-person effects (Raudenbush & Bryk, 2002). The within-person model mapped the developmental trajectory of child behaviors (adaptive and clinical) at five data points from 2 to 6 years of age. The between-person model estimated how prenatal Sandy status influenced changes in child behavior. All models in the analysis were corrected for non-normal distributions of level 2 residuals by applying the full maximum likelihood estimation with robust standard errors to incorporate the missing data imputation (Maas & Hox, 2004).

Specifically, to compensate for missing data, common in many longitudinal cohort studies, HLM was used to yield parameter estimates for the missing time points for dependent variable data (i.e., child behaviors) at level 1 (i.e., within subject variability), but not for predictor variables at level 2 (i.e., between subject variability) (Raudenbush & Bryk, 2002). Given that there was a considerable follow-up time (2–6 years of age) and the majority of the children were missing behavior data for at least one time point during the follow-up period, this approach was optimal for examining differences in developmental trajectories. In the current sample, there was 1.2% missing data for the severity of Sandy at level 2. Following Schafer and Graham (2002), HLM computed average estimates that reflected the uncertainty of the missing level 2 data.

Change in child behaviors over time without a predictor (Model 1)

Model 1 was designed to characterize trajectories of child behaviors across five points in time from 2 to 6 years of age, free of a predictor and covariates. For each outcome, we first tested the model for linear change (Model 1a). As behaviors may not display linear change, especially in early development, we also tested for curvilinearity in each outcome model by adding a quadratic term for age (Model 1b). If a significant or marginally significant quadratic effect was not observed, the quadratic term was removed from the model. This was followed by tests of relative model fit by comparing the deviance statistics between the quadratic and linear models. If the chi-square test of difference demonstrated a significant reduction in deviance scores, the quadratic model was kept. The indices are presented in Table 2. In all models, random effects were included in the intercept and change coefficients (all linear terms and the quadratic were retained). Finally, age was centered at 2 years; that is, the intercept represented the average behavior score when children were 2 years of age.

Model 1a – linear model

In the linear model, child behavior was taken as a function of an intercept plus a linear effect for time (i.e., assessment wave). The equation is:

Level 1

$$\text{Behavior}_{ij} = \beta_{0j} + \beta_{1j} \times (\text{time}_{ij}) + r_{ij}$$

Behavior $_{ij}$: is behavior (outcome) at time i for participant j. β_{0j}, and β_{1j}/β_{2j}: are the intercept, and slopes (linear / curvilinear) respectively. γij: is the Level 1 regression residual term. $\gamma 00$ and $\gamma 10/\gamma 20$: indicate the average intercept and average slopes (linear /curvilinear), respectively. u0j, and u1j/ u2j : they are residual terms.

Level 2

$$\beta_{0j} = \gamma_{00} + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + u_{1j}$$

Model 1b – quadratic model

In the quadratic model, child behavior is a function of an intercept, and linear and quadratic effects for time (i.e., assessment wave). The equation is:

Level 1

$$\text{Behavior}_{ij} = \beta_{0j} + \beta_{1j} \times (\text{time}_{ij}) + \beta_{2j} \times (\text{time}_{ij})^2 + r_{ij}$$

Behavior$_{ij}$: is behavior (outcome) at time i for participant j. $\beta 0j$, and $\beta_{1j}/\ \beta_{2j}$: are the intercept, and slopes (linear / curvilinear) respectively. γij: is the Level 1 regression residual term. $\gamma 00$ and $\gamma 10/\gamma 20$: indicate the average intercept and average slopes (linear/ curvilinear), respectively. u0j, and u1j/ u2j : they are residual terms.

Level 2

$$\beta_{0j} = \gamma_{00} + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + u_{1j}$$

$$\beta_{2j} = \gamma_{20} + u_{2j}$$

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Table 2. Linear and quadratic change in child behaviors (clinical and adaptive dimensions) between ages 2 and 6

	Intercepts			Linear			Quadratic			Model comparison	
Behavior outcome	γ_{00}: *M*	*SE*	*t* ratio	γ_{10}: *M*	*SE*	*t* ratio	γ_{20}: *M*	*SE*	*t* ratio	$\Delta\chi^2$ (4)	*p*
Clinical scales											
Hyperactivity	49.40	.57	86.91*****	0.09	.40	0.22	−1.44	.53	−2.70***	14.58	.006
Aggression	47.89	.54	88.83*****	0.75	.41	1.84^{+}	−2.39	.56	−4.30***	22.02	.0002
Anxiety	52.16	.63	82.85*****	2.79	.45	6.16***	−3.16	.70	−4.54***	36.92	<.00001
Depression	49.65	.60	82.61*****	0.92	.47	1.94*	−2.29	.57	−4.00***	18.78	.0009
Somatic complaints	49.30	.53	92.69*****	1.13	.56	2.03*	−.19	.14	−1.41	4.92	.296
Atypical behaviors	51.06	.58	87.65*****	0.30	.59	0.51	−.12	.14	−0.84	3.52	.342
Withdrawn behaviors	49.32	.55	89.77*****	−.11	.36	−0.31	.14	.04	3.46***	21.52	.0003
Attention problems	49.92	.56	89.06*****	−1.05	.55	−1.91^{+}	.22	.14	1.59	10.91	.028
Adaptive scales											
Adaptive skills	47.45	.56	83.52*****	1.50	.40	3.72***	−1.14	.56	−2.03*	4.52	.338
Social skills	53.57	.61	86.96*****	2.86	.46	6.17***	−4.57	.71	−6.45***	65.51	<.00001
Activity of daily living	50.23	.63	80.18*****	1.41	.47	2.98**	.37	.69	0.54	9.16	.057
Functional communication	49.96	.51	97.32*****	2.31	.40	5.76***	−2.58	.57	−4.54***	33.84	<.00001

Note: γ values (*M*) represent the average, or fixed effects. *SE* = standard error. The chi-square difference test ($\Delta\chi^2$) yielded comparison between linear and quadratic models. + p ≤.10; *.05 ≥ p >.01; **.01 ≥ p >.001; ***.001 ≥ p >.0001; ****p <.0001.

Models with a predictor of intercepts, slopes and covariates (Model 2)

After choosing the linear or quadratic model, where appropriate, we explored whether Sandy status explained significant variance in the mean intercept or slope of child behavior. Covariates were added in the adjusted models, including child's sex, child's race, SES, maternal age, prenatal maternal substance use (tobacco, cannabis, and alcohol), prenatal maternal anxiety, prenatal maternal depression, and severity of Sandy exposure. If any aspect of child behavior displayed neither linear nor quadratic change over time, predictors were added to calculate the intercept only.

Model 2a – linear models with a predictor and covariates

Level 1

$$\text{Behavior}_{ij} = \beta_{0j} + \beta_{1j} \times (\text{time}_{ij}) + r_{ij}$$

Level 2

$$\begin{aligned}\beta_{0j} = {} & \gamma_{00} + \gamma_{01} \times (\text{Sandy status}) + \gamma_{02} \times (\text{child sex}) \\ & + \gamma_{03} \times (\text{child race}) + \gamma_{04} \times (\text{SES}) + \gamma_{05} \times (\text{maternal age}) \\ & + \gamma_{06} \times (\text{maternal anxiety}) + \gamma_{07} \times (\text{maternal depression}) \\ & + \gamma_{08} \times (\text{severity of Sandy exposure}) + \gamma_{09} \times (\text{tobacco use}) \\ & + \gamma_{010} \times (\text{cannabis use}) + \gamma_{011} \times (\text{alcohol use}) + u_{0j}\end{aligned}$$

$$\begin{aligned}\beta_{1j} = {} & \gamma_{10} + \gamma_{11} \times (\text{Sandy status}) + \gamma_{12} \times (\text{child sex}) \\ & + \gamma_{13} \times (\text{child race}) + \gamma_{14} \times (\text{SES}) + \gamma_{15} \times (\text{maternal age}) \\ & + \gamma_{16} \times (\text{maternal anxiety}) + \gamma_{17} \times (\text{maternal depression}) \\ & + \gamma_{18} \times (\text{severity of Sandy exposure}) + \gamma_{19} \times (\text{tobacco use}) \\ & + \gamma_{110} \times (\text{cannabis use}) + \gamma_{111} \times (\text{alcohol use}) + u_{1j}\end{aligned}$$

Model 2b – quadratic models with a predictor and covariates

Level 1

$$\text{Behavior}_{ij} = \beta_{0j} + \beta_{1j} \times (\text{time}_{ij}) + \beta_{2j} \times (\text{time}_{ij})^2 + r_{ij}$$

Level 2

$$\begin{aligned}\beta_{0j} = {} & \gamma_{00} + \gamma_{01} \times (\text{Sandy status}) + \gamma_{02} \times (\text{child sex}) \\ & + \gamma_{03} \times (\text{child race}) + \gamma_{04} \times (\text{SES}) + \gamma_{05} \times (\text{maternal age}) \\ & + \gamma_{06} \times (\text{maternal anxiety}) + \gamma_{07} \times (\text{maternal depression}) \\ & + \gamma_{08} \times (\text{severity of Sandy exposure}) \\ & + \gamma_{09} \times (\text{prenatal tobaccouse}) + \gamma_{010} \times (\text{prenatal cannabis use}) \\ & + \gamma_{011} \times (\text{prenatal alcohol use}) + u_{0j}\end{aligned}$$

$$\begin{aligned}\beta_{1j} = {} & \gamma_{10} + \gamma_{11} \times (\text{Sandy status}) + \gamma_{12} \times (\text{child sex}) \\ & + \gamma_{13} \times (\text{child race}) + \gamma_{14} \times (\text{SES}) + \gamma_{15} \times (\text{maternal age}) \\ & + \gamma_{16} \times (\text{maternal anxiety}) + \gamma_{17} \times (\text{maternal depression}) \\ & + \gamma_{18} \times (\text{severity of Sandy exposure}) \\ & + \gamma_{19} \times (\text{prenatal tobaccouse}) + \gamma_{110} \times (\text{prenatal cannabis use}) \\ & + \gamma_{111} \times (\text{prenatal alcohol use}) + u_{1j}\end{aligned}$$

$$\begin{aligned}\beta_{2j} = {} & \gamma_{20} + \gamma_{21} \times (\text{Sandy status}) + \gamma_{22} \times (\text{child sex}) \\ & + \gamma_{23} \times (\text{child race}) + \gamma_{24} \times (\text{SES}) + \gamma_{25} \times (\text{maternal age}) \\ & + \gamma_{26} \times (\text{maternal anxiety}) + \gamma_{27} \times (\text{maternal depression}) \\ & + \gamma_{28} \times (\text{severity of Sandy exposure}) \\ & + \gamma_{29} \times (\text{prenatal tobaccouse}) + \gamma_{210} \times (\text{prenatal cannabis use}) \\ & + \gamma_{211} \times (\text{prenatal alcohol use}) + u_{2j}\end{aligned}$$

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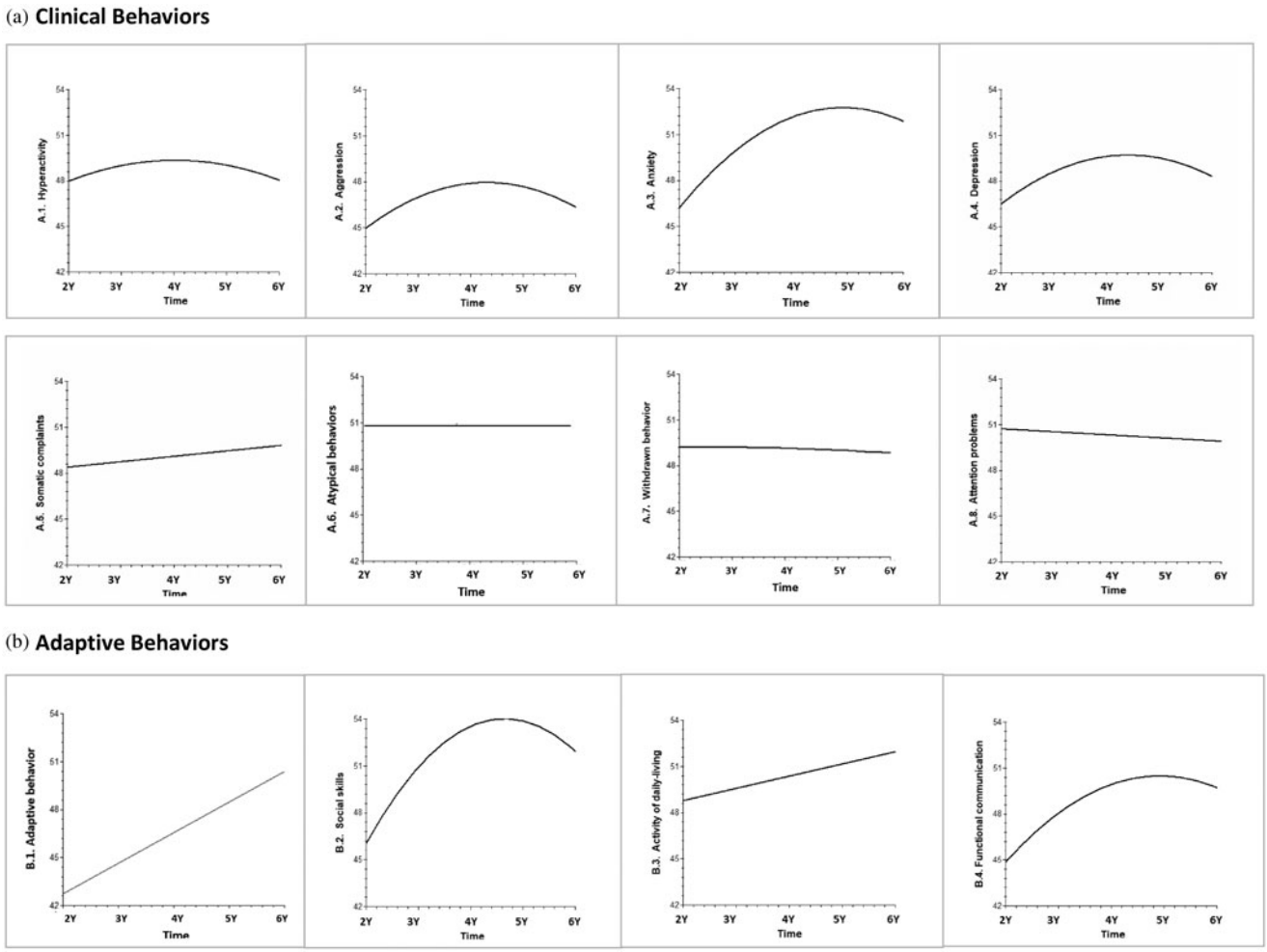

Figure 2. Developmental trajectories of child behaviors between ages 2 and 6 for the overall sample. A. Clinical behaviors. B. Adaptive behaviors. Linear models were selected for somatic complaints (A5), adaptive behaviors (B1), and activity of daily living (B3). Curvilinear models were selected for hyperactivity (A1), aggression (A2), anxiety (A3), depression (A4), social skills (B2), and functional communication (B4). Neither linear nor quadratic models were selected for atypical behaviors (A6), withdrawal behavior (A7), and attention problems (A8).

Effect size

Cohen's f^2 for sequential multiple regression, such as HLM, was used to determine the magnitude of effects when significant, with f^2 defined as:

$$f^2 = \frac{R^2_{AB} - R^2_A}{1 - R^2_{AB}}$$

where R^2_A is the variance accounted for by our predictor (exposure to Sandy) and R^2_{AB} is the combined variance accounted for by all variables included in the model. Guidelines for interpretation of f^2 indicate that 0.02 is a small effect, 0.15 is a medium effect, and 0.35 is a large effect (Cohen, 1992; Lorah, 2018).

Results

Model 1: Change in behavior scores over time without a predictor

We modeled child behavior as a function of the intercept plus the linear/quadratic effect of time (i.e., child's age). Figure 2 depicts the developmental trajectories of each behavior in two domains (clinical and adaptive). In the clinical domain, somatization increased linearly with age. Hyperactivity, aggression, anxiety, and depression increased in the earlier age range (2–4/5 years of age), but then decreased gradually till age 6. Withdrawn behavior slightly increased with age and peaked around 4 years of age, but the degree of change was nominal. Attention problems decreased linearly and marginally with age. There was no notable change in atypical behavior and an intercept-only model was selected. In the adaptive domain, adaptive skills and activity of daily living increased linearly with age. Social skills and functional communication increased between 2 and 4.5 years of age and then plateaued.

Model 2: A predictor of intercepts and slopes

A model incorporating curvilinear change over time was selected for hyperactivity, aggression, anxiety, depression, and withdrawn behavior in the clinical domain, and social skills and functional communication in the adaptive domain. A model evaluating linear change over time was selected for somatization and attention problems in the clinical domain, and adaptive skills and activity of daily living in the adaptative domain. An intercept-only model

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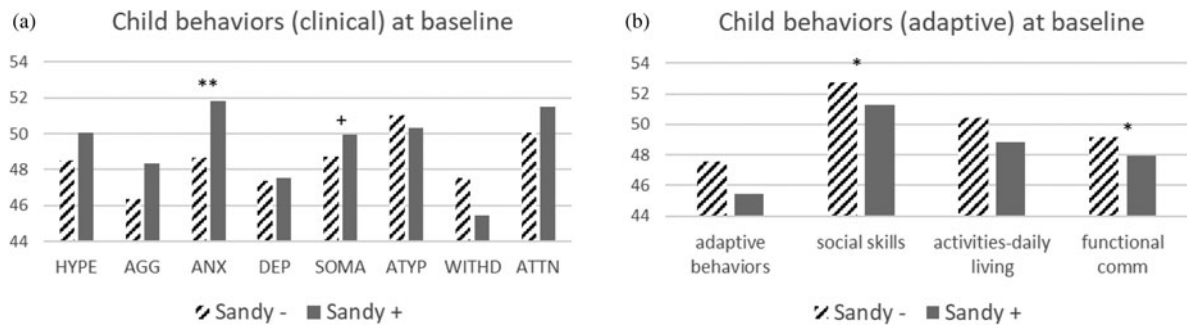

Figure 3. Comparisons of child behavior scores at 2 years (intercept) between children exposed and unexposed to Superstorm Sandy *in utero*. There were significant or marginally significant differences in anxiety (ANX, $p = .009$) and somatic complaints (SOMA, $p = .08$) in (A. Clinical behavior) and social skills (SS, $p = .04$) and functional communication (FC, $p = .02$) in (B. Adaptive behavior). The differences at the baseline were not significant in hyperactivity (HYPE), depression (DEP), atypical behaviors (ATYP), withdrawn behaviors (WITHD), atypical behaviors (ATYP), and attention problems (ATTN) in (A), adaptive behavior (ADAP) and activities in daily living in (B). $^{**}p < .01$; $^{*}p < .05$; $+ p < .10$.

was selected for atypical behavior in the clinical domain. After retaining the best-fitting model without a predictor, we examined whether Sandy exposure predicted mean intercepts (i.e., behaviors at 2 years) or slopes (i.e., rate/direction of change over time). In the interest of brevity, only significant results related to Sandy exposure are reported in the following. Adjusted models included all covariates.

Sandy exposure status during pregnancy

Intercept

There was a significant or marginally significant main effect of Sandy exposure status in predicting intercepts for anxiety (t ratio $= 2.71$, $p = .01$), social skills (t ratio $= -1.98$, $p = .05$) and functional communication (t *ratio* $= -2.26$, $p = .04$), indicating that children exposed to Sandy *in utero* had higher anxiety and lower social skills and functional communication than the unexposed control at 2 years of age (Figure 3).

Slopes

There was a significant main effect of Sandy exposure in predicting the linear slope for anxiety (t ratio $= 2.20$, $p = .03$), withdrawn behavior (t ratio $= -1.99$, $p = .05$), somatization (t *ratio* $= 2.20$, $p = .03$), social skills (t ratio $= 2.60$, $p = .01$) and functional communication (t ratio $= 3.88$, $p < .001$). Specifically, between ages 2 and 5, children exposed to Sandy *in utero* had a significantly greater increase in reported anxiety, social skills, and functional communication than those unexposed. Somatization rapidly increased with age in children exposed to Sandy *in utero* and mildly increased in children unexposed. Withdrawn behavior decreased in children exposed to Sandy *in utero* and increased in those unexposed to Sandy between ages 2 and 4. Of note, social skills and functional communication were lower at age 2 in children exposed to Sandy than in the unexposed group, but those exposed to Sandy caught up around age 2.5 with skill levels and continued to exceed the capacity for such skills compared to those unexposed over time.

We also observed significant curvilinear slope differences in children exposed to Sandy, compared to those unexposed, for anxiety (quadratic: t ratio $= -2.06$, $p = .04$), withdrawn behavior (quadratic: t ratio $= 1.98$, $p = .05$), social skills (quadratic: t ratio $= -2.81$, $p = .006$), and functional communication (quadratic: t ratio $= -3.42$, $p = .001$) (Figure 4). Specifically, the findings with quadratic trajectories demonstrated that children exposed to Sandy showed a continuous and more rapid increase in anxiety between 2 and 4 years of age followed by a slight decrease, although they remained at a higher level of anxiety, whereas those unexposed showed a slower increase in anxiety from 2 to 6 years of age.

Regarding withdrawn behaviors trajectories, there was a significant contrast between exposed and unexposed. The exposed had a greater level of withdrawn behaviors at age 2, which decreased as they grew older, with the lowest rate around age 4, and then increasing again. For unexposed children, lower withdrawn behaviors were evident at age 2, followed by an increase, peaking at around age 4, before decreasing again at age 6.

Regarding adaptive behaviors, both exposed and unexposed children had a curvilinear increase in their social skills and functional communication as they grew older, peaking at around 4/5 years of age. However, the rate of increase was significantly greater among the exposed group, compared to the unexposed. Of note, while unexposed children had significantly greater social skills and functional communication at age 2 than the exposed children, the exposed caught up by age 2.5 and thereafter exceeded the capacity, relative to unexposed. There were also differences in the patterns of growth in the skills between the two groups: the exposed children reached their peak slightly earlier (around age 4.5) than the unexposed (around age 5).

Discussion

This study investigated the effects of prenatal exposure to Superstorm Sandy on the trajectory of childhood behaviors from 2 to 6 years of age. The results suggest that an index of exposure to Sandy *in utero* would be a robust predictor of clinical behavioral problems, especially for anxiety, depression, and somatization in childhood. Such an index of *in utero* exposure could also be used to demonstrate greater adaptive functioning, especially social skills and functional communication among exposed, compared to unexposed, children over time. The current findings are consistent with our previous research, which showed that prenatal exposure to Sandy was associated with both an increase in positive emotionality (resilience) and in negative affect (risk) in exposed children (Zhang et al., 2018). Our findings on this continuous upward trajectory of anxiety, depression, and somatization, as well as the accelerated acquisition of social skills and functional communication among exposed relative to unexposed children, suggest that the stress a child experiences *in utero* has

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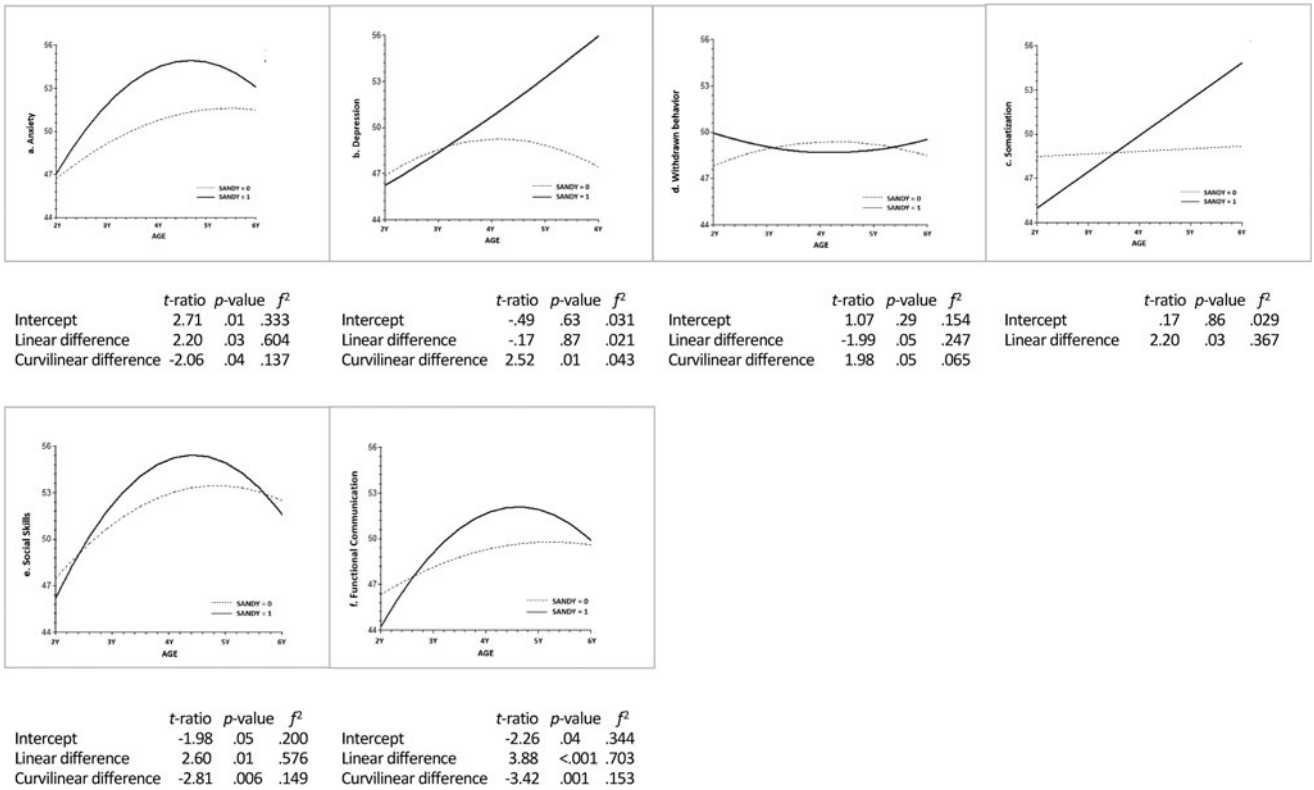

a. Anxiety	*t*-ratio	*p*-value	f^2
Intercept	2.71	.01	.333
Linear difference	2.20	.03	.604
Curvilinear difference	-2.06	.04	.137

b. Depression	*t*-ratio	*p*-value	f^2
Intercept	-.49	.63	.031
Linear difference	-.17	.87	.021
Curvilinear difference	2.52	.01	.043

d. Withdrawn behavior	*t*-ratio	*p*-value	f^2
Intercept	1.07	.29	.154
Linear difference	-1.99	.05	.247
Curvilinear difference	1.98	.05	.065

c. Somatization	*t*-ratio	*p*-value	f^2
Intercept	.17	.86	.029
Linear difference	2.20	.03	.367

e. Social Skills	*t*-ratio	*p*-value	f^2
Intercept	-1.98	.05	.200
Linear difference	2.60	.01	.576
Curvilinear difference	-2.81	.006	.149

f. Functional Communication	*t*-ratio	*p*-value	f^2
Intercept	-2.26	.04	.344
Linear difference	3.88	<.001	.703
Curvilinear difference	-3.42	.001	.153

Figure 4. Developmental trajectories of child behaviors (clinical and adaptive) between ages 2 and 6 among children who were exposed and not exposed to Superstorm Sandy *in utero*. Solid line = Sandy exposure; dotted line = control. Linear/quadratic slopes were significantly predicted by Sandy status for anxiety (a), depression (b), somatic behaviors (c) withdrawn behaviors (d), social skills (e), and functional communication (f).

detrimental effects on their mood and anxiety problems, but it may also influence their ability to acquire adaptative skills in the postnatal environment.

In the current study we aimed to investigate whether both clinical phenotypes around anxiety and adaptive skills would be influenced by distal exposure to Sandy *in utero*. While we hypothesized that children exposed to Sandy *in utero* would have a distinctive trajectory of clinical behaviors and adaptive behaviors, our HLM analyses were based on groups (exposed vs. unexposed) rather than individuals. As such, it cannot be assumed that the same individuals exhibited both elevated clinical and adaptive phenotypes – having greater scores on clinical and adaptive domains are not mutually exclusive. It is important that future studies examine what it means for children to function with both elevated anxiety and adaptive scores, especially during adolescence – a period of the highest risks for affective disorders.

Our findings are consistent with the growing literature regarding the effects of high stress levels on brain development, including alterations of both structure (De Brito et al., 2013; Gold et al., 2016; Lee, Kang, Chang, & Cho, 2019; Tottenham et al., 2010) and function (Gee et al., 2013; Posner et al., 2016; Suzuki et al., 2018). Neural circuits relevant to our findings relate to the prefrontal cortex and the limbic system, which includes the amygdala, hippocampus, and hypothalamus, involved in the generation and regulation of emotion (Dixon, Thiruchselvam, Todd, & Christoff, 2017; LeDoux, 2000). The amygdala, in particular, is a critical center for threat detection and fear behavior (Levy & Schiller, 2021). While the current study did not include *in vivo* neural assessments of the brains of our participants, it is notable that our clinical and behavioral findings are consistent with neuroimaging studies that identify brain structure and functional alterations of the mesocorticolimbic structures in relation to the effects of stress.

There are several strengths of this study. First, the choice of analytical strategy (HLM) enabled us to estimate missing data and optimize the longitudinal data, thus allowing the potential to detect changes in child behaviors, both clinical and adaptive, as a function of *in utero* Sandy exposure. Second, unlike most studies, where prenatal stress has been largely addressed as a normative stress in everyday life (Rubonis & Bickman, 1991), as maternal psychopathology, or as a result of low SES during pregnancy, our study examined the effects of a large-scale disaster first hand (i.e., Superstorm Sandy) on developmental programming (Brand et al., 2006; Huizink et al., 2007; King & Laplante, 2005; Kuvacic, Skrablin, Hodzic, & Milkovic, 1996; Laplante, Brunet, & King, 2016, 2008; Meijer, 1985; Nomura et al., 2019; Tees et al., 2010; Yehuda et al., 2005; Zhang et al., 2018). Third, we were in the rare position of being able to conduct a quasi-experiment using a profound natural disaster (Sandy). All our participants were exposed to Sandy at different times during their index pregnancy. As stress is an intangible concept, much of the interpretation is subjective. The use of objective natural disaster stress allowed us to eliminate some subjective perceptions of stress. Moreover, the severity of exposure, leveraging existing objective measures of natural disaster (i.e., Storm32), was measured and the difference in the severity that each participant experienced was adjusted in the models. Another important aspect of this study is the use of BASC2-P to assess behavioral and emotional phenotypes in children over 4 years. Similar to the broadband Child Behavior Checklist (CBCL) scales, one of the

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strengths of the BASC2-P scale is its cost-effective approach in identifying offspring at higher risk for psychopathology. BASC2-P scales have several items that provide information correlated to diagnostic criteria for various clinical categories listed in DSM-IV-TR (the text revision of the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition), for example, ADHD, conduct disorder, oppositional defiant disorder, generalized anxiety disorders, and depression (Kamphaus, VanDeventer, Brueggemann, & Barry, 2007; Papazoglou & Ferrari, 2013). Using self-report screeners such as BASC or CBCL for the assessment of childhood behavioral/emotional problems, various longitudinal studies have documented a link between early problems and later psychopathology in youth (Bradstreet, Juechter, Kamphaus, Kerns, & Robins, 2017; Thompson et al., 2015; Volker et al., 2010) and in adults (Faraone et al., 2005; Mesman & Koot, 2001). For example, in a longitudinal study, Hofstra, Van Der Ende, and Verhulst (2002) showed that persistent self-reported emotional and behavioral problems were linked to a higher lifetime prevalence of DSM-IV disorders. Self-report screener instruments, such as BASC2-P scales, are suitable tools for investigating trajectory and different psychiatric disorders risks later in life.

This study also has several limitations. First, the severity of the experience of Sandy was ascertained by maternal self-report. While this information is also a strength, the measure was designed to assess the number of objective challenges due to the disaster and could have been influenced by maternal psychological functioning. Second, the index for *in utero* Sandy exposure was a simple index pregnancy based on when Sandy made landfall. While we recognize the possible simplicity of the index, the overall focus of the study was to understand the implication of fetal programming, and objective severity of exposure was controlled for in all analyses. Third, child clinical and adaptive behavior measures were based on maternal report, using screening instruments for child clinical problems. It is well-known that maternal characteristics (i.e., depression and stress) can influence the way of reporting child developmental and behavioral patterns. However, at the baseline, our participants were relatively young (2 years of age) and during the time of early development, mothers are considered to be the best informants. Fourth, while a well-validated instrument of child behavior (clinical and adaptative) was used in this study (BASC2-P), diagnostic outcomes ascertained by clinical interviews could have increased the validity of the clinical measures. Fifth, although we included various sociodemographic confounders determined a priori, we might have overlooked some important confounders.

Despite these limitations, our research suggests that stress from Sandy, experienced *in utero*, was associated with a greater level of clinical behaviors (anxiety, depression, and somatization), but induced an elevation in adaptive functioning (social skills and functional communication) over time, which supports the fetal programming and evolutionary perspective. Our study also has clinical significance as maternal exposure to disaster-related stress may produce alterations in child behavioral development, with implications for developmental psychopathology. Our findings underscore the benefits of mental health intervention and stress management during prenatal care to reduce later developmental health burdens. Our findings also reaffirm the need to invest in pediatric practices to increase the effectiveness and capacity of services for national disaster preparedness. In particular, our results highlight how pregnant mothers are an especially vulnerable population, and there is an urgent need for vigorous post-disaster outreach to this population during their gestation and postnatal periods. The frequency of natural disasters may be increasing due to climate change, but we can counsel policymakers, educators, and health care providers on the importance of early intervention to ameliorate possible adverse effects on child neurobehavioral outcomes and foster further improvements in the adaptability of future generations that will help bend the trajectory curves for better child outcomes.

Supplementary Material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579421000304

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Conflicts of Interest. None.

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